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Précis of *Lifelines: Biology, freedom, determinism*[1]

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Abstract: There are many ways of describing and explaining the properties of living systems; causal, functional, and reductive accounts are necessary but no one account has primacy. The history of biology as a discipline has given excessive authority to reductionism, which collapses higher level accounts, such as social or behavioural ones, into molecular ones. Such reductionism becomes crudely ideological when applied to the human condition, with its claims for genes "for" everything from sexual orientation to compulsive shopping. The current enthusiasm for genetics and ultra-Darwinist accounts, with their selfish-gene metaphors for living processes, misunderstand both the phenomena of development and the interactive role that DNA and the fluid genome play in the cellular orchestra. DNA is not a blueprint, and the four dimensions of life (three of space, one of time) cannot be read off from its one-dimensional strand. Both developmental and evolutionary processes are more than merely instructive or selective; the organism constructs itself, a process known as autopoiesis, through a lifeline trajectory. Because organisms are thermodynamically open systems, living processes are homeodynamic, not homeostatic. The self-organising membrane-bound and energy-utilising metabolic web of the cell must have evolved prior to so-called naked replicators. Evolution is constrained by physics, chemistry, and structure; not all change is powered by natural selection, and not all phenotypes are adaptive. Finally, therefore, living processes are radically indeterminate; like all other living organisms, but to an even greater degree, we make our own future, though in circumstances not of our own choosing.

Keywords: autopoiesis; developmental trajectories; evolutionary theory; homeodynamics; human behavior; metabolic webs; neurogenetics; reductionism; self-organisation; ultra-Darwinism

Preface

In the last decade, especially in the context of dramatic advances in the sciences of both genes and brains, the stream of ultra-Darwinist and biologically determinist claims has become a torrent. First the Human Genome Program and then the Decade of the Brain have not merely offered vastly greater knowledge of aspects of human biology, but they have also held out the promise of further technological power to manipulate both genes and minds in the interests of individual health and greater social tranquility.

Techniques of intervention barely imaginable a decade ago, at best the stuff of science fiction, now rate stock market quotations and turn academic researchers into entrepreneurial millionaires. To judge from headlines in daily newspapers, or the titles of academic papers in major scientific journals, the issues of a decade ago have been settled. Vulgar sociobiology may be out, but what I have called "neurogenetic determinism" is strongly entrenched. There are genes available to account for every aspect of our lives, from personal success to existential despair: genes for health and illness, genes for criminality, violence, and "abnormal" sexual orientation – even for "compulsive shopping." And genes too to explain, as ever, the social inequalities that divide our lives along lines of class, gender, race, ethnicity; and where there are genes, genetic and pharmacological engineering hold hopes for salvation that social engineering and politics have abandoned.

The challenge to the opponents of biological determinism is that while we may have been effective in our critique of its reductionist claims, we have not offered a coherent alternative framework within which to interpret living processes. We may reply that we have been too busy attempting to rebut the determinists, but sooner or later it becomes necessary to spell out more coherently our contrasting biological case. *Lifelines* (Rose 1997) originated as an attempt to meet that challenge, first, to try to convey what it means to "think like a biologist" about the nature of living processes, second, to analyse both the strengths and limitations of the reductionist tradition which dominates much of biology, and, third, to offer a perspective on biology which transcends genetic reductionism, by placing the or-

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ganism, rather than the gene, at the centre of life – this is the perspective that I call *homeodynamic.* To stress my positive case, it has also in places been necessary to set it against the counter-case made at its rhetorical strongest. To do so, I have had to choose appropriate foils. The two authors who have most clearly served me in this way are the sociobiologist Richard Dawkins, whose several books speak with a single ultra-Darwinist voice, and the philosopher Daniel Dennett, whose *Darwin's Dangerous Idea* (Dennett 1995) carries ultra-Darwinism to the furthest reaches of excess.

Because *Lifelines* is written for a general audience, several sections, notably those on genetics and development, include a substantial amount of explanatory material which will be familiar to most readers of this journal, and I have therefore omitted them from this précis, or abbreviated them to summary statements of the examples employed.

Chapter 1: Biology, freedom, determinism

The power of western science derived from its capacity to explain and later to control aspects of the non-living world studied by physics and chemistry. Only subsequently were the methods and theories shaped by the success of these older sciences turned towards the study of living processes themselves. The past successes of science have been based not so much on observation and contemplation but on active intervention into the phenomena they wish to explain. Biologists are now beginning to lay claims to universal knowledge, of what life is, how it emerged, and how it works. Throughout all life forms and all living processes, certain general principles hold; certain mechanisms, certain forms of chemistry, exist in common. But intervention into living processes confronts us with moral dilemmas, because biology impinges directly on how we live. Its technologies transform our personal, social, and natural environments and make claims as to who we are, about the forces that shape the deepest aspects of our personalities, and even about our purposes here on earth.

The science we do, the theories we prefer, the technologies we use and create as part of that science, can never be divorced from the social context in which they are created, from the purposes of those who fund the science, the world views within which we seek and find appropriate answers, to the great what, why, and how questions that frame our understanding of life's purposes. So with modern biology, whose diverse answers to these questions are imbued with social and political significance. The dominant fashion for giving genetic explanations to account for many if not all aspects of the human social condition – from the social inequalities of race, gender, and class to individual propensities such as sexual orientation, the use of drugs or alcohol, or the failures of the homeless or psychologically distressed to survive effectively in modern society – is the ideology of biological determinism. I offer an alternative vision of living systems which recognises the power and role of genes without subscribing to genetic determinism, which recaptures an understanding of living organisms and their trajectories through time and space as lying at the centre of biology. It is these trajectories that I call "lifelines."

1.1. Themes: Biological questions. There is commonly supposed to be a hierarchy of sciences, ranging from physics through chemistry, biology, and the human sciences. In this convention, physics is seen as the most fundamental of the sciences to which the others must aspire, or be reduced. But biological science raise themes which cannot be reduced to physics. To show why, consider a frog jumping into a pool. The cause of the jump may be described as the contraction of the leg muscles, preceded by nerve impulses, and so on. Or one could explain the jump in terms of intention – to escape a predator – or in terms of ontogeny, or of phylogeny – or in terms of the actin and myosin fibres of which the muscle is composed. All are necessary and valid parts of description; only the last is reductionist. Which explanation one finds satisfying depends on the purposes for which it is intended.

1.2. Themes: Time. The concept of time and the direction of "time's arrow" are central to biology. Living processes are complex, often irreproducible because historically contingent, and hence also practically irreversible. Dobzhansky (1973) asserted that "nothing in biology makes sense except in the light of evolution." I claim that nothing in biology makes sense except in the light of history – the history of life in general, of individual development, and of our own science and its concerns. The past is the key to the present.

1.3. Themes: Space. The second deep theme with which biologists are concerned is that of structure. To the three dimensions of space must be added the dimension of time. Organisms have forms which change but also persist throughout their life's trajectory, despite the fact that every molecule in their body has been replaced thousands of times over. How is form achieved and maintained? Cells, organisms, are more than simple lists of chemicals. Neither their three-dimensional structures nor their lifelines can simply be read off from the one-dimensional strand of DNA. Today the task of a biology of structure has become one of understanding how to reassemble these components, to explain both form and its transformation and persistence through time.

1.4. Themes: Dynamics. Homeostasis is a dominating motif of biological thought. But the metaphor of homeostasis constrains our view of living systems. Lifelines are not purely homeostatic, they have a beginning at conception, and an end at death. Organisms, and indeed ecosystems, develop, mature, and age. The set points of homeostatic theory are not themselves constant during this trajectory but change over time. To put the organism and its lifeline back at the core of biology means replacing the static, reductive, DNA-centred view of living systems that currently pervades biological thinking by an emphasis on dynamics, process, the relationships between object and fields, and the paradox of development by which any organism must simultaneously be and become.

1.5. Themes: Autopoiesis. These processes of development transcend the crude dichotomies of nature and nurture, gene and environment, determinism and freedom. Instead we must speak of the dialectic of specificity and plasticity during development through which the living organism constructs itself. The central property of all life is autopoiesis, the capacity and necessity to build, maintain, and preserve itself.

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Chapter 2: Observing and intervening

Science begins with observation, but no one observes neutrally. We construct our world. Observation demands sampling and categorising events and processes, distinguishing object from field. Most sciences are also interventive, requiring that experimental conditions be constrained and manipulated – and hence reduced (e.g., my own work on memory in chicks, Rose 1992).

This example also points to another feature of science, the use of resemblance (chick memory as equivalent to human memory). Metaphors occur when we liken some process or phenomenon observed in one domain to that in a quite different one. Metaphors are not meant to imply identity of process or function, but should serve to cast light on the phenomenon one is studying. Analogy implies a surface resemblance between two phenomena, perhaps in terms of the function of a particular structure. By contrast, homology implies a deeper identity, derived from an assumed common evolutionary origin. Thus, homology carries with it an assumption of shared history and origins, and, by inference, it implies common mechanisms (e.g., forelimb hooves of a horse and the human hand; chick and human memory).

Does memory consitute a Platonic natural kind? Are there indeed "natural kinds" in biology? Neither individuals, nor species, nor complex biological molecules such as proteins can be so regarded. Each is defined operationally and has a unity given by process, not by composition.

Chapter 3: Knowing what we know

The purpose of observing and experimenting is to derive knowledge of the world and its workings, to enable us to predict and control it. This action imperative characterises modern science. Bacon understood this when he described experiments as being of two kinds, those that brought light and those that brought fruit. For Bacon, knowledge came through induction. Popper replaced induction with hypothesis-making. Kuhn argued instead for paradigms, frameworks within which the problem-solving of normal science was set. But where do paradigms come from? For Kuhn the question was an individualistic, almost psychological one. However, following both the older Marxist tradition and the newer insights of the sociologists of science, Kuhn's question allows the social into science. We frame our questions of the world in ways which are constrained both by the material reality of that world and by the paradigm blinkers: our social expectations and the history of how our science chooses to ask its questions. Here the power of metaphor in biology – often derived from technological or social artefacts – becomes important (e.g., ATP as a currency system, DNA as a code, brains as computers; social organisms as conforming to monetarist cost-benefit economics). Favoured paradigms and hypotheses are rarely simply disconfirmed (e.g., an experiment of mine done jointly with Sheldrake to test so-called morphic resonance). Nor does the fact that a technology "works" mean that the framework in which it is based is true (e.g., claims that children who misbehave at school suffer from attention deficit disorder and can be treated with the drug ritalin; ritalin may indeed make a child more tractable, but that does not confirm the original diagnosis).

A further constraint on science is the available technology. The questions that we ask of the living processes we study are not merely not answerable without the technology, they are unthinkable. Before the development of light microscopes in the seventeenth century, the existence of bacteria and other single-celled organisms was wholly unsuspected. So was the "unit" of life, the cell, until the microscopists of the mid-nineteenth century. Until the advent of the electron microscope in the early 1950s, the internal constituents of cells were unobservable and hence unknown. It was impossible either to build theories about the partition of cellular functions which such subcellular particles might embody.

Technology solves certain problems and suggests others. But it also constrains the way we view the world. Example: electron microscopy (EM). To prepare living tissues for EM it is necessary to fix, embed, stain, and section. The apprentice electron microscopist is taught how and what to see, what to regard as "real" and what as "artefact" – the unwanted consequences of one or more of the procedures which the living tissue has been put through. Thus, the new observer is initiated into the conventional wisdom developed by forty years of work in the artificial world of electron microscopy. This fixed pattern of the electron micrograph forms the basis for drawing of cells in biology textbooks and provides the convention within which even experienced biologists mainly think of them. So powerful is the technology that it becomes hard to move beyond it, to think in three, let alone four dimensions (e.g., x-ray diffraction patterns; gel separation of proteins).

These are the sciences made possible by technology, the technologies made possible by science. The world view we create is derived from the intimate interaction of technology and science with the eye of craft experience, shaped by the theoretical expectations within which we as biological researchers must live. It is a world which presents challenges which go deeper than Popperian hypothesis-making, Kuhnian paradigms, and truth versus performance with which those studying the epistemology of science attempt to make their own sense of what we do. Wresting reliable knowledge from the world we biologists study is, as Koestler described it, an Act of Creation.

So what justification can there be for claiming that it is possible to obtain reliable knowledge of the living world? The evolutionary lineage which led to humans has been characterised by the development of more flexible organisms with bigger and more powerful brains, able to adapt to respond to rapidly changing circumstances. To survive and succeed, our evolutionary forebears had to rely on their brains to make reliable hypotheses about the world around and to act appropriately upon them. If the mental world which we construct in this way did not correspond reasonably accurately to the way the world outside "really" is, we could not survive. Such hypothesis-making may be seen as the starting point for science. But science is socially organised hypothesis-making. Hypotheses must be shared, tested, and agreed on amongst a community. Nothing that occurs in the non-human animal world matches the cumulative nature of hypothesis-making which constitutes human science. We are able to build on the tested and seemingly validated hypotheses not merely of those who are currently alive but those of all previous generations.

Humans, however, are more than just scientific hypothesis makers. We live in communities shaped by many other cultural and economic forces that provide strong guidance

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as to how we should view the world around us and our fellow humans. In Britain in the 1990s, where the gap between rich and poor is greater than it has been for a generation, the world is seen from very different perspectives by the directors of the privatised utilities and by the people they have sacked. In a society in which there is a strong division of labour and power between men and women in every field of work from science to child care, their viewpoints on the world will also differ. A white racist football fan will be unlikely to make the same hypotheses about the world as will the black player he abuses.

For many fields of scientific hypothesis-making, these rather crudely drawn distinctions may be irrelevant, but biology claims to be in a position to tell us, as humans, who we are, where we came from, where we are going, and how we must live and relate to our fellow living creatures. The metaphors and analogies we find attractive are laden with cultural values and expectations that come from outside our science. Those who deny this are ignorant of the hard work done by philosophers and sociologists in developing an understanding of the nature of science and the knowledge it creates.

Despite this doubt at the very core of the scientific endeavour, we are not in a position to assert that "anything goes." Although the observations we make about the world are theory- and ideology-laden before we start, and the joints into which we carve nature are provided less by *a priori* definitions than by operational need, they nonetheless must make some more or less good fit with the world or we could not proceed. Our hypotheses would fail. However great their budget, genetic engineers will not be able to turn humans into angels, nor cryogenicists restore the memories of the past owner of a severed and frozen head.

Chapter 4: The triumph of reductionism?

To many scientists reductionism is first not second nature. As an example, an exchange took place at a Royal Society Meeting in 1986 between Popper, who argued that biochemistry is irreducible to chemistry, and Perutz, who drew on the role of haemoglobin to claim that it could be so reduced. But knowledge of the molecular structure of haemoglobin explains how it serves as an oxygen carrier, and cannot answer the biological question of what function such a capacity serves in a living organism.

4.1. Reductionism as methodology. We find it easier to understand phenomena if we can hold them relatively isolated from the rest of the world and alter potential variables singly, simplifying and enabling one to generate seemingly linear chains of cause and effect (e.g., studying enzyme kinetics by altering either temperature or pH; altering both simultaneously makes mathematisation almost impossible.) Hence the attraction of reductionism and why it has served science so well. But living systems are not simple. Variables interact. Parameters are not fixed. Properties are non-linear. And the living world is highly non-uniform. We fail if we are not careful to remember that what happens in the test-tube may be the same as, the opposite of, or bear no relationship at all to what happens in the living cell, still less the living organism in its environment. It all depends.

4.2. Theory reduction. One of the aims of science is to simplify, to embrace a maximal description of the world within the minimum possible number of laws and variables. The history of science contains a number of examples of what were originally believed to be different phenomena, and were only later discovered to be identical (e.g., the morning and evening stars, regarded as distinct in previous cosmologies, now understood to be a single entity, the planet Venus; the sciences of heat and light were once regarded as distinct; today both heat and light are seen as forms of electromagnetic radiation).

Some unifications have been immensely powerful, particularly at the interface between biochemistry and chemistry. For example, Lavoisier's recognition that the body's "burning" of the sugar glucose to carbon dioxide and water, with the concomitant production of utilisable energy, was in chemical terms the equivalent of oxidation. This understanding that living processes did not depend on some mysterious life force but involved chemical reactions which followed the same rules as those of chemistry and could be studied in isolation was one of the great reductionist triumphs of the nineteenth and early twentieth centuries. More than mere metaphor, homology, or analogy, it was an exact description. Yet there are dangers inherent in such theory reduction, which led to a philosophy of mechanical materialism amongst physiologists.

Modern textbooks offer the reduction of "gene" to "DNA" as a case parallel to that of the identity of the morning and evening stars. But "gene" and "DNA" are not (just) two names for the same object. And it is at this point that theory reduction tips over into its problematic, philosophical form which claims that ultimately chemical theory is reducible to a special case of physics, biochemistry to chemistry, physiology to biochemistry, psychology to physiology, and ultimately sociology to psychology and hence to physics (e.g., E.O. Wilson's [1975] claim that neurobiology will cannibalise sociology).

To see the implications of this type of reductionism, consider biochemistry and physiology. Although the two sciences may speak different languages, use different instruments, and read different journals, the phenomena they are studying are the same, but at different levels – also an ambiguous term. But what would the elimination of physiology for biochemistry imply? Are we trying to describe a causal relationship – that biochemistry (say, of a muscle twitch) is causally responsible for the physiological event? If so, this is a very different use of the word "cause" from the way in which we normally use it to describe the relationship in time between cause and effect – one event necessarily and specifically following from another. But the sliding of the actin and myosin filaments (the biochemist's description) does not precede the muscle contraction; it *is* the muscle contraction, or at least part of it. That is, the relationship between the events described in the two languages is one of identity, not cause – but a non-reductive identity. There are features of the muscle twitch, such as function, which physiology describes but biochemistry cannot. To put it formally, ontological unity permits epistemological diversity. The key feature which distinguishes a lower "level" from those above it is that at each level new interactions and relationships appear between the component parts, relationships which cannot be inferred simply by taking the system to pieces.

Furthermore, philosophical reductionism implies that whatever higher order properties emerge, they are somehow secondary to the lower order ones. Parts come before

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wholes. Yet the nature of evolutionary and developmental processes in biology means that there is no such necessary primacy. Wholes, emerging, may in themselves constrain or demand the appearance of parts. To understand the world's ontological unity we need the epistemological diversity that the different levels of explanation offer.

Chapter 5: Genes and organisms

The trajectory of any organism through time and space – its personal lifeline – is unique. Although each individual resembles all others of the same species, and resembles more closely still its parents and siblings, no two are identical. What confers such similarities, such identities and differences, on the space-time trajectories of life? These questions are the objects of study of genetics and developmental biology, which began by asking rather similar questions about the nature of life but at a key point in their history became damagingly separated one from the other. This has resulted in conceptual confusions which have persisted well into the present day. But to appreciate the consequences of these confusions we have to go back into the history of genetic and developmental thinking. Biology's own history is centrally engaged within these current disputes.

The genesis of genetics lay with Mendel, who not only showed that certain characters were transmitted independently but introduced quantitative measures, observing that they appeared in successive generations in simple and reproducible ratios. Inheritance was discrete; each surface property was represented by an unobservable particle or store of information, on the basis of which the colour and shape of the succeeding generations was determined (hence, e.g., 3/1 ratios). Mendel was lucky. By contrast, the characters that interested Galton – human features such as height, strength of handgrip, head circumference, or intelligence – varied continuously across a broad range and offspring tended to occupy middle territory between their parents. Such continuously varying characteristics seemed to blend. Indeed Mendel's ratios turn out to be very special cases, even though, following their rediscovery in 1900, they formed the cornerstone of genetics. The individual hidden determinants of surface characters became genes, and the total of an individual's genes formed its genotype (nowadays genome). The surface characters themselves comprised the individual's phenotype. It is important to recognise that none of these terms were very precisely defined, and practically from the beginning meant different things to different researchers, varying from the specific features of any individual of a species to some Platonically idealised "species type" to which all actually existing members of the species approximated. Genes were essences: the ultimate, indivisible units on which the outward forms depended; the unmoved movers, unchanged changers, within each organism.

"Phenotype" is similarly ambiguous, and is used to refer to any or all observable or measurable features of an organism, from the presence of a particular enzyme to hair colour or body feature or even a piece of characteristic behaviour such as gait whilst walking. Dawkins (1982) even goes so far as to describe aspects of the external environment of an organism as part of its phenotype – for instance, he sees the dam that a beaver constructs as part of that beaver's phenotype. Yet the dam is not the product of the activity of a single individual, but of the collective labours of many beavers. It also harbours a multitude of other species. If the dam is a phenotype, it is the phenotype of a community, not of an individual, and its relationship to any individual's genes, genotype, or genome is thus tenuous.

The distinction between discontinuous variation and continuous variation remained problematic through the 1920s. Pearson developed many of the statistical methods still in use today to analyse complex data. Indeed the histories of genetics and of statistics have been thoroughly interlocked ever since. The resolution of the conflict depended on the recognition that continuous variation, in features such as height, could be regarded as a consequence of the interaction of many genes of small effect. Divergences from simple Mendelian ratios steadily accumulated (e.g., sex-linked characters). Other divergences from the ratios are less straightforward and the models became more complex to account for them. However complicated and varied the observed phenotypes, the modellers were still determined to explain them on the basis of the interaction of the indivisible causal particles which they conceived genes to be. If the ratios did not work it was because some other factors were obscuring the proper functioning of the genes (partial dominance; incomplete penetrance). Once these possibilities are admitted, there is virtually no distribution of phenotypes found in the population to which a genetic model cannot be fitted. In the traditional Popperian sense, such genetic models are strictly unfalsifiable. Given enough assumptions, any model can be "fixed" (e.g., schizophrenia).

Whilst the Mendelian rediscoverers were busy defining the phenotypic features they observed as the products of hypothesized genes, other biologists were looking at organisms from quite a different perspective. How does the union of egg and sperm ultimately produce an organism which may consist of 10^{14} such cells, differentiated into tissues and organs, precisely located in space? Embryologists described cell division, from fertilisation to the formation of the blastula and gastrulation, and identified a role for the chromosomes. This rhythm of cell division unrolling in a seamless sequence operates according to rules which the early embryologists found hard to fathom. For some, the only explanation was that the developing embryo was imbued by an irreducible life force. To most, this conclusion was unacceptable; they were observing a complex piece of living clockwork. Whichever philosophy one adopted, the dividing ball of cells was splendidly accessible to experimental manipulation. What would happen, for instance, if one removed a portion of the dividing cell ball, or cut it neatly in half? The results confused researchers for decades, for the conclusion seemed to be "it all depends." Depends on the organism, depends on how many divisions the ball of cells has made prior to the cut; depends from where in the ball one removes the sample (e.g., contrast between Roux, Driesch, and Loeb). Depending on the organism, at early stages in the cell division process, each cell still retains all the determinants – genes – to make an entire offspring; at later stages some regions of the developing ball of cells retain this capacity and others do not; later still the capacity is entirely lost, and the developmental fate of each region of the cell ball is fixed and cannot be modulated.

Transplant studies revealed more. Sometimes a cell's fate is determined by the environment into which it has been

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transplanted, in others it carries its own fate with it (e.g., transplant a group of cells from the region of a developing insect destined to become a leg, and insert it into the head region. Depending on the age of the embryo, the number of divisions it has undergone since fertilisation, the transplanted tissue may be incorporated into the developing head or it may develop into an additional leg projecting anomolously from the head). During mitotic division each cell receives an identical set of determinants or genes and is totipotent. Later, although all the genes are still present in all the cells, which genes are expressed depends on the developmental history of the particular cell. Thus gene expression depends on both time and space.

The major concern of developmental biology remained that unrolling programme which led from a single fertilised egg to the fully formed organism. How is it that what seem at first sight to be very similar cell masses, going through seemingly similar transformations, end up in the one case producing a mouse and in the other a human? Why do the daughters of a cell from one part of the dividing embryonic cell mass end up as liver and from another as brain or bones? How is it that all individual humans end up so astonishingly similar? Developmental biology is the science of the rules which produce regularities, similarities between organisms. Genes are part of a harmonious dialectic of interaction with the environment by which fertilised cells become mature adults through a trajectory described as ontogeny. And the constraints on this trajectory are only in part genetic.

By contrast, genetics was and is concerned with differences. Why is one *Drosophila* red eyed, the other white eyed? Why do people differ in height? Why do some have blood cells which carry a haemoglobin molecule which seems unable to bind and carry oxygen as efficiently as is normal? The question is to be answered in terms, ultimately, of the modern descendants of Mendelian determinants, the genes. Thus for genetics, genes came to be understood as discrete units which lead in linear fashion to red versus white eyes or any other character of interest. Ontogeny is of interest only insofar as genetic differences may produce abnormalities in development. Otherwise, the geneticists' organisms are empty of time and internal content; there are only genes and phenotypes. They have no trajectory, no lifeline.

Using *Drosophila,* Morgan and his group showed that unusual mutations were transmitted in a Mendelian manner and increased by X rays. Genes had a physical location in the cell, on chromosomes, and were thus distributed to daughter cells during mitosis, making possible the beginning of gene mapping. The term gene now had two different meanings. On the one hand it was still an abstract entity, the determinant of a particular phenotypic character. On the other it had a clear location, a map reference, and could be shown to be physically transmitted between cells and their offspring during both division and sex. The step which took genetics beyond Morgan's location of genes to chromosomes also brought it into conjunction with biochemistry for the first time. Mutations in *Neurospora* were even easier to induce and study than in fruit flies, but now the consequences were metabolic. Mutants lack specific enzymes which play a crucial role in the pathways which lead to missing metabolites. Each specific mutation leads to the absence of a specific enzyme. Hence Beadle and Tatum's formulation: one gene equals, or produces, one enzyme.

Genes were no longer to be understood only as hidden entities, metabolical accounting devices. They no longer determined characters, but instead, in a yet-to-be understood manner, were responsible for the production of enzymes. So what is a gene "for" a character? Consider eye colour, which depends on the presence in the cells of particular pigments. In their absence, the eye is blue, increasing quantities of the pigments provide colours which range from green to brown. Ignoring the developmental processes that lead to the formation of the eye, and within the eye the iris, and the biochemical steps whereby the necessary precursors to the synthetic pathway are produced, the direct pathway that leads to the synthesis of the eye pigments involves many different enzymes. Hence to biochemists there is no longer any gene "for" eye colour. Instead there is a difference in the biochemical pathway that leads to brown and to blue eyes. A gene "for" blue eyes means "one or more genes in whose absence the metabolic pathway which leads to pigmented eyes terminates at the blue eye stage." This rephrasing yet again exposes the distinction between a developmental and a genetic approach. For the developmental biologist, what is of interest is the route which leads to pigmented eyes. But the geneticist is still interested in the difference between brown and blue eyes and retains the misleading shorthand of genes "for" such colour differences. Dawkins, in *The Extended Phenotype* (1982), explicitly makes the same point, before going on to discount it as irrelevant, provided the system behaves as if such "genes for" existed. That is, his genes are purely theoretical constructs, combinations of properties which may or may not be embedded in specific enzymes or lengths of DNA, but which can be used to play mathematical modelling games. Sloppy terminology abets sloppy thinking. And it has implications for gene technology too. As more is learned about the human genome, so early simplicities, such as the existence of a single gene responsible "for" a particular disease, retreat. Many ostensibly "single gene disorders" are now known to result from different gene mutations in different people. All may show a similar clinical picture but the gene mutation and hence enzyme malfunction which results in the disorder may be very different in each case. This also means that drugs effective in ameliorating the condition in one person may be simply ineffective in another.

The history of how genes became DNA, culminating in the famous *Nature* papers of Crick, Watson, Franklin, and Wilkins et al. in 1953 is too well known to need retelling. But what made a length of DNA a gene? Genes were now coded for polypeptides, and the 1960s saw the breaking of this code and the formulation of Crick's Central Dogma:

$$\text{DNA} \rightarrow \text{RNA} \rightarrow \text{protein}$$

or, as Crick put it, "once 'information' has passed into the protein it cannot get out again."

A formulation which is as central to ultra-Darwinian theory as it is to molecular biology, DNA had become the master-molecule, and the nucleus in which it was located had assumed its patriarchal role in relationship to the rest of the cell. It is hard to know which had more impact on the future directions of biology, the determination of the role of DNA in protein synthesis, or the organising power of the metaphor within which it was framed (e.g., Dawkins's [1986] description of willow seeds as floppy discs).

Periods of great unifying simplicity in science are fre-

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quently followed by times in which simplicity dissolves once more into complexity. Not all DNA is coding; much is concerned with regulation (operons, etc.). And much more seems "junk" or "selfish" – Crick's term. (Note that Crick's DNA's selfishness is demonstrated by the fact that it does not do anything for the cell or the organism in which it is embedded; it simply allows itself to be copied. Dawkins's selfish genes on the other hand are so because they specifically aid the successful reproduction of the organism which contains them and hence their own replication.) Nor are the coding sequences for any particular polypeptide aligned along the DNA; they are separated by introns, and can be spliced, edited, read in different ways. The result is that far from being able to speak of one gene/one protein, both genes and proteins are disarticulated. Genes can be assembled from alternative pieces of DNA or rearranged so that their codes are read differently. And proteins take on multiple forms as a result of cellular processes distal to DNA itself. "Genes" are no longer coterminous with DNA-beads-on-a-chromosome. Nor are even these segments stably located on the chromosome; as McClintock showed, genes could jump. Hence the modern concept of the fluid genome.

Far from being isolated in the cell nucleus, magisterially issuing orders by which the rest of the cell is commanded, genes, of which the phenotypic expression lies in lengths of DNA distributed along chromosomes, are in constant dynamic exchange with their cellular environment. The gene as a unit determinant of a character remains a convenient Mendelian abstraction, suitable for armchair theorists and computer modellers with digital mindsets. The gene as an active participant in the cellular orchestra in any individual's lifeline is a very different proposition.

DNA is a stable molecule; what brings it to life is the cellular environment in which it is embedded. Genetic theorists have been misled by the metaphors that Crick provided in describing DNA (and RNA) as "self-replicating" molecules or replicators, as if they could do it all by themselves. But they aren't and they can't. Replication isn't an inevitable chemical mechanism. Copying requires the precursor molecules – which themselves must be synthesized – enzymes to unwind the two DNA strands, and others to insert the new nucleotides in place and zip them up again. And the whole process requires energy. Further, the histones surrounding the relevant region or regions of the double helix must be unwrapped, the DNA strands separated, enzymes must transcribe the "sense" strand into its matched length of RNA, individual RNA lengths spliced, edited, and further manipulated in the cell nucleus, and if and only if so permitted, leave the nucleus to be inserted into the copying machinery in the cell cytoplasm. Without the complex biochemical environment the cell provides, "genes" in the DNA sense of the term simply cannot function.

This is why an individual's lifeline requires more than merely the mixing of parental DNAs at the moment of fertilisation. Sperm provide only DNA. But an egg contains more than just the maternal complement of DNA to match that provided by the paternal sperm. It has in addition all the cellular apparatus required to bring both sets of the DNA together to play their part in the cellular orchestra, as well as the mitochondria with their own independent DNA. From this moment of conception on, the maternal cellular machinery is responsible for directing the activation of particular genes (DNA-sequences) and hence the synthesis of specific proteins. These proteins in turn include some whose function is to act as switches, regulators to turn on, and in due course turn off, other DNA-sequences. A continuous cycle of synthetic activity begins in which DNA-sequences are uncovered, transcribed into RNA, processed, spliced, edited, translated into proteins which then provide feedback control to the DNA, perhaps switching off their own synthesis, perhaps switching on the synthesis of other proteins by uncovering other DNA-sequences or influencing the splicing and editing steps. This exquisitely timed and subtly orchestrated cellular symphony culminates in due course in the synthesis of those proteins which begin the process of replicating and segregating the chromosomes once more, enabling the cell to divide and the cycle to recommence.

In the digital information metaphor all these cellular mechanisms are dumb, because they do not carry "information." But it is the cellular machinery which times and edits the synthetic processes. Insofar as the information metaphor is valid at all, it can be expressed only in the dynamic interaction between the DNA and the cellular system in which it is embedded. Cells make their own lifelines.

Thus in both the Mendelian and the biochemical senses genes are only partially determinate entities within genomes. How, when, and to what extent any gene is expressed – that is, how its sequence is translated into a functioning protein – depends on signals from the cell in which it is embedded, and, as this cell is itself at any one time in receipt of and responding to signals not just from a single gene but from many others which are simultaneously switched on or off, the expression of any single gene is influenced by what is happening in the whole of the rest of the genome.

When we talk about the development of an organism as being a product of the interaction of genes and environment therefore, the phrase masks as much as it reveals. Neither "gene" nor "environment" is an unproblematic term. A "gene" as abstract determinant is quite different from the complex processing mechanisms which put together particular DNA sequences which define the primary sequences of proteins. Nor are proteins merely defined as their primary sequences; they have complex secondary and tertiary structures which depend not just on their amino acid sequence but on their environment, on the presence of water, ions, and sometimes other small molecules, on acidity and alkalinity. The path from primary structure to fully fledged protein does not involve as many regulatory steps as that from DNA to protein, but it contains orders of complexity which move us yet further away from the one gene – one protein heuristic. And as proteins themselves become assembled into higher order structures within the cell, yet further constraints come into play.

The school textbooks which start with Mendel and his ratios have it wrong. Without Mendel, genetics would never have got off to such a start and he deserves honour for his experiments. But the founders of a field, by choosing experimental systems which seem to give clear-cut answers, often also produce an appearence of simplicity which is ultimately misleading. The famous and paradigmatic Mendelian ratios are the results of rather special cases, the phenotypic expressions of enzyme pathways rather little influenced by environmental circumstance, perhaps just because they reflect relatively trivial features of that phenotype. By contrast, the expression of most genes is modi-

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fied at several levels. It is affected by which other genes are present in the genome of the particular organism, by the cellular environment, the extracellular environment, and, in the case of multicellular organisms, by the extra-organismic environment. Example: the ambiguous consequences of knockout mutations. In many cases in which genes coding for proteins which are supposed to have vital functions within the cellular economy have been deleted, the absence both of the gene and of the protein whose synthesis it codes for seem to make little observable difference to the life of the animal. It has "no phenotype." This does not mean that the protein concerned does not play a vital role in the cellular economy; rather it is a demonstration of the power of developmental plasticity, of functional redundancy in the organism. Redundancy assists stability; it means that there may be many alternative routes that the cell and the organism can adopt during development which can lead to an essentially identical end-point. In the presence of a particular gene and protein, one route is adopted, and in their absence another is taken. Once again, there is no necessary linear path between gene and organism. Such plasticity is not infinite; there are sharp limits to the tolerance of any gene – or any phenotype – to environmental change. Outside these limits, the response is to curl up and die. But within them, the expression of any gene may be defined in terms of Dobzhansky's concept of norm of reaction – rather out of fashion amongst today's theorists who prefer a modern version of preformationism, in which genes are prime movers.

Chapter 6: Lifelines

At the heart of modern biology lies the issue of the nature of individual living units – organisms. Their lifelines may range over many orders of magnitude in both time and space. Some arise essentially fully developed, like a newly budded yeast, others grow to a reasonably stable adulthood before ageing and decaying, others grow incrementally throughout their lives. Yet others go through a series of radical transformations in which entire body plans become reconstructed, as when egg becomes caterpillar becomes chrysalis becomes butterfly. Life persists not in three but in four dimensions – a persistence, which is above all dependent upon the maintenance of order: order within the cell, order within the organism, and order in the relationship of the organism to the world outside it. Genes and genomes neither contain the future of the organism, nor are they to be regarded, as in modern metaphors, as architects' blueprints or information theorists' codebearers. They are no more and no less than an essential part of the toolkit with and by which organisms construct their own futures.

Neither cells nor organisms can be considered in isolation from their own external environments. The boundary between cell and environment is its semi-permeable membrane across which all trafficking must occur. For single-celled organisms, the environment of the cell is obviously also that of the organism, the ever fluctuating external world, inherently patchy. Some regions may be antithetical to survival – too hot, too dry, too acid – some may be rich in food sources, others poor. Faced with such patchiness, many single-celled organisms can take steps to seek out more favourable conditions (e.g., cilia and flagella); but their power to choose a favourable environment is limited by the range of environments available, and survival will also depend on the ability of the organism to adapt to less than optimum conditions. (Hence the operon, which is the mechanism whereby the organism in interaction with its environment determines which of its available genes are to be active at any one time.)

Such interactions between cells and environments become more complex for a multicellular organisms. Buffered by a regulated internal environment, cells no longer need the operon mechanism but instead lose their autonomy within the greater unity of the organism. They have surrendered their capacity for replication and their totipotency. They become specialised, as liver or brain, leaf or root. In the course of this specialisation, as ontogeny proceeds, particular DNA-sequences are switched on or off in defined temporal sequences. It is no longer only a case of proceeding through the cell cycle to division, but of establishing cells with an appropriate structure, shape, and pattern of enzymes to function as part of a particular organ. To ensure harmony at a multicellular rather than a cellular level, each cell has to be able to respond to the presence of its neighbours and to signals from distant parts of the organism (hence hormones, transmitters, modulators, etc.) arriving at its membrane surface. The cellular lifeline has become subordinated to that of the organism.

Like the term "gene," the term "environment" is thus complex and many-layered. For individual gene-sized sequences of DNA, the environment is constituted by the rest of the genome and the cellular machinery in which it is embedded; for the cell, the buffered milieu in which it floats; for the organism, the external physical, living, and social worlds. Which features of the external world constitute "the environment" differ from species to species; every organism thus has an environment tailored to its needs. Even for the individual gene, the genomic background against which it is expressed differs during the cell cycle as other genes are switched on and off. Outside the organism, change is virtually the only constancy. Stasis is death.

Boundaries between organism and environment are not fixed. Organisms are constantly absorbing parts of their environment into themselves as food, and as constantly modifying their surroundings by working on them, by excreting waste products, or by modifying the world to suit their needs, from birds' nests to beaver dams and termite mounds. Organisms and environment interpenetrate. Abstracting an organism from its environment, ignoring this dialectic of interpenetration, is a reductionist step which methodology may demand but which will always mislead. Nor are organisms passive responders to their environments. They actively choose, and work to change them.

The first phases of the life cycle are those of development. From the moment of fertilisation, cells grow, divide, and hence multiply. Daughter cells begin to align themselves with respect one to the other, to migrate to specific regions within the developing embryo. Within each cell, particular genes are switched on, others off, in intricate sequences, as originally totipotent cells become specialised and the mature form of the organism unrolls from its undifferentiated state. From very early in their development, organisms have to be capable simultaneously of quasi-independent existence and of growing further towards maturity. Moreover, the properties that enable them at any one moment to maintain their existence are not always merely miniature forms of those they will need in adulthood. This is obvious for some life forms. Frogs' eggs become tadpoles become frogs. Each

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stage requires a radical transformation of body plan, yet one during which the functions necessary to life must be preserved. But it is also true in quite subtle ways for organisms which seem to show linear developmental trajectories. When a newborn baby suckles at its mother's breast, the suckling reflex is not simply an undeveloped form of the chewing technique that will be needed when the child switches to solid food; quite different neural and mechanical processes are involved. Life demands of all its forms that they are able simultaneously to be and to become.

The unrolling processes of development are best understood in terms of specificity and plasticity. Many ontogenetic processes are relatively unmodifiable by experience (e.g., relatively fixed development of the visual system). But plasticity is also necessary (e.g., alterations to visual cortex neuronal connectivity dependent on rearing environment). Specificity and plasticity are embedded properties of the organism; both are completely made possible by the genes, and completely made possible by the environment. They cannot be partitioned.

Two contrasting metaphors have been used to describe the process by which multicellular organisms develop: selection and instruction. Consider the human brain, with its highly ordered pattern of characteristically shaped and located neurons. From conception to birth requires the generation of about a million cells an hour, whilst during postnatal development some 30,000 synapses a second will be created under each square centimetre of cortex, until the full complement is present. Development requires the ordered birth and migration of these cells. To arrive at the correct target site both distant and local signals must be involved (e.g., role of trophic factors). However, the migrating cells or growing axons also need to keep in step with one another; each has to know who its neighbours are. The diffusion of a local gradient molecule, together with the presence of some type of chemosensors on the axon surface, could enable each to determine whether it has neigbours to its right and left and to maintain step with them.

Part of this process – the achievement of long-range order – is compatible with a cooperative, instructionist model, but the overproduction of neurons and synapses implies also ultimate selection amongst cells or connections competing for targets. Selection in this sense can account for local but not distant processes. Instructive and selective mechanisms are only part of the picture of development. The maintenance of stable order requires the collective, cooperative properties of the entire ensemble of cells. Each depends on the others in the creation and preservation of the dynamic pattern of connections which impose new patterns on the world beyond the organism. Development is essentially a constructivist process; the developing organism, in its being and its becoming, in its specificity and its plasticity, constructs its own future.

Even the constructivist model of development discussed above however implies a degree of determinism, albeit a richer concept than that of the unidimensional gene. But we need to go beyond this in emphasising the role of chance, of contingency, at all levels of analysis of living systems. Consider the microlevel of the individual cell and its subcellular components. Biochemists deal not with individual cells or molecules but with aggregates of millions, and on this scale properties become relatively predictable. But what is predictable for the mass does not apply to the individual (e.g., there are only about 30 hydrogen ions in any single mitochondrion). Chance and contingency affect all cellular processes (e.g., numbers of bristles on *Drosophila* legs; different foetal circumstances for identical twins depending on placental relationships).

Organisms are supposed to maintain homeostasis. But in fact the set points around which conditions fluctuate are not constant but vary momentarily, diurnally, monthly, and over a lifetime. Furthermore, they are maintained dynamically, not statically. Hence homeodynamics. Seeing organisms as merely homeostatic is to deny them lifelines. Each of our presents is shaped by and can only be understood by our pasts, our personal, unique, developmental history as an organism. Even the moment-to-moment stability of the organism is maintained not statically but dynamically; molecules and cells turn over on timescales varying from minutes to months. Why this ceaseless flux?

The answer is simple: living systems need to be dynamic to survive, able to adjust themselves to the fluctuations which, even in the best-buffered internal milieu, their cooperative existence as part of the greater unity of the organism demands. It is to this irreducible dynamism as the generator of stable order that we must turn in order to understand how, having constructed itself through the processes of development, the organism is able to preserve its integrity and act upon the external world. These are the phenomena of autopoiesis, a concept introduced by Maturana and Varela (1980).

Internal cellular stability depends on two features. First, cells and organisms are thermodynamically open systems far from equilibrium, which for their existence depend on a continuous energy flow, generated through the catabolic/anabolic cycle which results in the synthesis of ATP, ultimately through the activities of autotrophic organisms (primarily photosynthesis). Equations relevant to closed equilibrium systems are largely inapplicable to living processes.

Second, the existence of an interacting metabolic web: individual reactions are catalysed by enzymes; by sequences of reactions, like glycolysis, by chains of enzymes whose overall rates are seemingly controlled by individual enzyme kinetic properties, so that in a chain the slowest reaction becomes the controller. However, just as in a living cell one cannot abstract an individual enzyme reaction from the metabolic dance of the molecules, so one cannot abstract any single reaction pathway. Most of its components are involved in many different reaction pathways, knitted together by multiple interconnections. Once such a web reaches a sufficient degree of complexity, it becomes strong, stable, and capable of resisting change; the stability no longer resides in the individual components, the enzymes, their substrates and products, but in the web itself. The more interconnections, the greater the stability and the less its dependence on any individual component.

Further, the cellular web has a degree of flexibility which permits it to reorganise itself in response to injury or damage. Self-organisation and self-repair are its essential autopoietic properties. These properties of stability and self-organisation are the key to the fundamental irreducibility of living cells. The stability is dynamic, and depends in part on metabolic oscillatory processes (e.g., calcium waves). Metabolic organisation is not merely the sum of cellular parts, and cannot be predicted from individual enzyme reactions and substrate concentrations.

Cells are not simply bags containing semi-random mixes but contain many internal structures (nucleus, mitochon-

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dria, etc). Each represents a separate compartment within which relatively segregated sets of reactions can occur. Communication between these compartments, in terms of exchange of substances and signals between them, takes place through selective membranes, which act as gatekeepers across which specialised signalling molecules and small inorganic ions control access. Homeodynamic order within the cell is thus maintained not merely through the self-stabilising properties of metabolic webs, but through internal structural constraints provided by semi-permeable lipid membranes in which are embedded proteins which recognise and regulate the entry and exit of key metabolites. Ionic changes also modulate the microenvironment within which protein structure is modulated, complex structures such as microtubules and ribosomes can self-assemble, and enzyme-linked reactions occur.

Lifelines, then, are not embedded in genes. Their existence is posited on homeodynamics. Their four dimensions are autopoietically constructed through the interplay of physical forces, the intrinsic chemistry of lipids and proteins, the self-organising and stabilising properties of complex metabolic webs, and the specificity of genes which permit the plasticity of ontogeny. The organism is both the weaver and the pattern it weaves, the choreographer and the dance that is danced.

Chapter 7: Universal Darwinism?

"Darwinism" has become an almost universal – and often abused – term. Darwinian protagonists offer a "tough-minded" ultra-Darwinism as a universal mechanism to explain all phenomena of life. Philosophers follow them; Dennett writes a book entitled *Darwin's Dangerous Idea* (1995) in which Darwinian mechanisms are described as a "universal acid" which eats away at everything it touches. Edelman (1987) interprets the brain processes concerned with experience, memory, and consciousness as representing "neural Darwinism." Hull claims that scientific theories themselves win or lose the struggle for acceptance according to Darwinian mechanisms. One reads of Darwinian psychology, Darwinian medicine, Darwinian economics. Dawkins caps the lot with his claim that human culture itself operates on Darwinian principles in which the units of transmission are not genes but "memes." It may be time to try to rescue Darwin from some of his oversolicitous modern friends if we are to do justice, but no more than justice, to the part he and his ideas have played in the history of biology and in our understanding of living processes.

Before Darwin, the interpretation of life on earth was trapped within a mode of thinking imposed by biblical traditions. Species were immutable, linked in a great chain of being, beginning with the lowliest and ending with that acme of god's creation, humankind (Man) himself. The industrial revolution shattered this stability; change (and evolution means simply change over time) became acceptable. The discovery of fossils implied extinct species, whose history could be dated. Before Charles Darwin, both Erasmus Darwin and Lamarck had sought to describe and account for such changes. It was Charles Darwin who offered the mechanism by way of a simple syllogism:

1. Like breeds like, with variations.
2. Some of these varieties are more favourable (to the breeder or to nature) than others.
3. All creatures produce more offspring than can survive to breed in their turn.
4. The more favoured varieties are more likely to survive long enough to breed.
5. Hence there will be more of the favoured variety in the next generation, and
6. Thus species will tend to evolve over time.

This process is natural selection. As a syllogism it has an inexorable logicality. If 1, 2, and 3 are true, 4, 5, and 6 follow inevitably. This is why philosophers such as Dennett are able to describe it as a universal mechanism. Yet for Darwin there were major theoretical difficulties at the heart of the theory; the mechanism of transmission both of similarities and variations; the classic argument from design; and the problem of speciation.

Darwin's achievement was to demolish the idea of the immutability of species and, even more important, of a great chain of being. Humans are no longer at the pinnacle of life. Instead, living forms can be drawn as related to one another as the branches and twigs of a bush. All of us currently alive, amoebae as well as humans, are in this respect equal, the successful survivors of evolutionary history. There is no scale of life on the basis of which one can judge some currently living forms as "lower" and others as "higher," more or less "evolutionarily successful." A further crucial feature of Darwinism is its insistance on the role of chance. Natural selection abolishes purpose from evolution, and, some felt in consequence, from human life itself.

7.1. Variation. The problem of transmission was resolved with the theory of the gene, although when Mendel was rediscovered at the turn of the century mutational change seemed to replace natural selection, and it was not until the "neo-Darwinian synthesis" of Fisher, Haldane, and Sewall Wright around 1930 that it was seen that the two theories supported rather than contradicted each other.

7.2. Heritability. Fisher's synthesis was directed at trying to understand the contributions of genes and environment to variation in populations. In a uniform environment all the variance would be contributed by the genes, and with identical genes all the variance would be contributed by the environment. But genotypes and environments both vary and the purpose of heritability estimates is to try to tease them apart. To do so however, it is necessary to make some simplifying assumptions. Variance describes the way in which any particular measure of a trait in a population is distributed about the mean value for that population, made up of a component contributed by the genes and a component contributed by the environment, which together can be added to give a total of nearly 100%. The remainder, which to make the mathematics work has to be a rather small proportion, is considered to be the product of an interaction between genes and environment. If genotypes are distributed randomly across environments, it is possible to estimate heritability, which defines the proportion of the variance which is genetically determined. However, the mathematics only works if all the relevant simplifying assumptions are made. If there is a great deal of interaction between genes and environment, that is if genes behave according to Dobzhansky's (1973) vision of norms of reaction, if genes interact with each other, and if the relationships are not linear and additive but interactive, the entire mathematical apparatus of heritability estimates falls apart. Thus

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the meaningful application of heritability estimates is only possible in very special cases, from which the majority of traits of interest outside the special world of artificial selection are likely to escape. Furthermore the figure derived for the heritability is itself dependent on the environment – that is, if you change the environment, the heritability estimate changes.

These caveats perhaps help to explain why heritability estimates have been so persistently misunderstood. They become wholly misleading when applied to aspects of human behaviour. Milk yield is a phenotype which is reasonably straightforward to measure. But intelligence? Political tendency? Likelihood of getting divorced? Religiosity? Job satisfaction? As one cannot treat human populations quite like breeding experiments with wheat or cattle, and distribute genotypes across environments, one has to make do with what between them nature and society provide. The standard techniques have involved comparing traits in siblings, MZ and DZ twins, and the use of adoption studies. The problems are manifold. To mention just two, separated twins tend to be placed in rather similar environments; whilst adoptive parents are unlikely to treat their adoptive child "exactly" as they do a natural one, and are far more likely to be anxiously on the lookout for tendencies which reveal the child to be "taking after" some undesirable character of its natural parent. Such real life problems are simply swept aside in the process of fitting the numbers obtained into the complex statistical manipulations required to generate the seemingly objective heritability estimate.

As a result, seemingly bizarre traits turn out to have "high heritability," for which the most parsimonious explanation is that they demonstrate the inappropriateness of attempting to apply a mathematical formalism devised for plant and animal breeding to such dubious phenotypic characters as the diversity of human social behaviour and attitudes. Yet some behaviour geneticists argue that even such high heritabilities underestimate the true influence of the genes. Bouchard (1997) proposes that our genes "predispose" us to seek environments congenial to the genetic imperatives. Thus genes create environments, and "environment" – whatever that term may mean – ceases to be a truly independent variable in the heritability equations. It is the genes, therefore, which are a major cause of everything from childhood accidents to divorce in midlife, for these genes lead their owners to place themselves in situations in which the probability of accident or divorce increases. Like the claims for the "extended phenotype," this argument perversely swallows the four-dimensional universe of lifelines entirely into the double helix of DNA.

7.3. Adaptation. The argument from design is confronted head-on in Dawkins's *The Blind Watchmaker* and its successors (1986; 1995): "What use is half an eye," he asks, and answers "One percent better than 49% of an eye, and the difference is significant." The trouble with this argument is that there is no way of determining whether, amongst our evolutionary ancestors, 50% of an eye was really significantly better in Darwinian terms – that it contributed significantly more to reproductive success – than 49%. It would depend what other costs the organism accrued in achieving this 1% advantage, and how much having eyes contributed to its success in finding food and avoiding predators so as to increase its chances of finding a mate and hence reproducing. There is of course no evidence on these issues, and so the claim must remain an undemonstrable assertion, although one which most biologists will find reasonably convincing. In the classical Popperian sense, such stories about evolution are unfalsifiable. All that we can do is to offer plausible accounts of how a process may have occurred or a structure evolved.

7.4. Sexual selection. If all adaptation serves the function of enhancing survival, why do so many animals – especially males – have traits which seem on the face of it to be inimical to long and efficient life (e.g., the peacock's tail)? Darwin's interpretation of sexual selection was that females were motivated to choose, and hence select beauty. More modern versions argue instead that seemingly dysfunctional adornments are markers of good health and hence good genes. Whichever version of the theory is adopted, sociobiologists have sought to press it into service to provide an evolutionary "Darwinian" explanation for the preferences expected in human sexual choice, once again treating metaphor as if it were homology. For example, competition amongst human males for mates is discussed as the macroversion of what is said to be the micro-level competition amongst individual sperm to be "the one" to successfully penetrate and fertilise the egg. Males and their sperm compete, females and their ova quiescently await their fate. The problem is that, as with most human extensions of evolutionary mechanisms, but in an even more extreme form, such accounts simply cannot encompass the rich diversity of human experience, and instead have recourse to traditional and often sexist caricatures which ignore the historical and anthropological evidence of variation in social practices across time and space and instead treat current western norms – or rather, assertive restatements of what the authors perceive as those norms, for they show as little respect for sociology as they do for history or anthropology – as if they were human universals.

7.5. Altruism. With the claims for the genetic mechanism and evolutionary significance of altruistic behaviour, we are at the heart of sociobiological thinking. If organisms seek to maximise their reproductive success, then how do we account for birds which on detecting a predator, draw attention to it, and simultaneously to themselves by uttering warning cries? Ought they not instead try to make themselves as inconspicuous as possible, so as to diminish the chance of being picked off? Earlier group-selectionist ideas, once discarded, are creeping back into the literature once more, but the dominant mode of explanation is kin selection, a mathematical formulation which, if one grants its basic assumptions – that is, that living forms exist primarily to perpetuate their genes – is as inevitable a syllogism as the original Darwinian formulation of natural selection. Although I see no reason to doubt the principle, proving it is harder. Certainly, behaviour which might be defined as altruistic does occur amongst group-living animals, although equally there is no shortage of evidence as to competitiveness amongst them. The empirical question is whether apparently altruistic behaviour can be shown to benefit preferentially the kin of the altruist rather than the group as a whole. Perhaps in response to the relative weakness of the data, Trivers (1971) offered an alternative version: reciprocal altruism – an act performed to benefit non-kin, but which is performed in expectation of a subsequent return of the compliment. And once again, human sociobiologists

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have sought, on the slightest of evidence, to deprive human unselfish behaviour of any function other than one of these forms of selectionist altruism, once again reducing metaphor to homology.

7.6. Speciation. It may seem extraordinary, but the Darwinian syllogism provides no mechanism for the formation of new species, which was after all ostensibly what *The origin* was all about. All that is claimed is that in any given circumstances, external conditions (the environment, nature) will favour the perpetuation of varieties which can do their species-thing a bit better than the rest. For example, Kettlewell's peppered moths, which also demonstrate another fundamental point. A "more favoured variety" is one which is favoured under current circumstances. Evolution by natural selection can respond only to the current situation. It cannot predict the future. At one point of the species trajectory in time it is the peppered form which has the greater survival value, at a later time the melanic, and then again the peppered. The environmental change occurs and natural selection trails along behind, following and responding but never leading and never predicting. Such evolutionary processes could obviously modify a species over time to such a degree that its members would no longer be able to mate fertilely with their ancestors, could these be brought back to life. In this sense, species can gradually be transformed through processes of natural selection steadily tracking environmental change. But this still does not explain how natural selection alone can result in one preexisting species splitting into two. For this, additional mechanisms are required; presumably primarily geographical separation and founder effects, of which the most famous example is Darwin's own, the Galapagos finches. But could this be all? For the orthodox ultra-Darwinian, there is nothing else available.

Chapter 8: Beyond ultra-Darwinism

Ultra-Darwinism has a metaphysical foundation: the purpose of life is reproduction of the genes embedded in the "lumbering robots" which constitute living organisms. There follow two premises: (1) the unit of life is an individual gene whose sole activity is to create the conditions for its own reproduction by directing the development and physiological function of the organism; (2) most aspects of the phenotype are adaptive, selected for by the honing force of natural selection. This metaphysic derives from a combination of Hobbes and Smith. Life is a war of all against all, but the invisible hand of the market generates even cooperative behaviour from competitive individualism. A further element is a restatement in scientific form of the theology of preformationism. Our task is to preserve and transmit copies of our DNA.

It follows that the prime function of every living organism is to maximise its inclusive fitness – that is, to ensure the maximal spread of its own and its close relatives' genes in succeeding generations. This determinism worries Dawkins and others, who therefore claim that we have the capacity to rebel against the tyranny of our genes. But if the power to rebel does not itself come from our genes, then the argument implies a sort of Cartesian dualism, and the mechanical materialism of selfish genery trips over into a type of idealism.

The case against ultra-Darwinism rests on the following claims: (1) The individual gene is not the only level at which selection occurs. (2) Natural selection is not the only force driving evolutionary change. (3) Organisms are not indefinitely flexible to change; selection is "table d'hôte" and not "à la carte." (4) Organisms are active players in their own destiny.

1. Any individual gene can only be expressed against the background of the whole of the rest of the genome. Genes produce gene products which in turn influence other genes, switching them on and off, modulating their activity and function. If selection ultimately determines whether a particular gene survives or not, it can only do so in context. A gene is only selected if it results in a selectable phenotypic change – yet what is required to produce such a change is not one but many actual biochemical gene-size lengths of DNA. Furthermore, Weismann's barrier is not as impermeable as implied, so developmental processes also affect transgenerational gene transmission. Waddington argued that developmental processes in multicellular organisms could help to both direct and, as he put it, canalise potentially favourable mutations. Bonner (1974) identified two routes around Weismann's barrier. Thus both plants and such animals as Hydra retain the capacity to differentiate into somatic cells or to become sequestered as gametes or to remain totipotent. Those cells which remain totipotent retain the prospect of becoming gametes after an indefinite number of cell divisions – and this means that any genetic variation occurring during those divisions will be heritable. That totipotency is available even to mammals is now proven by the cloned sheep Dolly and her successors. Further, during development, originally totipotent cells divide, become determined, and migrate to appropriate positions within the developing embryo, resulting in competive/selective mechanisms operating at the cellular level; cellular variation will affect the outcome of such competition.

So selection acts on genes, on genomes, and on cells, notably during development. But for multicellular organisms it is ultimately the organism as an integral unit which will or will not reproduce and hence dispatch copies of its genes into subsequent generations. So natural selection in the sense that Darwin originally conceived it can only occur through the actions and properties of the entire organism, its phenotype. For ultra-Darwinists that is not a problem; the phenotype is merely a proxy for the genes it contains, the gene's way of making copies of itself. But this implies a direct relationship, one-for-one, between gene and phenotype, and this "empty phenotype" view ignores development.

The fact that selection occurs at many levels dampens the effects of change at any one level. For instance, mutation rates in DNA are relatively constant, yet these are not necessarily reflected in phenotypic change (hence Eldredge and Gould's punctuated equilibrium). Furthemore there is a vast amount of hidden phenotypic diversity (e.g., isoenzymes) which is likely to be neutral so far as selection pressures are concerned.

Finally in this context, organisms do not exist in isolation but in populations, and populations in ecological communities involving many hundreds or thousands of different species locked into relationships which may be competitive or cooperative. Thus within species, evolutionary stable strategies (Maynard Smith) mean that any individual's character may or may not be selectively favourable, depending on the balance between that individual and others in the

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population, whilst the fact that any species' niche is defined in the context of all other species within an ecosystem (predator or prey, mutualistic or commensal, or even, in Margulis's term, symbiogenic, like mitochondria) and that all species are evolving, means that the evolution of any one is shaped and constrained by that of many others. Evolution means coevolution, built on both competitive and cooperative mechanisms.

2. Selection mechanisms include competition for scarce resources, sexual and kin selection, founder effects, expansion of populations into new niches, selective predation, and coevolution of populations and species. Selection at any given level of the hierarchy between individual genes and ecosystems does not automatically imply selection and evolutionary change at any other; there is sufficient flexibility and redundancy within living systems to make such tight coupling unnecessary. But not all phenotypic variation is adaptive, and not all change is selective. Contingency also applies (e.g., Gould's [1989] account of the Burgess shale fossils; dinosaur extinction following climate change). And what constitutes an adaptation is itself problematic, and evolutionary accounts become Panglossian Just-So stories (e.g., flamingo plumage as pink to confuse predators rather than as a consequence of their shrimp diet). Gould and Lewontin (1975) used the famous spandrels of San Marco as an example of how adaptationist assumptions could be misleading; their account has been criticised by Dennett (1995); however, architectural reassessment of the role of spandrels (pendentives) favours the original interpretation.

3. Within the adaptationist programme, the trajectory that any lifeline can take is ultimately limited only by the question of whether it is adaptive. Of course, evolution is cumulative and has to build on whatever materials it has at hand. So to arrive at any adaptive structure, behaviour, or molecular property there has to be a legitimate route from where the present state of the system is to some presumably more adapted place elsewhere. And these constraints determine what is or is not evolutionarily possible (e.g., limits on the size of single cells, and of multicellular organisms – surface area/volume and mass relationships, skeletal properties, etc., set by chemistry and physics). But there are deeper prospects opened by discussion of "laws of form" (e.g., Darcy Thompson's topological transformations; radiolaria crystalline forms, pentadactyl limbs; pinecone patterns in Fibonacci series; the role of morphogenetic fields).

4. The metaphor of natural selection is one in which nature sets a series of challenges which organisms either pass, in which case they are privileged enough to pass copies of their genes on to a successor generation, or they fail. By contrast, the autopoietic vision implies that organisms actively choose and transform their environments, to adjust and appropriate them to their own ends (e.g., unicells moving to food rich environments, growing axons finding and modifying targets, etc.). Organisms change environments and environments have their own trajectories constantly transformed, not merely by the workings out of the inanimate forces of weather, temperature, and cosmic history, but above all by the interactions of myriad lifeforms.

Chapter 9: Origin myths

Where once the definition of being alive was to be a breathing, metabolising, environment-sensing and responding organism, molecular biologists tend to see the basic function of life as the power to replicate, and the basic unit of life is therefore a molecule with this power, a naked nucleic acid polymer. There are religious undertones to his view that in the beginning was the word. But could life have begun with a naked molecule of DNA or RNA? So-called self-replicating experimental procedures are already quite complex. They must occur in a test-tube which serves as a surrogate cell, including all the necessary mix of enzymes, ions, and controlled temperatures. It follows that accurate replication could only have emerged long after the development of cell-like structures capable of such crucial living processes as metabolism, growth, and division. What characterises all living organisms, including ancient fossils, is the presence of a cell membrane, and such cells must precede replicators.

Origin of life theorists and experiments have shown how abiotic synthesis of amino acids and other organic precursors is possible. The biochemical parsimony which characterises modern living forms suggests that these substances must have appeared prior to their organisation into replicating organisms. Life consists primarily of arrangements of the elements carbon, hydrogen, oxygen, and nitrogen, together with smaller quantities of phosphorus and sulphur, ions of calcium, magnesium, sodium, potassium, and some heavy metals. Compounds of these most abundant elements are thermodynamically unstable but capable of relatively long life in watery solution; their synthetic chemistry requires energy but they trap energy easily in the form of sugars; they readily combine to form long chain molecules, lipids, polysaccharides, proteins, and nucleic acids, especially in a reducing atmosphere (Miller-Urey experiment). The earth's early atmosphere provided this environment; it is life itself which has subsequently modified it by trapping carbon and releasing oxygen. Oparin and Haldane proposed that droplets of concentrated organic chemicals could concentrate out of a prebiotic soup (coacervate drops). Other concentration mechanisms could include clay surfaces. Lipids spontaneously form micelles and droplets surrounded by bimolecular membranes, within which the soup would be concentrated. These represent protocells. Such cells would have another property seemingly fundamental to life in that there would be an electrochemical gradient across their membranes.

The next evolutionary step would be to stabilise the myriad potential chemical reactions that could occur within the proto-cells. Kauffman's models suggest that, given enough metabolic interactions, in due course catalytic and autocatalytic relationships would arise, especially granted the possibility of catalytic surfaces such as that provided by clay. Computer models of such processes show that a random set of chemicals in a constrained area soon settle into a robust and autopoietic metabolic web in which stable balances of constituents result. Traffic across the liposome membrane will bring new materials into the cell and excrete waste products, and cells which increase in size will simply split into two.

Thus cell formation and division, and sophisticated metabolic stability, have all been achieved by originally abiotic processes, in which the properties which are characteristic of life are embedded not in a single molecular substituent but in the entire system which constitutes the cell, and without replicators. The metabolic web must have extended beyond any individual proto-cell to embrace the en-

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tire population. If chemicals were to be exchanged between cells, by ingestion or by cell division, the reactions within each cell must have tended to converge.

Even before the problem of accurate replication had been resolved, there would have been another more pressing problem, that of energy. An evolutionary bottleneck would have been produced until the energy-generation problem could be solved. Early chemoautotrophes would in due course have been replaced by photosynthesizing organisms, which in turn were responsible for changing the earth's atmosphere (and could later be embraced within more complex organisms, as chloroplasts, by symbiogenesis). Only after the development of effective energy-generating and utilising mechanisms, though presumably before the development of the modern cellular systems, would it be possible for nucleic acid based replication – probably initially RNA – to emerge.

Once these molecules had been incorporated within the metabolic web of the cell, they would offer a whole new range of properties. For they would now achieve a level of fidelity in copying and reproduction which would have been unobtainable by mere random division without them. Just how this mechanism settled down into its present day form, based as it is on the trinity of DNA, RNA, and protein, is a matter of intense speculation. Contingency, rather than laws of molecular form or adaptation, may rule at this point in the story. But once a particular set of nucleic acid-amino acid correspondences had emerged, convergence within the web would be likely to help ensure its universality. In any event, the essential point is that once cells containing these mechanisms had arisen, they would multiply rapidly and swamp out all others, as only they could produce exact copies of themselves. Evolution, having generated nucleotide polymers within the primitive cells, had now also produced a mechanism which could be relied upon to amplify them, and before long to conquer the earth – yet another reason why whatever the processes by which life forms were first generated, so far as life on this planet is concerned, they cannot repeat themselves.

Chapter 10: The poverty of reductionism

This chapter shifts gears, to focus on reductionism as ideology in human affairs. The primacy given to reductionist explanations of human behaviour leads to claims, made by scientists but trumpeted by the media, that the origins of everything from sexual orientation to violence, criminality, and "compulsive shopping" lie in the genes. Neurogenetics claims to be able to answer the question of where, in a world full of individual pain and social disorder, we should look not merely to explain but even more potently to change our condition. Although this is not a new debate, the apparent power of modern genetics gives it new force, and the ideologues of neurogenetic determinism claim that their science will in due course render sociology, economics, and even philosophy, redundant.

Neurogenetic determinism is based on a faulty reductive sequence whose steps include: reification; arbitrary agglomeration; improper quantification; belief in statistical "normality"; spurious localisation; misplaced causality; dichotomous partitioning between genetic and environmental causes; and confounding metaphor with homology. The issue at stake is the appropriate level of organisation of matter at which to seek causally effective determinants of the behaviour of individuals and societies. The structure of the argument is similar whether the discussion focusses on intelligence, sexuality, or violence.

Reification converts a dynamic process into a static phenomenon. For example, violence as an interaction between people becomes "aggressive behaviour," regarded as the property of an individual.

Arbitrary agglomeration lumps together many different reified interactions as if they were all exemplars of the one character. Thus aggression becomes the term used to describe processes as disparate as a man abusing his lover or child, fights between football fans, strikers resisting police, racist attacks on ethnic minorities, and civil and national wars as if in fact they were all examples of the same underlying mechanism of "anti-social behaviour" (e.g., the claim that a genetic abnormality in monoamine oxidase predisposes to variants of these activities). Yet the identical act performed in different contexts (e.g., war or peace) may be socially desirable or undesirable (e.g., British troops in Northern Ireland).

Improper quantification argues that reified and agglomerated characters can be given numerical values (e.g., the IQ scale, which reifies "intelligence," agglomerates many different processes within the term, and then claims to be able to provide a single number which defines where an individual lies in the intelligence hierarchy). Belief in statistical normality then assumes that in any given population the distribution of such behavioural scores takes a Gaussian, normal distribution. Yet such curves are a product of the test design. There is no biological necessity for such a unidimensional distribution, nor for one in which the population shows such a convenient spread. Yet the power of this reified statistic is that it conflates two different concepts of "normality"; it has normative implications, but also to lie more than two standard deviations from the mean is to be "abnormal" (e.g., the bell curve).

Having reified processes into objects and arbitrarily quantified them, the reified object ceases to be a property even of the individual, but instead becomes that of a part of the person (e.g., schizophrenic brains, genes – or even urine – rather than of brains, genes, or urine derived from a person diagnosed as suffering from schizophrenia). This shorthand of "gay brains" or "selfish genes" does more than merely sell books for their scientific authors; it both reflects and endorses the modes of thought and explanation that constitute neurogenetic determinism, for it disarticulates the complex properties of individuals into isolated and localised lumps of biology.

Misplaced causation is involved in aggressive encounters when people show dramatic changes in, for instance, the levels of circulating steroid hormones and adrenalin in their bloodstream and the release of neurotransmitters in their brains, all of which can be affected by drug treatments. A person whose life history includes many such encounters is likely to show lasting differences in a variety of brain and body markers. But to describe such changes as if they were the causes of particular behaviours is to mistake correlation or even consequence for cause. Drugs such as ritalin may make children more tractable at school; the cause of their so-called attention deficit hyperactivity disorder is unlikely to be too little amphetamine in their brains. The search for "first causes" seems to lead inevitably to genes.

Confounding metaphor with homology occurs as follows.

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If first causes are genetic, then they must have evolved and similar behaviours should be found in non-human animals. Example: "aggression," measured in rats by noting how long they take to kill mice, and taking this as a surrogate for "violent behaviour" in humans.

Reductionist ideology not only hinders biologists from thinking adequately about the phenomena we wish to understand, it has two important social consequences: it serves to relocate social problems to the individual, thus "blaming the victim" rather than exploring the societal roots and determinants of a phenomenon; and second, it diverts attention and funding from the social to the molecular (e.g., alcoholism research in the United States or Russia). For any aspect of the living world, multiple forms of explanation are possible. But for any such phenomenon there are also determining levels of explanation – those which most clearly account for the specificity of the phenomenon and also point to potential sites of intervention into it. Effective science requires a better recognition of determining explanation and hence the determining level at which to intervene. Failing this it becomes a waste of human ingenuity and resources, a powerful ideological strategy of victim-blaming and a distraction from the real tasks that both science and society require.

Chapter 11: Making biology whole again

This chapter summarises the main arguments of the book in the form of ten slogans, as follows:

1. Our history shapes our knowledge
2. One world, many ways of knowing
3. Levels of organisation
4. It all depends
5. Being and becoming
6. Stability through dynamics
7. Organism and environment interpenetrate
8. Structure constrains evolution
9. The past is the key to the present
10. Life constructs its own future.

To conclude: for humans as for all other living organisms, the future is radically unpredictable. This means that we have the ability to construct our own futures, albeit in circumstances not of our own choosing. And that it is therefore our biology that makes us free.

NOTE

1. *Lifelines: Biology, freedom, determinism (1997)* was published by Allen Lane, The Penguin Press in the United Kingdom in September 1997, and three months later by Oxford University Press in New York as *Lifelines: Biology beyond determinism.*

Open Peer Commentary

Commentary submitted by the qualified professional readership of this journal will be considered for publication in a later issue as Continuing Commentary on this article. Integrative overviews and syntheses are especially encouraged.

Lifelines *appeared in two different editions, with some minor differences in wording. Quotations are from the Oxford University Press edition unless it is specifically indicated that they are from the Penguin edition or the précis.*

The myth of genetic determinism – again

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Abstract: *Lifelines* mounts a vigorous attack on sociobiology on the utterly mistaken grounds that sociobiologists believe that genes single-handedly determine social behavior. The many previously published rebuttals to this pernicious criticism are conveniently ignored by the author.

In an undeservedly gentle review of *Not in our genes,* an earlier ideological polemic against sociobiology by Steven Rose (Lewontin et al. 1984), Patrick Bateson wrote,

> The issues of evolution, individual development and current function are not the same. The majority of scientists who now call themselves sociobiologists know it and say so. Therefore, to criticize the evolutionary and functional arguments of these people on the grounds that they are genetic determinists is to make precisely the mistake that [E. O.] Wilson made, treating different problems as if they were the same. Doubtless, it spoils the fun when the hate-object ceases to be hateful, but Rose, Kamin and Lewontin had a duty to deep themselves better informed about current developments in the subject they chose to attack so freely. (Bateson 1985)

What was true then is even truer now. But for Steven Rose to have troubled himself to learn what sociobiology is really about would have definitely spoiled his fun, eliminating the "need" to write his current book, *Lifelines,* which stridently covers much the same ground as *Not in our genes.* Once again, the pre-eminent charge is that sociobiologists believe in rigid genetic determinism in which possession of a given gene guarantees the development of a particular phenotype. Rose is keen to portray sociobiologists as drooling determinists because he wishes to demolish the discipline by showing that there is no simple one-to-one relationship between gene and behavior. Rose makes this point at great length, but his "achievement" is irrelevant, not only because sociobiologists are as aware as he of the complexity of development, but also because sociobiologists leave it to others to study how genes and environment interact during behavioral development. As Bateson noted, sociobiologists deal with the evolution and current function of complex social traits. Like all evolutionary biologists who study phenotypes, they simply assume that some genetic *differences* among individuals have the potential to cause differences in phenotypic development an assumption universally accepted in biology.

A naive reader of *Lifelines* would never guess that sociobiologists have responded fully and often to the genetic determinist epithet (e.g., Daly & Wilson 1987; Maynard Smith 1997). Rose, however, regularly repeats shopworn charges without letting his readers know that the accused have had something to say about these accusations. For example, he repeats Gould and Lewontin's old canards that sociobiologists believe that (1) natural selection is the only process affecting evolution, (2) every trait is an adaptation, and (3) every half-baked speculation about the possible adaptive value of a given trait is true. He does not mention a single rebuttal (for starters, see Borgia 1994; Endler 1986). And he

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indulges in Gould and Lewontin's penchant for aggressive ridicule, producing an unnamed adaptationist who supposedly has said that the pink legs of flamingos provide adaptive camouflage for the birds when flying at sunset.

What Richard Alexander had to say about Gould and Lewontin's use of the "just-so story" applies with equal strength here.

> Those . . . who parade the worst examples of argument and investigation with the apparent purpose of making all efforts at human self-analysis seem silly and trivial, I see as dangerously close to being ideologues at least as worrisome as those they malign. I cannot avoid the impression that their purpose is not to enlighten but to play upon the uneasiness of those for whom the approach of evolutionary biology is alien and disquieting. (Alexander 1987)

That Rose has little interest in enlightened debate is apparent on most pages of the book. For example, his dismissive treatment of heritability studies of humans is based largely on ridicule and dated criticism, such as the notion that separated twins are probably placed in similar environments. His readers never learn that behavioral geneticists replied to this methodological criticism long ago (e.g., Bouchard 1983).

Having trashed behavioral genetics and sociobiology, what does Rose put in their place? In 1984, it was *dialectical biology.* In 1998, we are offered *homeodynamism* and *autopoiesis,* two impressive examples of obfuscatory jargon apparently intended to demonstrate that development is a very complex process indeed. When Rose says, "it is in the nature of living systems to be radically indeterminate," he means that we still do not know exactly how development occurs. He embraces our current (and presumably temporary) ignorance about some elements of proximate causality as evidence that we truly have the unbridled capacity to control our destiny.

Although a "philosophy" of this sort may appeal to those with New Age sensibilities, other readers will note that Rose avoids even one detailed scientific test of an autopoietic hypothesis matched against a sociobiological alternative. Were he to have attempted such a test, Rose might have seen that proximate, developmental hypotheses cannot substitute for those concerned with the ultimate, evolutionary causes of behavior. This would have been a useful lesson to convey to his readers, but instead Rose rails against a mythical genetic determinism because he finds it easier to debate a strawman than a sociobiologist.

The science of life as seen through Rose-coloured glasses

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Abstract: This commentary takes issue with two of Rose's central themes from the perspective of the psychology of intelligence. In the case of *reductionism,* I argue that Rose fails to live up to his own rhetoric by claiming a veto from his own discipline (biology) over facts of the matter in another (psychology). In the case of "*Lifelines,*" Rose's argument is contradicted by evidence from both individual differences and developmental change in intelligence.

Steven Rose is grappling with issues in biology that have long been a concern in psychology. My review will focus on two of them – reductionism, and the necessity for a developmental perspective (lifelines) for an adequate biological or behavioural science – to show how palpably the book fails to live up to its own rhetoric.

Reductionism. I can agree with Rose that the excitement of developments in molecular biology does on occasion threaten to overwhelm us with arrant and simplistic nonsense – his example of statements about "genes for homelessness" is sufficient to make this point. Yet, while arguing that scientific understanding of complex phenomena can involve a number of different levels of description and while presenting a conventional taxonomy of these levels on page 9, at the bottom of the very same page Rose asserts that "some properties of living systems are not quantifiable, and attempts to put numbers on them produce only mystification (as, for instance, with attempts to score intelligence or aggression)." Whether intelligence is quantifiable or not depends on whether we have a theory that allows for the quantification of its constructs. The arbiter of the possibility of quantification is a psychological *theory,* because intelligence is a *psychological* construct and is not something that a *biologist* can decide by fiat. Again on page 69 (during an embarrassing attempt to save his research program from the logic of his own position), we are told that despite the complexity of life the scientific method is at least capable of falsifying some hypotheses, among them that "the Earth is flat, the Moon is made of green cheese, or that IQ tests measure some fixed, biologically determined feature of an individual." But exactly which science has adjudicated this last point? Certainly not psychological science, because this is a proposition that is the centre of a lively current debate. I can only assume that Rose believes that some biological fact establishes this psychological fact. I would like to know what that fact is. How genes relate to DNA, how DNA controls the synthesis of proteins, how sensitive gene expression is to the environment of the cell, or whatever – all of this is of no demonstrable significance to whether there is such a thing as "general intelligence." To think otherwise is to be a reductionist.

Lifelines. Rose uses two related arguments to deride the scientific utility of behaviour genetics. The first is the necessity of a developmental perspective (I have no problem with this) and the second is the indivisibility of the effects of nature and nurture at the level of an individual lifeline. The second is supposed to be a consequence of the first, but I believe it is a red herring. Rose argues that each individual organism can be understood only in terms of its own developmental history. But what science would ever attempt such a thing? Taken to its extreme, science seen through Rose-coloured glasses is the science of Freddo the Frog, and Freda the Frog, and son of Freddo, and so on to near infinity. In psychology it has long been recognized that there is indeed a tension between nomothetic and idiographic approaches (the first is the search for general laws of psychology and the second is the attempt to understand the functioning of specific individuals). A complete psychological science must surely accommodate both (see Smith & Tsimpli 1995 for just such an attempt in the case of an analysis of the abilities of a linguistic savant set against the background of general theories of intelligence). Seen in this light, behaviour genetics (in the nomothetic tradition) is but one method of determining the general phenomena that a psychological theory must accommodate – in this case whether a theory of intelligence needs to take into account the fact that differences and developmental changes in intelligence are influenced by genetic differences. Estimating heritabilities is just the behaviour geneticist's specialized method for estimating what in the truly experimental sciences is called "effect size." The basic scientific logic is the same: Do genetic differences (the "experimental" variable) contribute to measured differences in intellectual performance? This is not a silly or misguided question, and all who make serious attempts to answer it know that it is nonsensical to think we can subsequently calculate what proportion of individuals' intelligence is due to their genes and how much is due to their environment. So this critique is a red herring and perhaps represents posturing on Rose's part, because the answer to this sensible question militates against his own reductionist and *a priori* position. For despite the myriad potential interactions between DNA, RNA, "genes," processes of embryogenesis, cellular and extracellular environments (and whatever else), it turns out that identical twins reared apart are so alike in intellectual performance that measuring two identical twins is just about indistinguishable from measuring the same twin twice. So much, then, for lifelines. Not being a reductionist, I see no conflict between what Rose claims is true for studying the biology of organisms, that is, the hegemony

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of individual lifelines, and what appears to be true for studying and understanding individual differences and changes in intellectual performance.

Finally, if we take off our Rose-coloured glasses for a moment we might discover some information that is surely of interest for theorists of intelligence. Is it not of some interest, for example, to discover that early in development there is some similarity between adopted children and their nonbiologically related siblings (never greater than that shown with their biologically related offspring with whom they have not shared a home), but that this similarity falls to the levels of similarity between any two randomly selected individuals by early adulthood (see Scarr 1992)? Why should this be so? None of this is to say that genetic studies can explain what intelligence is. On the contrary, it will be a psychological theory, not a genetic one, that will do the real explanatory work. But if such a theory can accommodate such data, then it will be all the richer for it.

A clash of competing metaphors

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Abstract: Metaphors have three important functions in scientific discourse: heuristic, rhetorical, and epistemic. I argue that, contrary to prevailing opinion, metaphors are indispensable components of scientific methodology as well as scientific communication. Insofar as the choice of metaphors reflects ideological commitments, all science is ideological. The philosophically vexed question is how to characterize the sense in which science is not *merely* ideological.

The seventeenth century was the locus of a dispute centered on the question of the proper modes of representation for scientific knowledge, a dispute that was to shape what we now call modern science. Following the legacy of the Royal Society in England, metaphors were condemned to play ancillary roles to mathematical representations.

This view is mistaken (Bradie 1999). Metaphors have three important functions in scientific discourse: heuristic, rhetorical, and epistemic. In their heuristic capacity, they serve as guides for further research and formation. In their rhetorical capacity, they are useful devices for the communication of research and results. Finally, they have an epistemic function. Scientific descriptions and explanations are fundamentally metaphorical. Mathematical models in biology are representations of the physical interactions of biological organisms. As such, they are "metaphorical redescriptions" of phenomena in mathematical terms (Bradie 1997; Hesse 1966). With these brief remarks to set the stage, let us turn to an analysis of Rose's critique of the metaphors of reductionism.

Rose's book is a critique of an "ideology" that he labels "ultra-Darwinism" and a brief for replacing it with a better vision, a vision he calls a "homeodynamic" view of the evolution and development of organisms. This is a clash of competing metaphors. While I agree with Rose that scientific disputes are not merely ideological conflicts, in light of the framing remarks on the nature of metaphors in science, it may be harder to spell out the nonideological character of science than some may suppose. It should also be noted that I am using the concept "metaphor" in a stronger sense than Rose's. For him, metaphors are often *mere* metaphors, what I have labeled heuristic or rhetorical metaphors. But all scientific modeling involves unavoidable distortions – whether one calls them metaphorical redescriptions or not.

In Chapter 2, Rose remarks on the "paradox of science": in trying to provide a "true" account, we must filter our understandings through conceptual frameworks framed by the "experience and expectations of its practitioners" (p. 24). The deep question is: How should we best view biological phenomena, in terms of entities or processes? These are, in fact, two deep metaphors which shape the kinds of solutions we are prepared to accept for particular problems (p. 42).

Rose cautions about the "seductive trio" of metaphor, analogy, and homology. When we use mere metaphors as analogies or homologies (such as when we say that the brain *is* a computer), or when we use analogies as homologies (as in treating animal conflict as homologous to human aggression), then we "delude ourselves" (p. 68). The metaphors and analogies we find "attractive" are not based in the "science," but are ideologically based (p. 69). The bottom line: science is ideologically based, but it is not *just* one ideology among others. There are, Rose claims, "reality checks." But how are we to characterize these checks? Rose here finds himself in the familiar position of someone who wants to acknowledge the ideological source of our representational schemas without slipping down the slope into epistemological anarchy.

There are many examples of the use of metaphors of varying probity in Rose's book but limitations of space preclude a discussion of them here. The fact is that scientific discourse is rife with metaphorical formulations, allusions, insinuations, and applications. Rose's account brings out both the positive and the negative features of many metaphors. Rhetorical devices have the power to inspire and suggest as well as confuse and obscure. Can we do without them? Can we somehow abandon our metaphorical models of speech and representation and get down to the "plain, unvarnished truth" of things? I suspect not. In promotion, explanation, and dissemination, metaphors play a central role, and prospects for metaphor-free speech and representation is virtually nil. To take the easy case first, consider the rhetorical dimension, which covers the transmission of information among specialists and from specialists to the general public. Scientists must be able to communicate their programs, techniques, and successes to a wide audience. As our collective understanding of the world increases, our individual understanding decreases. The history of science has been the history of increasing specialization. The result is that as our cultural understanding of particular features of the world gets deeper, scientists have difficulty communicating their technically coached results effectively to wider audiences, let alone understanding disciplines other than their own.

Even so, if my argument is correct, scientific theories and their applications are fundamentally metaphorical (hence, ideological) in character. The "sins" of the reductionist ideology (such as problems of agglomeration, spurious localization, etc.) are the unavoidable by-product of scientific conceptualization. As soon as we agree that cases can or are to be grouped together, we are engaged in "ideology." The reductionist might be an extreme case but when we reject reductionism (in favor of Bishop Butler's dictum that "everything is what it is and not another thing") then we swing toward the other extreme where all cases are unique. To go all the way in this direction is to forego *all* possibility of scientific understanding or of producing any systematic understanding of nature. We need to stop somewhere between the two extremes. Is there a *natural* stopping point? Is there (1) some place "dictated" by nature, as it were, or do we inevitably (2) choose places to stop on the basis of interests and convenience? If the latter, then science can never be free from ideology and rather than pretend that it is, perhaps we should promote analytical practices that bring to the surface our ineluctable biases.

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What the human annals tell us

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Abstract: Evidence reveals numerous cross-cultural universals regarding human mental processes and behavior. Similarly, cross-cultural data are consistent with predictions from theories of kin selection, reciprocal altruism, and sexual selection inspired by Darwin's theory of evolution by natural selection. Thus, the "annals of human behaviour" do provide "example[s] fitting the sociobiological bill," (*Lifelines,* p. 202) thereby, supporting sociobiological accounts of human behavior.

In *Lifelines,* Steven Rose wants to persuade the reader that reductionism – as a method, but more especially as a theory – is a bankrupt and dangerous paradigm. On Rose's view, reductionism, with liberal (not guilty) help from wrong-headed ultra-Darwinists, points falsely to the genes, aided by natural selection, as the sole determinants of the functioning of biological organisms. As foils for his argument, Rose singles out such prominent sociobiologists or friends of sociobiologists as Daniel Dennett, Richard Dawkins, and E. O. Wilson, who have tried to account for biological minds and behavior in light of selectionist theory. Rose targets, in particular, sociobiological accounts of kin selection, reciprocal altruism, and sexual selection as examples of the troubled waters into which unwary biologists, seduced by the reductionist agenda, might find themselves wading. Rose asks whether any examples from the annals of human behavior might fit the sociobiological bill. As a recovering cultural relativist trained in the Franz Boas school of anthropology, I want to take up Rose's challenge.

Selectionists predict that we should find psychological and behavioral uniformity in response to shared opportunities and challenges that the world has to offer to our species. So, taking up Rose's challenge, are there any such uniformities in human functioning as described by anthropologists? Here is a list of cultural universals from the guru of social anthropology, George P. Murdock (1945): age-grading, athletic sports, bodily adornment, calendars, cleanliness training, community organization, cooking, cooperative labor, cosmology, courtship, dancing, decorative art, divination, division of labor, dream interpretation, education, eschatology, ethics, ethnobotany, etiquette, faith healing, family feasting, fire making, folklore, food taboos, funeral rites, games, gestures, gift giving, government, greetings, hair styles, hospitality, housing, hygiene, incest taboos, inheritance rules, joking, kin groups, kinship nomenclature, language, law, luck superstitions, magic, marriage, mealtimes, medicine, obstetrics, penal sanctions, personal names, population policy, postnatal care, pregnancy usages, property rights, propitiation of supernatural beings, puberty customs, religious ritual, residence rules, sexual restrictions, soul concepts, status differentiation, surgery, tool-making, trade, visiting, weaving, and weather control. Other observers of human cultures have proposed other lists which extend the number of universals. So anthropologists find uniformity in how human beings organize their lives. Cultural relativists tend to focus on local variations. Some people are monogamous and some are polygynous and some are polyandrous, they assert, so we can't find anything shared across cultures. This misses the astonishing fact that, in every known human culture, people stand up in front of their neighbors and commit themselves to a culturally sanctioned mate. Ditto for other efforts to focus on variations on the theme across cultures while missing the theme itself.

What about Rose's target sociobiological theories? Is there anything in the annals of human behavior that might cause us to view kin selection, reciprocal altruism, and sexual selection theory as something less than bankrupt? Any anthropologist will tell you that the kin group is the basic functional unit of organization across societies. Generations of anthropology students have been bored to tears by the endless focus on kin lineages and kin terminology and kin groups with which anthropology professors regale them. People across cultures depend upon relatives as opposed to non-kin for their domestic, religious, economic and political well-being. They share resources and cooperate on tasks preferentially with kin and leave their wealth to kin (Broude 1994). Across human societies, it sure does look as if nepotism flourishes and blood is, indeed, thicker than water.

Ethnographic descriptions also indicate that human beings conduct their interpersonal relationships in ways that are consistent with reciprocal altruism theory. Patterned exchange among nonkin is endemic across cultures. Friendships, voluntary associations, and cooperative work groups of all sorts, in which mutual aid and support are expected, are found everywhere. Anecdotes are endemic in the ethnographic literature in which the failure to reciprocate a gift given or a favor done is met with indignation and thinly veiled hints or threats (Broude 1994). The husband-wife relationship, itself a cultural universal, is a compact between unrelated individuals in which each contributes to the welfare of the other and of the offspring of both. Even in cultures that are invoked as examples of the altruistic ethic, we find expectations of reciprocity lurking below. Among the Kalahari !Kung, for example, people give away their possessions freely, and the !Kung are frequently hauled out as an example of a communitarian society. But while gift-giving is universal among the !Kung, so is gift-receiving. Moreover, exchanges tend to take place among a small number of partners, who may even be inherited and each of whom keeps a running tab of who has given what to whom and when (Shostak 1981).

Theories operating under the umbrella of sexual selection also live comfortably with the ethnographic data. We see sexual selection played out, for instance, in the almost universal double standard regarding extramarital sex in which women's sexual infidelities are viewed as more serious than those of men, as sexual selection theory would predict. Thus, in one cross-cultural survey, women were permitted to engage in extramarital sex in only 11.2% of 116 traditional cultures around the world, while extramarital sex norms were more permissive for men than for women in 77.7% of these cultures. Even when extramarital sex is condemned for both spouses, punishments for infringements of these norms are typically considerably more severe, and often dramatically so, for wives. And where extramarital sex norms for wives are permissive, the husband is likely to receive a benefit. For example, among the Lesu of New Ireland, a wife who engages in sexual relations outside her marriage receives a gift from her lover, which she then hands over to her husband (Broude 1994).

Rose argues that biological organisms, including human beings, construct their own futures. Gene-mediated selectionist theory on his view perverts this fact of life. What salvages it? Interactionism. We all say that we are interactionists, observes Rose. But some of us, on Rose's view, are more interactionist than others. Rose is a good interactionist. Sociobiologists, he claims, are not.

Interactionists recognize that the behavior and development of biological organisms are the joint product of biology and environment. But, then, selectionist theory is deeply interactionist. Who takes the cooperation of biology and environment in producing lifelines more seriously than a selectionist? I am interested in hearing how Rose reconciles evidence of human universals from the human annals with an interactionist theory that does not make room for selectionist thinking.

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No short cuts to science

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Abstract: Steven Rose regards oversimplification of biology as the supreme sin, inevitably leading to evil consequences, and requiring an unique distortion of scientific practice to avoid it. To avoid this, he proposes a short-cut to scientific knowledge by defining certain areas of biology that are intrinsically flawed. But this achieves only a subordination of science to politics. There are no general-purpose shortcuts for evaluating the validity of theories, and no substitutes for testing specific theories using relevant evidence.

Steven Rose is against "reductionism" in science. For his major example, he invents a theoretical framework termed "ultra-Darwinism" and characterized by intellectual bankruptcy and moral depravity. The insistent refrain of *Lifelines* is: "things are more complex than that."

But this is not enough, because in science things *are* always more complex than that – science is a search for simplified, unifying theories. The proper question is whether or not the simplification is useful, whether or not the simple model *works* as a theory, whether its consequences are consistent with the test of further observations.

Hence it is clear that what Rose objects to is *inappropriate* simplification, rather than simplification per se. But inappropriate simplification is merely a type of bad science – simplification is inappropriate when it does not work. So, Rose is against bad science. Nothing controversial about that. The problem then becomes: how can we tell bad science from good? How can we tell oversimplification from the right amount of simplification?

To paraphrase Einstein: theories should be as simple as possible, but no simpler. In other words, we should simplify as much as possible (because simple models are more rigorously testable and more useful), but we will know that a scientific theory is oversimplified when its consequences are inconsistent with structured observation. An oversimplified theory will not be predictive, because it has missed out important variables. Simplification is bad *only* when it does not work. In opposing oversimplification, however, Rose conflates the pragmatic and the ethical. For Rose, oversimplification in biology is evidence of moral depravity. He does not accept that any wickedness due to oversimplification is accidental; he is trying to argue for a special logical link such that oversimplification inevitably leads to evil consequences. Implicitly, he sees oversimplification as the supreme sin, requiring an unique distortion of scientific practice to avoid it.

Rose is transfixed by a specific ethical danger: that biological theories about human being may be oversimplified and that such oversimplified biological theories are more likely to be misinterpreted, misapplied, and used to justify moral harm than are any other sorts of theory. With this in mind, throughout *Lifelines* Rose is trying to find a formula by which we might know *in advance of testing* whether a biological theory is oversimplified. The idea is that theories and scientists grouped under the ultra-Darwinist umbrella will be condemned *a priori.* It seems to me that Rose wants to do this in order that the general public (who lack specific scientific knowledge) can prevent themselves from being hoodwinked by repressive political propaganda, probably right-wing, that is masquerading as science.

In trying to rule out this particular source of harm, Rose has implicitly set himself the task of constructing a general-purpose shortcut to measuring the truth of scientific theories – to know whether a theory is valid or vapid without having to go through all the hard work of reading, understanding, observing, and experimenting. In other words, Rose is seeking a shortcut to scientific knowledge.

If I am right about the fundamentally ethical drive behind the writing of *Lifelines,* this would explain why Rose hardly seems to have read about, let alone made the intellectual effort to understand, the major work in evolutionary biology over the past 20 years. Yet this has been a period of remarkable progress during which the theory and practice of evolutionary biology had been transformed. A few of these major advances are name-checked; but never explicated, engaged with, or refuted. Throughout *Lifelines,* Rose fails to confront the best and most recent scientific work and attacks obviously inferior studies, garbled media reports of research, or old papers from the 1960s and 1970s that have often (as is the way for most science) since been revised or superseded. This is merely shooting fish in a barrel.

Mistakes may be forgiven in a book of this scope. But some are evidence of a failure to do the elementary homework necessary for a person who is purporting to critique and redirect evolutionary biology. For example, on page 227 Rose writes "to cling to 'the gene' as the sole unit and level of selection under these circumstances, as Maynard Smith and the ultra-Darwinists do, seems perverse." Well, it happens to be the case that Maynard Smith is co-author of a book (Maynard Smith & Szathmary 1995) called *The major transitions in evolution* (which was also published as an essay in *Nature;* Szathmary & Maynard Smith 1995) – a major work on exactly the topic of the many units and levels of selection that Maynard Smith is supposed to have perversely ignored. Maynard Smith's book forms part of a significant branch of mainstream evolutionary biology that includes important work by David L. Hull, Richard Dawkins, and some others who are elsewhere categorized in *Lifelines* as being among the single-gene-obsessed-ultra-Darwinists. Perhaps Rose does not know this work or understand its implications – or perhaps he knows but has excluded it. Whatever the explanation, this line of evolutionary research torpedoes Rose's major argument. Scientists like Maynard Smith have *already* achieved a level of understanding of multi-level selection and interaction far beyond that called for in *Lifelines.*

Evolutionary biology is a science like any other, if it is allowed to be. It should not be treated as a special case. Blending ethical and social criticism with science, as Rose does, is a recipe for dishonesty and double standards. He has subordinated human biology to politics, and is mainly concerned to fit human biological knowledge into a pre-existing agenda of what is acceptable.

By contrast, I would argue that biology is oversimplified only when it is untrue; and not because simple theories are uniquely susceptible to misapplication. An oversimplification of human nature might be used to justify repression; but then again, tortured casuistic logic or a denial of human nature can be used to justify repression with equal facility. Hence, "reductionism" and "ultra-Darwinism" are merely boo-words, and are irrelevant to the proper practice of science. The validity of a specific theory can only be determined by the laborious work of evaluating its consequences on the basis of specific relevant evidence. There are no short cuts to science, and no substitutes for substantive knowledge.

Metabolic complexity has no bearing on genetic determinism

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Abstract: Metabolic systems are complicated and contain very large numbers of interacting reactions and many internal regulatory mechanisms. This does not prevent the genetic composition of an organism from influencing its behavior, however, nor does it preclude the possibility that some aspects of its behavior may be amenable to simple explanations.

In 1774, Leonhard Euler claimed at the court of Catherine the Great that he could prove the existence of God, silencing his opponent, Denis Diderot, with the following challenge: "Sir, $(a + b^n)/n = x$, hence God exists; reply" (quoted by Singh 1998). Like

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Diderot, Rose finds algebra hard to follow (p. 160) and might find it just as difficult to recognize the irrelevance of Euler's argument to the proposition it was supposed to prove. Rose's own style of discussion is similar, however, apparently in the hope that his readers will be sufficiently ignorant of biochemistry to think that his emphasis on its complexity has some bearing on the question of whether genes influence behavior.

Books that set out to explain why organisms behave as they do describe *observations* of behavior on almost every page. The books of Richard Dawkins, whom Rose selects as his special target, illustrate this well: readers can reject all of the author's interpretations while remaining fascinated by the purely factual information that these books contain. How can one hope to convince anyone of the truth of a theory without supporting it with abundant facts? Yet hard biological information is extremely sparse in Rose's book. There is a great deal about what he thinks of other biologists' opinions, but almost no observations from behavioral biology. Nonetheless, in his preface (p. x) he aligns himself with the practising biologists "who spend a significant part of every working day thinking about and designing experiments," dismissing Dawkins and Dennett as "people who either no longer do science or never did it." What a pity, therefore, that Rose chose to include so little of the experimental basis of his ideas in his book. There are a few vague remarks about how chicks behave, and that's about it.

Rose claims throughout the book to be a biochemist, and in the remainder of this review I shall concentrate on the section (pp. 158–66) that deals with the complexity of metabolic networks and underlies the suggestion at the end of the book (p. 307) that genes are just individual workers in the great molecular democracy of the cell. As this section occurs in a chapter with the same title as the book, it is fair to regard it as the core of the book.

However, even as a standard biochemical account of the basic ideas of metabolic regulation, divorced from its role in the whole thesis, it is peculiar. In a muddled account of enzyme catalysis that does not contain any algebra, Rose confesses that he finds the algebraic relationship between a reaction rate and a rate constant hard to grasp. He then presents metabolic regulation in terms of the tired old myth of the *rate-limiting reaction,* saying that in practice it often turns out that the rate-limiting step is one of the first in the sequence – obviously advantageous so far as the cellular economy is concerned: Does it "turn out," or is it assumed without considering any other possibility? The enzyme phosphofructokinase is asserted in innumerable textbooks to be the rate-limiting enzyme for glycolysis. If it were, then overexpressing it would increase the glycolytic flux, but even though the relevant experiments were done in yeast more than a decade ago (Heinisch 1986), and have since been repeated in other organisms, the results – *no* detectable increase in glycolytic flux when phosphofructokinase is overexpressed 3.5-fold – have yet to be taken seriously by the hordes of biotechnologists in search of the mythical rate-limiting enzymes.

Rose appears to be conscious that his account is faulty, because at the end of it he quotes Kacser and Burns (1979) to the effect that control is shared among all the enzymes of a system. But does he mean the *mea culpa* that follows? If Kacser and Burns are right, then what was the point in wasting pages on a muddle? If they are wrong then why are they being quoted? Presumably the attraction of their paper lay more in its title and some quotable sentences than in any serious study of its content.

If these pages have any point at all it is to establish that metabolism is complicated, involving very large numbers of interacting reactions. True enough, but what does that have to do with the idea that genes can affect behavior? A system can be highly complicated, with many internal regulatory devices, yet its behavior may still be amenable to explanation in terms of a particular set of external influences: Does anyone doubt that the path taken by an airliner is affected by the actions of its pilot, or must one just say that it is a complicated device and the pilot is just one of many components in its democratic organization?

Behavioral neurogenetics beyond determinism

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Abstract: Rose's *Lifelines* justifiably attacks the rigid genetic determinism that pervades the popular press and even some scientific writing. Genes do not equate with destiny. However, Rose's argument should not be taken too far: genes do influence behavior, in animals as well as in man.

Let us start with my conclusion: *Lifelines* by Steven Rose should be required reading for every biologist, especially those working in behavioral and neural genetics. Rose presents a meticulously argued case against the deterministic, reductionist mode of thinking that pervades much of modern biology. However, much as I find myself in agreement with many of Rose's starting points and conclusions, I disagree with many of his arguments that lead from the one to the other. In addition, Rose weakens his arguments by frequently inserting politically inspired barbs (against Thatcherism, or the privatization of utilities in the United Kingdom, for example).[1] This is unfortunate, because in my experience this allows people to brush aside his otherwise valid scientific arguments, with the brief observation that Rose is ideologically biased.

The first chapters of the book offer excellent reading and, although a few strained arguments will occasionally be encountered, it is mainly the last few chapters, where Rose discusses behavioral and neural genetics, that I find myself most in disagreement with him. For instance, on page 190 Rose argues that heritability estimates can be valuable for an animal or plant breeder interested in crop yield or milk yield in cows, but not when applied to human behavior. In principle, I agree with this observation (Crusio 1990), but I disagree with Rose's way of arriving at this conclusion. His argument is that milk yield is a phenotype that is more straightforward to measure than intelligence, religiosity, or other such characters. This reminds one of the contention that IQ, because it is a complex character, is a soup, and that whereas genetic analysis of its components, such as the size of tomatoes and the weight of potatoes is reasonable, analyzing soup is not (Roubertoux & Capron 1990). But is such a deceptively simple measure as milk yield not also a "soup"? Milk yield is, in fact, a complex character, the sum of many different "subcharacters" such as fat content, chemical composition, water content, and so on. If Rose finds that heritability estimates of milk yield can be done, then it follows that such estimates are also possible for IQ in human beings, at least in principle. The point he misses is that the sole utility of a heritability estimate lies in the fact that it predicts the response to selection of the character in question (Crusio 1999), something nobody in his right mind would envision for IQ or religiosity in human beings, I hope.

The picture that Rose paints of behavior genetics is quite a caricature. It seems almost as if he wants to argue that genes and behavior have nothing to do with one another and that if genes do influence behavior, then they only do so in animals and surely not in man. In his efforts to show this, Rose sometimes misrepresents the things said by others, for instance, when he discusses the work of Cases et al. (1995) on aggressive behavior in a mouse mutant lacking monoamine oxidase A (MAOA) activity: Rose first describes a test used by some to measure aggression in rats: mouse killing behavior. Of course, whether killing a mouse that belongs to a totally different species is a good measure of the aggressive tendencies of a rat is debatable. But Cases et al. were studying aggression of male mice vis-à-vis conspecifics, a completely different situation. Rose then continues to say that "this behaviour in the rat is transmogrified into an analogue of the aggression shown by drive-by gangs shooting up a district in Los Angeles, as in the concluding sentences to the paper by Cases" (p. 294). Again, we have a groundless (and needless!) accusation. At no point in the Cases paper are gang shootings or even distantly similar events

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mentioned. All Cases et al. (1995, p. 1766) are saying is that the results obtained with their mice support the notion that the behavioral problems described in males from a Dutch kindred (Brunner et al. 1993) are due to their MAOA deficiency and not to "unusual genetic background or complex psychosocial stressors." This conclusion seems to me completely warranted by the data. Still, as I have argued before (Crusio 1996), although the knock-out mice may be a good model for the problems encountered in the Dutch kindred, it is debatable whether these troubles are adequately described by the word "aggression." In addition, it is becoming increasingly clear that the MAOA deficiency is a very rare mutation, so far described only for this single Dutch family (Stamps & Gurling 1998). So once again I find myself in agreement with Rose, namely, that the MAOA mutation does not explain most (or even any) of the everyday violence that we observe in the streets – without agreeing with the arguments he uses to arrive at this conclusion.

In fact, both my agreement and my disagreement with Rose can be illustrated by studying a small experiment more closely: a comparison of aggressive behavior in two inbred mouse strains, NZB and CBA/H. The behavioral test is a situation in which some standard opponent (a male mouse from a non-attacking strain) is introduced into the cage of the animal to be tested. The behavioral measure is simply whether or not an attack occurs. Of course, this test situation is very simplified compared to natural situations, but not more so than the learning tests that Rose deems acceptable for his chicks. In this testing situation, about 80% of males from the NZB strain attack, as opposed to about 30% of CBA/H mice. In a series of careful experiments, Roubertoux, Carlier, and colleagues (Carlier et al. 1991; Roubertoux et al. 1994) have shown that apart from some important effects of the (maternal) environment, this strain difference is owing to genetic differences between these strains. The results warrant two conclusions. First, genes *can* influence behavior, even a complex one like attacking a strange conspecific. However, this experiment also tells us another important fact: although all animals within a strain are highly similar genetically, about 20% of the supposedly aggressive NZB mice do not attack, whereas about 30% of the non-attacking strain CBA/H do. In short, even under the controlled conditions of a simplified laboratory test, where all environmental conditions are standardized as much as possible, genes do not determine behavior in a rigid fashion. Apparently, mice have a "choice." And if this goes for mice, then I agree with Rose that it will not be different for human beings.

Rose is right in attacking the deterministic notion found so often in the popular press (and even in scientific writings) to the effect that genes equal destiny. This is an important and valuable message. However, we should take care not to let the pendulum swing too far by denying any influence of genes on behavior. It is the task of behavioral neurogeneticists to find the correct balance between these opposing views.

NOTE

1. Some of these barbs degenerate into inadmissible *ad hominem* attacks. A glaring example of this can be found in note 17 on pp. 206–207. Rose asserts that many of T. J. Bouchard's articles appeared in "*Acta genetica gemellologica*" (*sic:* the correct title of the journal is *Acta geneticae medicae et gemellologiae*), a "somewhat obscure journal . . . or in volumes of conference proceedings, rather than in refereed journals." Of course, it is evidence and arguments that matter, not the place where they have been published. Not only does such a nasty attack distract from Rose's main arguments; the accusation is also wrong. A Medline search renders 42 articles for "Bouchard TJ," of which 40 are on twins. These 40 articles were published in 30 different journals. Three articles (plus a news item) appeared in *Science,* one in *Nature.* Only two articles are actually published in the *Acta.* I expect and demand that Dr. Rose offer his apologies to Dr. Bouchard.

Stability of behavioral traits within the framework of neural plasticity

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Abstract: *Lifelines* supports the theme that behavioral development is a fluid, life-long phenomenon. In contrast, many emotional and cognitive traits are subject to strong genetic influence, and are highly stable over many years. The manner in which neuroplasticity and trait stability co-occur needs to be modeled. An outline of such a model is provided to promote discussion of this complex issue.

Rose elegantly supports the theme emerging in many life and behavioral sciences that development is a life-long phenomenon characterized by the ability of the organism to reorganize and change in the face of changing environments. In contrast to this theme is the fact that many emotional personality traits are subject to strong genetic influence (Bouchard 1994; Tellegen et al. 1988), and are highly stable (in terms of an individual's rank order) over as many as 20 years (Costa & McCrae 1994). Furthermore, twin studies have shown that the stable component of positive emotional levels itself has a heritability that approaches 80%, and that stress-induced variation in positive emotional levels are temporally quite limited, returning to pre-stress trait levels as if a hedonic set-point influence were operative (Lykken & Tellegen 1996). The same finding occurs with respect to some forms of cognition: IQ levels (which can be thought of as a cognitive trait marker) of identical twins reared apart show equally high heritability as those for identical twins reared together (Bouchard 1997), and IQ is relatively stable after adolescence.

Clearly, the development of cognitive abilities and intelligence, as well as emotional behavior, must be dependent on powerful experience-dependent neuroplasticity processes. And just as clearly, more intense environmental conditions can effect major changes in behavior. Extremely stressful conditions, or stressors occurring during early stages of development, may cause long-term modifications of neurotransmitter and/or neuropeptide functioning that can be accompanied by "personality" changes (Le Moal & Simon 1991; Yehuda & McFarlane 1997). Similarly, animal work indicates that transitions in hedonic set point may occur under certain conditions (Ahmed & Koob 1998). But, within the normal range of environmental circumstances, temporal stability of behavioral traits is often observed. The manner in which neuroplasticity and trait stability co-occur needs to be modeled, but *Lifelines* provides no clear guidelines for this problem. I offer the following possibilities on which Rose's thoughts would be valuable.

Specifically at issue is the manner in which individual differences in emotional personality and some cognitive traits become stabilized. To be clear, let me define personality traits, like extraversion, anxiety, and fear, as emotional systems that have evolved to increase our adaptation to critical stimuli in the environment, such as rewards, uncertainty, and aversive stimuli, respectively. I and others (Gray 1992) assume that individual differences in the neurobiological sensitivity (or reactivity) to these critical stimuli underlie phenotypic variation in the traits. Moreover, the neural foundation of these emotional systems involves networks of brain regions that interact to emotionally evaluate and respond to environmental stimuli (LeDoux 1992). It seems likely that individual variation in the neurobiological sensitivity to critical stimuli is due, not to structural variation in brain regions that comprise the network associated with an emotion system, but rather to variation in the functional properties of neurotransmitters/neuromodulators that modulate the functional processes of the network.

A large body of animal research suggests that behavioral stability may occur within a plastic neural environment via a sequence of factors (Depue & Collins 1999).

1. Genetic factors can strongly influence a biological variable that plays a central role in a trait's phenotype, such as a neuro-

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transmitter that strongly modulates a network's functioning. For instance, genetic effects on the number of midbrain neurons of transmitters of wide distribution (e.g., dopamine, serotonin) have been demonstrated to markedly and stably influence trait levels of emotionally-relevant behaviors in animals. This factor can be viewed as the lowest-order foundation of the concept of trait.

2. Significantly, this biological foundation may strongly modulate the impact of the environment on experience-dependent processes. To extend our example, the genetically-influenced number of transmitter-synthesizing neurons may influence the mean range of magnitude of critical stimuli that can (i) activate neurotransmitter projections sufficiently strongly to (ii) generate their effects on neural and behavioral processes. With an increasing number of neurons, the range of magnitude of effective stimuli that can activate emotional behavior is increased into the weaker extreme of the range. Animal research strongly supports the operation of this factor.

3. The product of this positive genotype × stimulus-efficacy interaction, which may be reflected in the synaptic density within the trait's neural network, may begin to develop during early postnatal life, and may represent a relatively stable psychobiological foundation that mediates the influence of trait-salient contexts over emotional processes. This psychobiological foundation serves as the background upon which future experience-dependent processes may act, and may be seen as the basis of temperament.

4. Stability of this psychobiological foundation or temperament level is assumed to be maintained over time by at least two factors: (a) the psychobiological foundation of the temperamental trait has now established the mean range of effective critical stimuli more strongly, thereby further influencing the extent to which the environment has "access" to experience-dependent neuroplastic processes. At later stages of development, one can presume that the operation of an active genetotype × critical environment interaction will play an important role in maintaining initial differences. But most importantly, (b) individual differences in neurotransmitter functioning (e.g., due to variation in neuron number) may strongly influence the experience-dependent neuroplastic processes themselves, thereby influencing the extent to which critical stimuli gain control of and facilitate emotional processes.

By way of example, this model may be applied to the personality trait of extraversion (Depue & Collins 1999), which has strong genetic influence and is stable over many years and life stressors (Lykken & Tellegen 1996; McGue et al. 1993; Tellegen et al. 1988). Genetic variation in the number of dopamine neurons in the midbrain is reflected in very stable variation in extraversion-relevant behavior in animals. Moreover, dopamine is critical to the development of control of rewarding contexts over behavior through its modulation of heterosynaptic plasticity: dopamine facilitates the connection of efferents from brain regions carrying the salient context of reward to brain regions involved in integrating extraverted behavior. And, most importantly, individual differences in dopamine functioning in animals correlate with the degree to which the environment gains control over extraverted behavior.

Thus, the important point concerning stability is this: because animal research indicates that neurotransmitter functioning can play an integral part in (a) determining the range of effective critical stimuli that have access to an emotional system, and (b) the extent to which those stimuli gain influence over behavior, individual differences in neurotransmitter functioning may modulate both of these processes and, hence, the extent to which trait-salient contexts facilitate emotional processes. In this way, early experiential processes may lay the foundation for trends in emotional behavior by moderating the strength of later experience-dependent processes involving the functional capacities of a significant neurotransmitter projection system (Collins & Depue 1992). Therefore, across the lifespan, individual differences in neurotransmitter functioning may modulate synaptic arborization within network circuitry associated with an emotional system, which in turn would modulate responsivity to critical stimuli and be manifested in stable variation in emotional expression.

Multidetermination

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Abstract: Professor Rose is inflamed against the views of Richard Dawkins concerning genetic determinism. He has constructed an image of a much stupider and eviller Dawkins view than is likely to be true and argues against it. This is unrewarding, but otherwise his discussion of biology is very interesting.

Steven Rose is writing against reductionism, gene determinism, and Richard Dawkins, whose *Selfish gene* (1976) was, for me, a revelation in its day. He is offended by Dawkins's way of looking at biology, which he sees as a crude and oversimplified view of organisms. There would be nothing wrong with this if he simply engaged in open combat with Dawkins's cheerful geneticism. But he seems vehement. A danger of vehemence is a tendency to misdescribe the opposition. In the Preface Rose tars the opposition with Nazism, eugenics, racism, anti-Semitism, elitism, and sexism. When a scholar and a gentleman is seen doing something as unscholarly and ungentlemanly as poisoning the well, it is embarrassing. No, let me explain: It was more than embarrassing. It made me so angry I nearly refused to read the book at all. That would have been a pity, because it is otherwise an informative and well-written book

Rose blames this crude biology on reductionism and determinism. Fortunately he does not take on the whole problem, but only asserts that organisms are not genetically determined. He proposes to demolish "radical" reductionism. Reductionism is the view that every phenomenon can, in principle, be explained using only terms and laws of physics: no irreducibly novel stuff or laws need be introduced.

Rose concentrates on trying to break the chain at the reduction of behavior to genetics. Everyone knows behavior is sensibly described and explained only in intentional, teleological, functional, and instrumental terms. Even so, if you deny the theoretical possibility of (someday, somehow) reducing those terms (by stages) to purely physical terms your alternatives are:

1. asserting that the behavior of living things is supernatural (owing to immaterial forces);

2. denying that there are *any* laws that govern organism behavior; and

3. asserting that new primitive terms must be added to the ones about quarks and mesons and whatnot, and new primitive laws added to the ones about strong and weak forces, gravity, and electromagnetism. This is emergentism.

Rose is an emergentist. Emergentism is appealing but incoherent. If life is material, then it obeys the laws of matter; if the laws of matter have to be augmented with laws of life, then what does it mean to say the universe is *material*? Could you add terms about the Life Force, or souls, to the physical primitives and still call the universe material?

Rose's first step in destroying reductionism (as he sees it) is to undermine naïve faith in the pure objectivity of "science." This is a healthy corrective to any starry-eyed ideal of science surviving into this decade. Robust skepticism is the basis of science, not its bane. Nor does the point have much to do with reductionism.

He demonstrates, for those of us who missed the last several decades, that observation is neither direct nor passive but directed by the theory that provides its motivation and context, by the instrumentation that makes it possible, by the ideology of the person doing the research, and by the underlying metaphors of the time. Again, while none of this is part of ideal science, it is certainly part of the human beings who do science. Although we can separate out a lot of personal bias, it is hard, as they say, to prove a negative; even cross-cultural peer review cannot screen out the unknown bias of an age. (Again, it does not have much to do with reductionism.)

Rose spends the best part of *Lifelines* on an account of molec-

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ular biology, genetics, embryogenesis, and development. This is a wonderful review and I recommend it for the teaching of children. There is a lot of fluidity, self-organization, dynamism, process. This is fine and true, except that he seems to believe that reductionism cannot handle them; that it can deal only with a motionless Erector-set reality. *I* don't think reductionism is so feeble.

I was struck by the refrain that cells and organisms are "free" to make (choose, construct) "their own futures." It sounds *so* bracing and hopeful and brave, until it occurs to one to wonder why, if this is true, so many of them construct such short and painful futures for themselves, and almost all the rest construct inglorious and finite ones. "Choice," when applied to cells, is peculiar. The "self-determination" of a cell, or an embryo (by which he means that not all its story is read off from its genes) is not related to the free will essential for moral responsibility, and pretty remotely kin to persuadability in everyday life and politics. I wondered whether Rose thought it had something to do with either of them, and, if not, why it seemed so important to him. The "freedom" of cells from total determination by their genes is just "slavery" to their environments as well. This is so even if their environments are acted upon by their natures, which are chemical and physical. That a cell during embryogenesis does not behave like a player piano, with its genes as bumps on the player roll, but is sensitive to and interactive with its continually changing environment, does not imply that its behavior is undetermined by the laws of physics and chemistry. The only alternative determinants are impersonal laws of randomness, or whims of supernatural agencies.

Sometimes Rose is so gleeful at denying *genetic* determinism a point that he awards it to *chemical* determinism (pp. 170 ff). In even a noncellular context, he explains, ribosomes and microtubules, provided with the right chemicals, will assemble themselves from components "spontaneously, like oil films on water, without the need for specific genetic instructions – an intrinsic molecular property which turns out to be at least as important for the origin of life as do the famous replicating molecules of DNA and RNA."

Rose makes a useful distinction between the "genes" of Dawkins, which are "inferred entities," and the actual "genes," blurrily visible under powerful microscopes, that are biochemical loci on chromosomes. These "real" genes are messy; they are spread along the chromosome; they are in pieces that are assembled, like words into sentences, to do different things at different times; most of them seem to have no function, some seem impervious to natural selection, and there is (he says) evidence that it is technically possible that Lamarckian effects might obtain. Dawkins's "genes," in contrast, are whatever in the organism explains the heredity of phenotypic characters. Rose believes that this view of genes contributes to a careless and "brutish" view of organisms, and that the carelessness and brutishness spreads right up to a view of human beings and human society, where it is dreadfully harmful. Which is why he got so untoward in the preface, I suppose.

I am not wise enough to decide whether any view based on an attempt to understand and explain the world ought to be rejected because it might have harmful political consequences. I shall instead assert that genetic determinism is not as stupid and untrue to the world and its ambiguities as Rose urges. The law of gravity seems simple and deterministic, yet not everything on earth is glommed together in one immobile mass. The law applies to every molecule of water – yet water is not always at the lowest point. It is found suspended in the atmosphere, travelling up the tissues of trees, hydrating cells by the gazillion, and piled up as snow on high places. Despite these apparent exceptions, the law of gravity is universal and holds. Similarly, if genes – the theoretical kind, which must ultimately be reduced to the physical kind – govern behavior, they will do so subject to interference from other determinants, and there will be detours and apparent exceptions, which we have not *begun* to work out. I am prepared to believe that we shall work them out and that the unity of explanation without emergent laws and entities will hold.

Neurogenetic determinism is a theological doctrine

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Abstract: In "*Lifelines*" Steven Rose constructs a case against neurogenetic determinism based on experimental data from biology and in favor of a significant degree of self determination. Two philosophical errors in the case favoring neurogenetic determinism are illustrated by Rose: category mistakes and an excessively narrow view of causality restricted to the linear form.

Fred Allen, a comedian in the golden age of American radio in the 1930s, used a metonymic device he called "Allen's Alley" to make social commentaries. In one of his mock interviews, this of identical twins, a Wall Street financier and a Bowery bum, he asked them to explain their differing circumstances. The broker boasted, "My mother was a drunk and my father was a gambler, so I hadda make something of myself." The bum whined, "My mother was a drunk and my father was a gambler, so what chance did I have?" Here is the crux of Steven Rose's argument. Genetic determinists are willing to grant a share of control to the environment, but they are unwilling or unable to assign even a minute fraction to self-determination, because that would be equivalent, in their view, to explaining the operations of the body by resort to mental or spiritual causes, or even regression to miraculous suspension of the laws of physics and molecular biology. This is a matter of principle, because assigning a fraction of 1% or even 0.1% of the determinants remaining after identifying the nature–nurture sources of control would violate the law of universal causality. Yet self-control in everyone is like a spark in a bit of tinder, which, if it catches, can ignite a life of adventure, but if it goes out, can lead to a life of stale drudgery. Rose has fanned his spark into a campfire; now the neurogenetic wolves are circling, looking to make a meal of warm flesh.

The thesis that Rose advances is not an arcane exercise in theoretical biology. It addresses core questions: How are individuals in societies to confront the political and ideological forces by which some degree of social conformity must be achieved, and how can individuals understand and deal with the rationales and explanations of social controls that are provided by academics in neurobiology, sociobiology, and medicine? Chapter by chapter, Rose wades through the experimental data and statistical analyses that medical, social, and biological scientists use to demonstrate their conclusions that, in all societies, the choices that we citizens make are caused by the chemicals in our foods, the pollutants in our cities, and the sequences of base pairs in our genes. These materials mediate the release of hormones, the firing of action potentials in our brains, and the laying down of memory traces, starting even within the uterus, that determine whom we will love or kill, and which stimuli and signals coming to us from our environments, outside our control, we will respond to.

In the course of 309 pages of erudite exposition and argument Rose notes in passing that experimental biologists seldom read philosophy and often regard their peers who do as declining through the stages of "philosopause" and anecdotage into senility. What a mistake! Many of the critiques that Rose directs against reductionism in biology have already been well thought through by philosophers.

One of the threads that links his materials is Gilbert Ryle's (1949) concept of "category mistakes." His now classical example is confusing a university with its collection of buildings or its faculty. This "reification," as Rose calls it, is the most damaging aspect of reductionism, because it identifies a character trait with a molecule, each in an entirely distinctive realm of discourse. This simplistic kind of thinking has led a generation of otherwise intelligent college students to ingest a broad variety of substances in the fruitless pursuit of chemical enhancement of their IQs, and by

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so doing to obviate the necessity for hard study, and it has been used to justify the wholesale sell-out of the profession of psychiatry to the HMOs in the pursuit of quick and inexpensive chemical fixes of complex emotional problems, to mention only two of the more obvious meretricious social practices based in category errors.

In his pivotal Chapter 10 on "The Poverty of Reductionism" Rose writes (p. 279): "Neurogenetic determinism, I argue, is based on faulty reductive sequence whose steps include reification, arbitrary agglomeration, improper quantification, belief in statistical 'normality', spurious localization, misplaced causality, dichotomous partitioning between genetic and environmental causes, and the confounding of metaphor with homology." I summarize this thread by means of the philosophical distinction between linear causality and circular causality (Freeman, in press). There are two major meanings of the verb "to cause." The more common is to make an effect happen, which requires an agent to perform as a cause in a strict and invariant temporal order. This view supports what philosophers call "linear causal chains," so that in theory, by linear extrapolation, every event can be derived by steps from a First Cause, which for theists is God and for atheists is the Big Bang, or perhaps the latter as the tool of the former. In all cases this belief system provides the foundation of neurogenetic determinism.

The alternative and less common meaning is to explain without invoking agency and with permission of simultaneity and even reversal of time order. Philosopher Donald Davidson (1980) asks:

> Why on earth should a cause turn an action into a mere happening and a person into a helpless victim? Is it because we tend to assume, at least in the arena of action, that a cause demands a causer, agency and agent? So we press the question: if my action is caused, what caused it? If I did, then there is the absurdity of an infinite regress: if I did not, I am a victim. But of course the alternatives are not exhaustive. Some causes have no agents. Among these agentless causes are the states and changes of state in persons which, because they are reasons as well as causes, constitute certain events as free and intentional actions.

Circular causality explains the interactions between semi-autonomous elements at multiple hierarchical levels. Rose provides a full array of examples from multiple fields of biological research, particularly his own in the molecular changes that relate to learning in simpler systems. But they are not that simple, ever.

Is the lifeliner objectively free?

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Abstract: Although Rose claims to rely on Marx's paradoxical view of history to explain the freedom enjoyed by what he calls "lifelines," he blurs what one might call the "objective" and "subjective" senses of freedom. This, in turn, reflects his overreaction to biological reductionism. Consequently, in discussing biology-related policy issues, Rose fails to distinguish genuinely efficacious interventions and merely convenient ones.

Steven Rose periodically invokes the Marxist motto "Men [sic] make history but in circumstances not of their own choosing" to capture the concept of "lifeline," his nonreductionist, autopoietic version of Richard Dawkins's "extended phenotype." According to Rose, an organism's genetic and ecological resources constrain the course of its development without fully determining it. The organism's actual trajectory is the result of multivariate interactions at several levels of organization, which together constitute the organism's "homeodynamics," that is, its ongoing struggle to construct a viable future from a fluctuating present. Rose's firm grounding in the biochemistry of the nervous systems precludes vitalism as a metaphysical commitment, yet he sympathizes with the vitalist picture of organisms as actively seeking alternative means in an everchanging environment to achieve ends that are, in some sense, independent of that environment. In light of this picture, Rose argues that at least some organisms – certainly humans – enjoy a significant measure of freedom. But does Rose's concept of lifeline actually live up to the Marxist motto? More generally, does it constitute an adequate conception of freedom?

The paradoxical character of the theory of history epitomized in the Marxist motto is normally explained in terms of a proposition found in Hegel but traceable to Spinoza, Leibniz, and ultimately the Stoics: *freedom is the recognition of necessity.* Rose (p. 18) takes this to mean that once we realize the exact extent to which we are constrained, we can act within our means to construct a world worth inhabiting. However, this is a misleading, or at least incomplete, rendering of what Marx, Hegel, and their predecessors were trying to say. In addition, they believed that you cannot determine the degree of freedom someone enjoys without looking at what *follows* from that person's actions. Freedom does not exist in a world where agents pursue many different courses of action that then issue in a much narrower range of outcomes. When the present appears open to alternative futures, agents tend to regard the world solely in terms of the resources it provides to realize their ends. Rose's rhetoric sometimes veers this way. Yet, depending on the pattern of actual outcomes, the situation may be exactly reversed – our diverse action may simply be means to some other larger end. In that case, as long as we act within the relevant options – almost any action will do – the larger end is served.

No doubt this sounds like I am inching towards Dawkins's "gene's-eye view of the world," in which biological diversity has no proper end other than the safe conveyance of genetic information. Yet, Marxists have also pointed to the proliferation of consumer choices in advanced capitalism as having this character. Consumers spend increasing time and energy deliberating over possibilities; the long term and large scale consequences of these are usually negligible for their own lives, but they latently serve to rejuvenate the circulation of capital. Because consumers are regularly presented with multiple options that force them to refine their wants more precisely than they otherwise might, they are unlikely, in the normal run of things, to discover just how overdetermined their world really is. Thus, their subjective sense of freedom lacks an objective basis. Joined in their belief in the cunning of reason in history, Dawkins and Marx see this point very vividly in a way that Rose does not.

Consider what Rose rejects under the rubric of "reductionism." There is more at stake than simply a denial of the gene's-eye view of the world. Also at issue is the very idea of a coherent systemic perspective from which the biological world can be regarded. For Hegelians and their kin, there is a gold standard of objective freedom, whereby an agent inhabiting a world can be said literally to know what it is doing. If the standard is to be met in a given population, then the diversity of its members' actions must be matched by an equal or greater diversity of outcomes. In addition, these outcomes must generally correspond to what the members wanted, ideally in the terms they expected. If both conditions are satisfied, then there are good grounds for concluding that the population is objectively free. From a systemic perspective, such a population exerts significant control over its collective future.

Although the gold standard of objective freedom is impossibly high, it does enable us to distinguish between types of intervention one might make to improve the human condition. Specifically, the most *convenient* level of intervention is not necessarily the most *efficacious,* if it turns out that once a cause is removed, its deleterious effects are largely reproduced by another factor either present or latent in the environment. The freedom exemplified by the intervention, then, would be merely subjective. Rose falls potentially foul of this critique in his discussion of lung cancer treatment (p. 305). Tobacco companies are currently major funders of research into the molecular biology of the lungs and the localization of "predisposing" genes for cancer. Rose regards this research as a strategic misdirection that capitalizes on the persuasiveness of reductionist rhetoric. A more direct route to cutting

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lung cancer, according to Rose, is simply to restrict the sale of tobacco-based products, the use of which is known to be highly correlated with incidence of lung cancer.

Before judging the adequacy of Rose's proposed intervention, we would need to know its ultimate aim. What is the curtailment of tobacco sales meant to eliminate: lung cancer per se, premature death, excessive healthcare costs, a certain kind of lifestyle, or the unpleasantness of a smoke-filled environment? In some of these cases, the intervention may have the efficacious results Rose seeks. But in others, tobacco may simply be replaced by another set of products that engender largely the same effects or even worse. Which is which depends just as much on a clarification of ends as on the presence of scientific knowledge and political will. Because Rose fails to provide this clarification – what philosophers call "axiology" – his conception of freedom, though defended with scientific evidence, does not have the objective character promised by his reliance on the Marxist motto.

Rose succeeds where Wilson fails

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Abstract: Rose's accomplishment in combining ontological unity with epistemological diversity contrasts it with Wilson's failure in overemphasizing the former and not appreciating the latter. This commentary cites the two most authoritative discussions of the inapplicability of heritability to human data, corrects several historical errors or inaccuracies in genetics, and criticizes the characterizations of Jacques Loeb and Robert Plomin.

I congratulate Professor Rose on a fine accomplishment. Though not perfect, *Lifelines* is a tour de force of good sense and packed with useful information, elegantly presented and well written. Not only does he give us an eloquent account of what modern behavioral biology had become and how it got there, but he has also taken the trouble to delineate what it is not: he has expended the time and effort to use Dawkins (and Dennett) as his foils.

Immediately prior to receiving this book, I had completed reviewing E. O. Wilson's 1998 *Consilience: The unity of knowledge* (Hirsch 1999). The contrast between the two books in their approaches to reductionism could not be more stark. In Rose's language, Wilson argues for an absolute ontological unity: "The central idea of the consilience world view is that all tangible phenomena, from the birth of stars to the working of social institutions, are based on material processes that are ultimately reducible, however long and tortuous the sequences, to the laws of physics" (Wilson 1998, p. 297). Wilson fails completely to appreciate what Rose correctly emphasizes: "Each level of organization of the universe has its own meanings, which disappear at lower levels" (p. 296); and "To understand the world's ontological unity we need the epistemological diversity that the different levels of explanation offer" (Précis, Ch. 4, last sentence).

In addition, Wilson repeatedly advocates heritability analyses: "Heritability . . . measure has considerable merit . . . is the backbone of human behavioral genetics . . . is a sound measure of the influence of genes on variation in existing environments. . . . Human behavior genetics is an infant field of study. . . . In only one level of analysis, the estimation of heritability, can it be said to be an advanced scientific discipline" (Wilson 1998, pp. 154, 155, 170).

Despite coming down on the side of the angels in his discussion of the misapplication of heritability analysis to human data, when a problem has already been handled effectively in the literature by recognized authorities, there is no excuse for Rose (or Wilson!) to ignore their work and to attempt not very effectively to re-invent the wheel. As I have previously emphasized in agreement with Cavalli-Sforza, "in science we all have an obligation to be familiar with the current state-of-the-art and to incorporate previous developments into whatever we do" (Hirsch 1997, p. 213). Today no one interested (pro or con) in human heritability can ignore the following analyses by the two distinguished statistical geneticists, Oscar Kempthorne and Albert Jacquard, and both published in the highly respected journal *Biometrics:*

> The idea that heritability is meaningful in the human mental and behavioral arena is attacked. The conclusion is that the heredity–IQ controversy has been a "tale full of sound and fury, signifying nothing." To suppose that one can establish effects of an intervention process when it does not occur in the data is plainly ludicrous. Mere observational studies can easily lead to stupidities, and it is suggested that this has happened in the heredity–IQ arena. The idea that there are racial–genetic differences in mental abilities and behavioral traits of humans is, at best, no more than idle speculation. (Kempthorne 1978, p. 1)

> The need for great rigour exists particularly in the case of research projects which have serious implications for us all; this is the case when psychologists study the "heritability of intellectual aptitudes." They should take the precaution of systematically defining in a precise way the sense in which they use the word "heritability"; they should also state whether the assumptions under which this word can be used hold true in their studies. It is highly probable that most of the time this exercise in rigour would lead them to the conclusion that none of the three parameters proposed by geneticists can be of any use in solving their problems. (Jacquard 1983, p. 476)

I must correct some historical errors and/or inaccuracies, which though unfortunate, do not vitiate the important message of Rose's book.

1. It is not correct that "colour-blindness or haemophilia . . . occur only in males" (p. 103). Because the gene correlates of these two traits are located on the human X-chromosome and are recessive, they occur far more frequently in males than females. But there certainly are affected women.

2. It is inaccurate to suggest that "Mendel's laws . . . were confirmed and extended during the 1920s and 1930s when Thomas Hunt Morgan and his team . . . found a suitable animal model . . . *Drosophila melanogaster*" (p. 110). It was in 1909 that Morgan started studying the fly and by 1915 he and his team had already published their first important book on the fly work, "*The mechanism of Mendelian heredity.*" By 1933 Morgan had been awarded the Nobel prize for that work. Rose's account of his fly eye-color experiment states "Morgan found some which seemed unusual . . . they had red rather than white eyes (p. 110). It should read the reverse: white rather than red eyes.

3. It is inaccurate to say that "the other reason why *Drosophila* were interesting to him was that their cells contained unusually large and readily visible chromosomes" (p. 111). The giant polytene chromosomes in the nuclei of the salivary glands of Drosophila and other dipteran larvae were unknown until described by Painter in 1933.

4. It is not historically correct that "*norm of reaction* . . . [is] a term originally introduced by . . . Theodosius Dobzhansky in the 1950s" (p. 133). It was first defined and introduced in 1909 by Richard Woltereck, professor of zoology at Leipzig (Dunn 1965, p. 96; Platt & Sanislow 1988, p. 254). It is certainly true that later Dobzhansky emphasized the importance of the concept.

5. After having characterized Richard Dawkins as "theoretician-polemicist," it is grossly unfair to call Jacques Loeb (p. 109) "the Dawkins" of his day. Merely because, from today's perspective, Loeb might appear to have exaggerated the organism-as-machine analogy and might have influenced Rockefeller funding policy, this is no justification for dismissing Loeb's entire career. He was a very productive, influential and highly respected scientist (an intimate and long-time [1891–1924] friend of T. H. Morgan, who certainly did not brook fools lightly). As reported: "Between 1901 and 1924 [year of his death], Loeb was proposed for

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a Nobel Prize by about a hundred sponsors in ten different countries" (Nobel Foundation, 1962, p. 256). For evaluations by colleagues (e.g., Osterhaut) and his students (e.g., Northrup, a Nobel laureate) who knew him well and/or collaborated with him see (Hirsch 1973, especially pp. xvi–xix).

6. To Bouchard's refusal to allow his data to be scrutinized by Rose and other scientists (p. 207) can be added the case of Robert Plomin discussed on p. 275 whose analyses Professor Peter Schoenemann (Purdue University) has been unable to examine, even after a member of the U.S. Congress (Collins, 22 June, 1995) repeated the request for him.

To conclude, this is the best treatment, with which I am familiar, of the limitations on reductionism, especially with the added integration of the very important concept of epistemological diversity.

Steven Rose's alternative to ultra-Darwinism

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Abstract: Stephen Rose's formulation of evolutionary theory is too scattered and impressionistic to serve as a genuine alternative to ultra-Darwinism. In addition, he has muddied a distinction that is crucial to our understanding of evolutionary phenomenona – the distinction between homologies and homoplasies.

Creationist and Marxist critics of present-day evolutionary theory have much in common. They both parody their opponents, but this argumentative strategy is common throughout science, not to mention every other intellectual discipline. Most of their works consist mainly in criticism. Constructive alternatives are notable for their absence. "If you find present-day versions of evolutionary theory so deficient, where is your alternative?" Phillip Johnson, the current spokesman for creationism, replies that he is not obligated to provide such an alternative. Steven Rose, a Marxist, thinks that criticism alone is not enough. "The challenge to the opponents of biological determinism is that, while we may have been effective in our critique of its reductionist claims, we have failed to offer a coherent alternative framework within which to interpret living processes" (p. ix). Rose wrote his *Lifelines* to meet this challenge.

How successful is Rose at providing an alternative to what he terms "ultra-Darwinism"? I am afraid not very. I agree with much of what Rose has to say and find many of his asides right on target. For example, he argues that the conventional distinction between science and technology is artificial. Building a bridge that stays up is a test of the theoretical principles used to build it, and developing a new instrument for conducting an experiment is as much a technological achievement as building a bridge (p. 57). According to Rose, science is more than individual scientists thinking up hypotheses and testing them. It is "*socially organized* hypothesis-making" (p. 66). In response to those who would reduce highly complex systems to the actions of their simplest parts, he notes that physicists are still unable to derive the properties of a substance as simple as water from the quantum states of its constituent atoms (p. 88). And, finally, he argues that Edelman's neural Darwinism is a "seductively misleading metaphor" (p. 144).

As much as I agree with all the preceding observations, I just as strongly reject other parts of his exposition. Some of my reservations concern minor points. For example, Rose repeats the tired old story of how male scientists have portrayed sperm as active agents in finding their way to fertilize passive ova. He states that it is "increasingly apparent that the ovum is not merely the passive recipient of the victorious sperm, but plays an active part in the process" (p. 152), a view that has been commonplace for over half a century. But some stories are too good not to keep retelling even if they are not true. As we all know, lemmings march to the sea to commit mass suicide, the good-tasting viceroy butterfly protects itself from its predators by mimicking the bad-tasting monarch, and male praying mantises succeed in copulating with female mantises by offering the upper parts of their body as food while the lower parts do their business. That none of these stories is true seems to bother no one. To add to this list, doubt has been recently cast on the most famous example of all – industrial melanism in the peppered moth.

But not all of my reservations with respect to Rose's book are so minor. Through the years I have come to realize that the distinction made by biologists between homologies and homoplasies is fundamental to our understanding of natural processes. We can perceive regularities and patterns in nature. Some of them are due to common descent; for example, the spiracle in sharks is homologous to the Eustachian tube in humans. But other patterns do not depend on genealogical relations. Some are due to the performance of common functions in similar processes; for example, a torpedo-like shape is common among aquatic organisms that spend a good deal of time swimming. The similarity in shape among so many aquatic organisms is homoplastic. If one wants to reconstruct phylogenies, homologies are the message and homoplasies are noise. If one wants to understand the evolutionary process, homoplasies supply the message and homologies are noise.

Rose succeeds in obliterating this important distinction. For Rose, "homologies" imply a "deeper identity, derived from an assumed common evolutionary origin" (p. 34), but then he goes on to offer two alternatives to homologies – metaphors and analogies. In metaphors, phenomena that occur in different domains are compared; for example, the energy flow controlled by ATP and the flow of currency in a bank. Analogies concern superficial resemblances between two phenomena; for example, the blood circulates in animals as sap circulates through plants. I am afraid I fail to see any significant differences between metaphors and analogies as Rose defines them. But more important, he leaves out those entities that are deemed similar because they perform the same function in similar processes or, as they are called in the philosophical literature, natural kinds. Nature can be subdivided into kinds in numerous different ways. The task is to find kinds that function in natural regularities (commonly termed laws of nature).

Rose admits that within the world of human artifacts "it may not be unreasonable to try to seek the essence of, say, a table or chair" (p. 35). Such an essentialist view of the world "may even be possible when one is studying inanimate phenomena such as comets, electrons or chemical elements" (p. 35). But the living world, so he claims, cannot be subdivided into kinds that function in laws of nature. He first argues that lineages such as species and subspecies are not natural kinds because they are temporary, spatiotemporally restricted, and have at best fuzzy boundaries in conceptual space. I could not agree more.

I have been arguing this position almost as long as Ernst Mayr and Michael Ghiselin. Rose then turns his attention to individual organisms. They are not natural kinds and for the same reasons. Finally, Rose moves to the molecular level arguing that proteins and enzymes are not natural kinds either because they must be functionally, rather than structurally defined. He does not say why functionally defined kinds cannot function as natural kinds. But, more important, he ignores all the genuine kinds to be found in ecology (such as carnivores, herbivores, and parasites) and evolutionary biology (such as founder populations, peripheral isolates, and possibly the species category itself).

But to return to the opening paragraph of this review: Does Rose provide anything like a coherent alternative to ultra-Darwinism? As far as I can judge, he has not.

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Selection and "freedom" in biology and psychology

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Abstract: Rose provides a coherent account of how a number of simplifying assumptions apparently come together to support neurogenetic determinism, or "ultra-Darwinism." This view, he demonstrates, is deeply flawed. He proposes instead that we must take account of the interaction of processes that determine our developmental trajectory at every stage. Unfortunately, he associates this defensible position with the claim that this gives freedom of action to humans. The implications of this for the interpretation of his general thesis are discussed.

The greatest obstacle to progress in the behavioural and brain sciences is the use of inappropriate theoretical models. In the Anglo-Saxon tradition, we often fail to see the need to articulate the unstated assumptions which underpin our proposed explanations in specific areas. In this book, as previously, Rose does a great service by attending meticulously to questions of underlying assumptions. Some of these are summarised, late in the book, in his succinct summary of neurogenetic determinism.

In Rose's view, neurogenetic determinism involves the steps of "reification, arbitrary agglomeration, improper quantification, belief in statistical 'normality', spurious localization, misplaced causality, dichotomous partitioning between genetic and environmental causes, and the confounding of metaphor with homology." (p. 279). Behaviour analysts, from Skinner (e.g., 1950) onwards, have shared many of these concerns. One of Rose's strengths is his clear recognition that from a general belief in unified science – the belief that phenomena at one level can in principle be translated into phenomena at "lower" levels – nothing in particular follows about preferred levels of description and explanation. Rather, each level has its own explanatory system and particular purposes which it can achieve. In this context, behavioral accounts of psychological phenomena may, for example, be seen as independent of, rather than poor substitutes for, psychological or genetic accounts of human behaviour. Also, as Rose illustrates, there are aspects of psychological phenomena which exist solely at that level and cannot be related to direct genetic influence.

There is much to applaud here, as elsewhere in *Lifelines.* There are many themes in this impressive and exciting book which chime with the selectionist and functional-analytic themes of contemporary behaviour analysis. This may lay the groundwork for the analogy between natural selection as an evolutionary mechanism for species and the selection of behaviour in the individual by its consequences (see Skinner 1969, Ch. 7) to be extended.

Much public discussion of the behavioural and brain sciences, in broadcast media as well as in print, is dominated by those who would persuade us that we need only one level of explanation, and one explanatory principle. Throughout this book, Rose points up the inadequacy of these tactics in general and as used by neurogenetic determinists in particular. This is a task well worth doing, and it is done well. However, as with his earlier treatment of some related issues (Rose et al. 1984), I am left uneasy as to how nonspecialists (or even specialists) will interpret its implications for the psychological explanation of human behaviour. The problems arise out of Rose's concern to point out the limitations of excessive reliance on natural selection at the level of the single gene to explain all biological phenomena.

Let us examine Rose's summary of his own case against neurogenetic determinism or "ultra-Darwinism" (p. 215). There are four claims:

1. Selection occurs at levels other than that of the single gene.
2. Natural selection is only one of several factors producing evolutionary change.
3. Organisms are not indefinitely responsive to change.
4. In Rose's words: "Organisms are not mere passive responders to selective forces, but active players in their own destiny."

While accepting Rose's assertion that his "principal target is the dogmatic gene's-eye view of the world," I want to consider Rose's account of the fourth claim which will tend to catch the attention of all general readers as well as that of psychologists. With this fourth claim he allows for the possibility that conventional mentalism may take the place of rigorous philosophical and scientific analysis. Very early in the book, he sets the stage for this disappointing coda. There is an assertion that: "The central property of all life is the capacity and necessity to build, maintain and preserve itself, a process known as *autopoiesis.* This why . . . we, as living organisms and specifically as humans, are free agents." (p. 18). Along with this (p. 9), he has specifically included mentalistic psychology as one level of explanation in a critique of the notion of the hierarchy of the sciences. Taking the book as a whole, then, it is possible for the reader to conclude that Rose concurs with those nonscientific commentators who wish to believe that a deterministic and scientific account of human psychology is not possible.

I do not think this is Rose's position. The problem is a subtle one of presentation. In a further account of autopoiesis (p. 245), he is at pains to distinguish his account from one in which purpose directs evolutionary change: "Autopoiesis, organisms as active players, is as apparent when a single-celled organisms swims away from a depleted food source towards a richer one, as . . . in the decision of an impoverished Mexican to cross the border into California" (p. 245). This comparative analysis is really no different from that used by Skinner (1972), who noted that the behaviour of many species including humans is modified by contingencies of reinforcement. Furthermore, although humans prefer to live within social systems that positively reinforce prosocial behaviour rather than those that punish antisocial behaviour, they are never "free" in the conventional sense of that word. Rather, their behaviour is selected by the prevailing contingencies of reinforcement. Skinner concluded that, in Western traditions, the aspiration "to be free" meant "to be free from aversive control." Rose says that we are "free," but he actually means that we are influenced by a complex set of forces, and no one of these can be said to determine our eventual behavioural state or psychological characteristics. As with other biological phenomena, human behaviour can only be understood in terms of the interaction of many processes over the lifespan.

Genetic and biological determinants of psychological traits

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Abstract: Rose seems to be arguing against an extreme ultra-Darwinism that probably has no adherents. He incorrectly argues that a number of psychological traits are very difficult to measure. This is not the case. Rose argues that intelligence has no biological correlates. In fact, it is correlated with brain size, EEG evoked potentials, and cerebral glucose uptake during problem solving. Data that Rose should be aware of are omitted when they do not fit the case he is trying to make.

Rose raises various objections to sociobiology and what he calls ultra-Darwinism. By the latter, he means a reductionist, determinist biology which, among other things, attributes a number of traits such as intelligence to genetic causes and, at least in some versions, claims that the only point of organisms is to replicate genes. His refutation fails for a number of reasons. Other commentators will doubtless point them out. I restrict myself to comments on his remarks on behavioral genetics.

Rose does not deny that there are biological determinants of traits such as intelligence. Environmentalists have surrendered on this issue (Sternberg & Grigorenko 1997). They have been reduced to quibbling about the proportion of variation in intelli-

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gence that can be attributed to genetics. Their preferred estimate is around 40% as opposed to hereditarians' preferred estimate of at least 70%. Rose tries various tricks to discredit the concept of intelligence. On page 69, he lumps together the idea that IQ tests measure "some fixed, biologically determined feature of an individual" with the ideas that the moon is made of cheese and that the earth is flat. In fact, because intelligence involves speed of mental processing, reaction time on a variety of tasks is correlated with IQ (Jensen 1982). More telling, there are reliable differences between more and less intelligent people in glucose uptake in the brain during problem solving (Haier et al. 1988). EEG evoked potentials are related to intelligence (Caryl 1994). Finally, there is no longer any question that intelligence is related to brain size (Jensen & Sinha 1993). Like it or not, intelligence is related to biology. It is difficult to imagine how one could learn one's brain size or glucose uptake rate; it is quite easy to see how such things could result from genetic factors.

On p. 190 we are told that intelligence is not easy to measure, and it is implied that it is as difficult to measure as political tendency, religiosity, job satisfaction, and so on. The latter traits are easier to measure than intelligence, but all can be quite reliably and validly measured. The possibility that Sir Cyril Burt's data on the heritability of intelligence may have been fraudulent is mentioned on p. 191. Rose neglects to mention that Burt's heritability estimate for IQ is about the same as that found in dozens of other studies in which no fraud has ever been alleged.

Another disquieting omission concerns the fact that Thomas Bouchard, who is involved in the largest study of twins reared apart, would not allow Rose to see his primary data. This was probably due to U.S. laws regarding confidentiality. Were I to do an innocuous study of preference for polygons, I could not show Rose my primary data. I would first have to remove all information concerning the identity of the participants. There are many studies of twins reared apart in countries with laws different from those in the United States. Looking at the primary data will not change the facts they contain.

The standard formula for computing heritability only works if genetic effects are additive. [See Wahlsten: "Insensitivity of the Analysis of Variance to Heredity – Environment Interaction" *BBS* 13(1) 1990.] However, Rose does not tell us that concordance rates (if one twin has a trait does the other also have it?) provide about the same information as heritability per se. If concordance is higher in monozygotic than in dizygotic twins, this is evidence for a genetic contribution to the trait. This is especially true if the twins were reared apart. Rose may not like it that there is a large genetic component in traits as diverse as attitude toward the death penalty and probability of divorce. To try to explain the findings away by saying that they arose from the use of statistics originally developed for plant and animal breeding will not do. Many statistics used in psychology are derived from or identical to statistics developed in agriculture. The numbers don't know what they are about; it would have been pointless to re-invent statistical wheels.

Attributing findings in behavior genetics to the statistics used does not explain why some things are consistently found to be genetic and others not. For example, anyone who does a study of manic-depressive illness will find a concordance rate of about 80% for monozygotic twins and 20% for dizygotic twins (Goodwin & Jamison 1990). Probably anyone studying attitudes toward pajama parties would find a concordance rate of about zero for both types of twins. We can think of paths from proteins to psychological traits. It is difficult to see why statistics would consistently associate some traits but not others.

Some misunderstandings and misinterpretations about sociobiology and behavior genetics in *Lifelines* by Steven Rose

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Abstract: *Lifelines* by Steven Rose is supposed to present a new perspective on biology replacing an emphasis on genes with one on organisms. However, much of the book is a highly biased critique of sociobiology and behavior genetics. Some of the flaws in Rose's description and depiction of these fields are presented and refuted. Also, it would appear that these aspects of the book and many others are, in fact, related more to Rose's perennial concern for the ideology, social origins or social consequences of behavioral biology. These concerns are, I believe based, in part, upon Rose's misunderstandings and misinterpretations of genetics, behavior genetics, and sociobiology.

Lifelines by Steven Rose is supposed to present a new perspective on biology which emphasizes organisms rather than genes. However, much of the book is a biased critique of sociobiology and behavior genetics.

Wilson defined sociobiology as "the systematic study of the biological basis of all social behavior" (1975, p. 4), and he included in it both proximate and ultimate explanations for the causes of social behaviors. These are similar to the five types of explanations described on pages 10–14 of Rose's book. One of these focuses on whether or not a social behavior is adaptive, and whether or not the adaptation is due to natural selection acting in the past on heritable variants.

Those concerned with the origin of behavioral adaptations are rarely interested in the exact details of their genetics (Barkow et al. 1992; Krebs & Davies 1993). However, Rose suggests that ultra-Darwinian sociobiologists do not recognize a possible role of epistatic interactions among genes in effects of selection on the evolution of social behaviors. This is false, at least for the following sociobiologists. Wilson (1975, p. 70) writes, "Real selection, however, is directed not at genes but at individuals, containing on the order of tens of thousands of genes or more, and . . . relatively small amounts of interactions between loci can generate sufficiently tight linkage disequilibria to make the entire chromosome respond to selection as a unit." These are not the words of Rose's ultra-Darwinist. Similarly, Dawkins (1982, p. 111) writes, "The statistical structure of the gene-pool sets up climate or environment which affects the success of any one gene relative to its alleles. . . . The point is the obvious one that selection at one locus is not independent of selection at other loci." Again, these are not the statements of Rose's ultra-Darwinist. Yet Wilson and Dawkins are, for Rose, archetypes of ultra-Darwinian sociobiologists.

Rose also suggests that they believe that all behaviors are adaptations due to effects of natural selection acting on heritable variants, and that there is no other explanation for the evolution of behavior. However, Dawkins (1982, p. 29) writes "This is not to say that all behaviour patterns necessarily have a Darwinian function. It may be that there is a large class of behaviour patterns which are selectively neutral or deleterious to their performers, and can not usefully be regarded as products of natural selection." He goes on to his own "critique of naive adaptationism" and his own list of constraints on the evolution of adaptations, including those for behavior. Similar material is discussed by Wilson (1975) with regard to phylogenetic inertia as a constraint on adaptations. He also considers the role of inbreeding and genetic drift in evolution, including that of behavior. Regardless, even Rose accepts the possibility that some behaviors are adaptations due to natural selection acting on heritable variations. But he seems to believe that most if not all involve selection at the group rather than the individual level.

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Rose appears to be more concerned with claims for behavioral adaptations in humans than in animals. He suggest that ultra-Darwinian sociobiologists, such as E. O. Wilson and R. Dawkins, believe that altruism and other social behaviors in humans are an effect of kin selection acting in the past on heritable variants, and that they reject cultural explanations for human social behaviors. However, this is not their position. Wilson (1975) recognizes four peaks in the evolution of social behavior. These occur in the colonial invertebrates, the eusocial insects, nonhuman mammals, and humans. He believes that kin selection was the most likely involved in the evolution of social behavior of colonial invertebrates and eusocial insects, and that it plays little or no role in the evolution of social behavior in nonhuman mammals or humans. In 1975 (p. 381), he writes, "The requisite refinement and personalization in vertebrate relationships are achieved by . . . a greater role of learning" and in 1978 (pp. 153 and 156), he writes, "Human social evolution is obviously more cultural than genetic" and "Through convention of reciprocation, combined with a flexible, endlessly productive language and a genius for verbal classification, human beings fashion long-remembered agreements upon which cultures and civilizations can be built." Similarly, Dawkins (1976) recognizes the role of learning and culture in human behavior and adaptations; To better understand their role, he developed the concept of the "meme."

In contrast to most sociobiologists, most behavior geneticists are concerned with the details of the genetics that are involved in individual differences in adaptive and nonadaptive behaviors in laboratory and natural populations of many organisms, including bacteria, protozoans, round worms, mollusks, fruit flies, and other arthropods, fish, amphibians, reptiles, birds, and mammals. However, most of the emphasis has been as elsewhere in genetics on round worms, fruit flies, mice, and humans. Since many variations in many behavioral traits are due to variation in alleles of more than one gene, the concepts and methods of quantitative genetics, which had their beginnings not only in the works of Fisher (1930) but also Wright (1968), have been widely used. This partitions the phenotypic variance into genetic and environmental components. The genetic variance is again partitioned into additive, dominance, and interaction components. All three components can be and have been assessed in quantitative genetic analyses of animal and human behaviors (Plomin et al. 1997; Rose 1994).

As Rose recognizes, these components can be used to estimate the broad (genetic variance/phenotypic variance) or narrow (additive variance/total variance) heritability of behaviors in animals. This has been done by Dobzhansky (1968), among others. It would appear that the biochemical, genetic, cellular, and developmental complexities and interactions which occur in both animals and humans would not be, for Rose, problematical in estimates of heritability for not only animal but also human behaviors. However, as Rose indicates, genetic and environmental effects on individual differences may be confounded in studies of humans but not animals. Adoption studies of individuals and of twins raised apart can address this problem (Plomin et al. 1997; Rose 1994).

Rose does not directly critique the latter method. Rather, he makes misleading statements about the studies of Thomas Bouchard on twins reared apart. He says that Bouchard and his colleagues have published their finding in obscure, unrefereed journals. However, Bouchard and his colleagues have published their findings in *Science* (Bouchard et al. 1990), *The Journal of Personality* (Bouchard & McGue 1990), and the *American Journal of Psychiatry* (Segal et al. 1990). He also claims that Bouchard has refused him and others access to the primary data, but he neglects to mention that both informed consent agreements and federal law prohibit release of primary data which would identify individuals in this study (Wright 1998).

Lifelines is a difficult book to read and to review. Among the reasons for this are:

1. Rose frequently makes disparaging personal comments about those that he disagrees with. For example, he writes (p. 5) of R. Dawkins, "with all the brash style of a cheeky adolescent cocking a snook at everything his elders hold dear." Similarly, he describes the young James Watson as ambitious and bumptious.

2. It is replete with factual errors. For example, on page 4, Rose writes, "genetic generalizations are still derived from just three organisms, the rat. . . ." Actually mice and not rats are the primary mammalian organism used in genetic research. He also overlooks the contributions of C. elegans and all plants to genetics. Also, he claims that an article of mine (Maxson 1996) argues for genes with effects on mouse killing by rats as a model for human aggression. In fact, my paper is not about interspecific aggression of rats but rather about intraspecific aggression of mice as animal models.

3. As already indicated, his comments on and critiques of sociobiology and behavioral genetics are seriously flawed. There are similar errors with regard to population genetics. For example, he writes (p. 216), "Fisher and Haldane's approach was derided as 'beanbag' genetics precisely because it depended for its mathematics on the assumption that each gene was an isolated unit." In fact, Fisher, Haldane, and Wright were aware of the existence of gene interactions, and the difference among the three was not with regard to the fact of gene interactions but rather with regard to the most effective population size for adaptive evolution (Provine 1971). Also, Lewontin (1998, p. 62) writes, "Thus, theory had a bad name . . . as bean bag genetics, solely concerned with individual genes. This reaction has hidden the essential contribution of Wright in 1931 and of Fisher in 1918. They were interested in coping with gene interactions from the very beginning."

4. Rose's book seems to be as much, if not more, about biology and politics. Although he writes (p. xi), "I largely refrain from discussing the ideology, social origins or social consequences of ultra-Darwinism and reductionism," he frequently takes up this topic as on pages 73–74, 115–16, 192, 212, and 272–301. These detract from the development of his theory of *Lifelines,* and it may account for the absence in his book of some relevant and significant topics such as homeobox genes and development (Gehring 1998).

A holistic developmental theory requires better research techniques

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Abstract: Research pragmatics, not a defective conceptual framework, supports modern biological reductionism. Conducting research to reveal the casual web underlying the multiple developmental pathways leading to any species-specific characteristic requires better research techniques than those commonly used. It takes much patience, time, and effort to gain even small glimpses of an answer to any developmental question.

Lifelines presents a coherent account of living systems that integrates physics, chemistry, and biology in a way that is intended to undercut any attempt to use only reductionistic explanations of the human condition. As Rose notes, deterministic biological explanations resurfaced during the late 1960s without any specific advance in biological science or theory. However, they have been advanced (to the dismay of thoughtful scientists) by recent biomedical and popular interpretations of modern molecular genetics and neuroscience. Rose argues that the reductionist trend stems from both a tradition of determinism in biological training and thinking and the failure of "opponents of biological determinism . . . to offer a coherent alternative framework within which to interpret living processes" (p. ix). However, many such frameworks have been proposed during the last 100 years and several quite recently (cf. Elman et al. 1996; Gottlieb et al. 1998; Michel & Moore 1995). Why then does reductionism persist?

The reductionist tradition certainly influences the way many biologically oriented investigators pose research questions, apply re-

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search techniques, interpret findings, and train students. It does so because this is more efficient in generating research results and providing ready interpretations than research that "celebrates complexity" and focuses on the specific dynamics of the organism-environment system over its life-span. While I appreciate Rose's efforts, biological determinism will continue to dominate because unraveling the organismic-experiential causal web underlying the multiple developmental pathways of species-typical characteristics is not easily done, nor is there a generally accepted set of techniques for doing so.

In contrast, researchers in neuroscience, molecular and behavior genetics, sociobiology, and cognitive science, have shown that it is relatively easy to conduct research on questions of proximate causation, phylogenetic relationships, and adaptive function. There are many ways to investigate the contributions of genes and various neural processes and structures to behavior and many of these involve exciting new technologies to attract popular interest. Of course, only reductionism assumes the nervous system to be organized by gene-controlled processes (Rose nicely illuminates the difference between genetic involvement and genetic control). Sociobiology has demonstrated the efficiency of collecting data that address alternative game-theoretical or economic models of generalized types of behavior, presumably controlled by genes. Such results fit current thinking about adaptation and natural selection so well that they tempt researchers away from pursuing developmental questions.

In contrast, typical developmental research techniques are limited and problematic. Many studies involve correlating individual differences earlier and later in development. This does not reveal whether the individuals have changed between measurement periods (a developmental event). Nor do these studies reveal the developmental processes that contribute to maintaining relative position across age periods. The absence of correlation may be interpreted by reductionists as indicating that early developmental events have no impact on later events because they are biologically controlled. Alternatively, high correlation may be interpreted as strong biological control during that age period.

Other developmental techniques examine the relation between a manipulation (or clinical event) occurring earlier in development and the individual's characteristics later in development. Even if a relation exists, nothing is revealed about the causal web connecting the two developmental periods. If a relation does not exist, nothing is revealed about the processes that blocked the connection.

A common technique of depriving an organism of certain experiences to determine their influence on development presumes that one already knows or suspects that such experiences contribute to development. A few carefully crafted programmatic studies have revealed that development can depend upon experiences which do not at first appear to be relevant. Thus, the absence of deprivation effects only means that the missing experiences were not relevant for that developmental outcome, or if they were, that their loss could be compensated for by other means. For example, in some bird species, song can develop without hearing other singing birds or the bird's own singing, but that does not rule out other auditory experience. Even if for some species all auditory experience were ruled out, other experiences (e.g., vestibular and/or respiratory experience, or specific social experiences) could be involved (Clayton 1994). Thus, although deprivation studies can fail to reveal how develop occurs, they can support reductionist interpretations.

Since models of learning often fail to provide insight into the development of many species-typical characteristics, some argue that individual experience is unimportant for them. However, learning models represent only a small part of the range of experiential events that contribute to development, some of them seemingly irrelevant. Most stimulation studies fail to identify the experiential influences on development because they provide either a broad-band level of stimulation that approximates those that are characteristic of the natural environment more than the usual laboratory situation, or they provide unusually high levels of very complex stimulation. No one would deny that approximating a normal environment can support normal development or that normal development can be disrupted by stress or overstimulation. Hence such studies do not challenge reductionism. Only a few studies (e.g., bird-song, imprinting, sex differences in rat behavior) have manipulated levels and patterns of stimulation within the bounds of their normal occurrence to identify their contribution to the development of species-typical behavior (cf. Gottlieb et al. 1998; Ten Cate 1994; Moore 1992). In each case, such experiences are profoundly involved in the normal development of the species-typical behavior. [See also Baker: "The Biology of Bird-Song Dialects" *BBS* 8(1) 1985; Johnston: "Development, Explanation and the Ontogeny of Bird Song–Nature" *BBS* 11(4) 1988.]

Careful, time-consuming, programmatic investigations need to be encouraged if we are to understand development and avoid what some see as the pitfalls of reductionism.

Determinism, omniscience, and the multiplicity of explanations

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Abstract: Complete determinism is, as Karl Popper said, "a daydream of omniscience." Determinism is usually conceived as linked with a particular science whose explanations are deemed fundamental. As Rose rightly points out, biological enquiry includes many different kinds of question. Genetic determinism, making genes central to biology, is therefore biased and misguided. The crucial unit must be the whole organism.

The clash between determinism and free will is not, as is often said, a hopeless, unavoidable philosophical deadlock. Mostly it is simply the product of an arbitrary, vague, overambitious concept of determinism. Karl Popper wrote the epitaph of that concept, observing, "Physical determinism . . . was *a day-dream of omniscience* which seemed to become more real with every advance of physics until it became an apparently inescapable nightmare."[2] He meant, of course, that claiming omniscience turned out to be incompatible with human freedom. We now know, too, that this slapdash deterministic claim has faults that go far beyond this particular inconvenience. Within physics itself, the traditional idea of determinism is now openly scheduled for rethinking.

However, the trouble is not confined to physics. The need to rethink determinism in biology is even more urgent, though it has not yet been so fully understood. *Lifelines* firmly grasps this alarming nettle. It both shows the urgency of the need and suggests constructively the general lines on which we might meet it.

Rose attends sharply to metaphors, which are of course the main channel through which such large-scale concepts shape our thinking. Biologists (he says) should insist that organisms *make themselves* rather than being helpless objects driven by the chemicals that compose them – rather than being, for instance, "the vehicles of their genes." Of course, both these formulations are metaphors drawn from the same human concept of agency. But the first formulation needs to be sharply stressed today in order to balance the second, which has lately got entirely out of hand.

The active-passive story has been used to give a bizarre dramatic twist to the notion of causality, suggesting a transaction where a genuinely active entity drives an inert, passive one rather than a neutral process within which they both play their part. Thus (as Rose points out), recent talk about genes often seems to abstract from their own causal context altogether, treating them as if they were somehow autonomous uncaused causes, prime movers in the evolutionary game.

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Current notions of determinism easily encourage this kind of one-sidedness, because the determinist framework is usually linked with a particular study, thus giving that study's favoured entities a strange kind of privileged causal force. We see many kinds of hyphenated determinism, of which Skinner's social-determinism was a striking example. Others have been economic-, physical- or neurogenetic. Each variant claims that its chosen study provides the most fundamental form of understanding available, that the causes it deals with are the only ultimately effective causes. These hyphenated determinisms are, then, necessarily biased. They are *reductive* in the general sense of downgrading other studies to secondary, provisional status. They embody the ambitious Enlightenment project of providing a single unified explanation of all phenomena – Popper's "day-dream of omniscience" – in the simplest possible way, by trying to conquer their neighbours in the name of unification. They try to streamline all our multifarious thought-forms into unity by drilling them into various formal hierarchies.

As Rose points out, however, the plurality of thought-forms is not a luxury. It mirrors real complexity in the world. Explanations take different forms because we need to answer many different kinds of question, and "the reason for asking the question will determine the most useful type of answer" (p. 14). Even within biology itself, many quite different kinds of question arise, simply because life is such a complex phenomenon. Ecology, ethology, and developmental biology ask their own distinctive kinds of question, often large ones. It is not sensible to treat these questions as somehow *less scientific* than those of genetics or neurology and to try to reduce them to those sciences.

If, however, we accept this plurality of questions, with the variety of methods for answering that it demands, we see that biology as a whole – indeed science as a whole – could never be, and is not meant to be, the kind of single vast, elegant, infallible, monistic calculation that unifying rationalists have visualised. It must always be a fallible co-operative enterprise, an ongoing, fluid, pragmatic compromise between many points of view. Those many points of view, if taken seriously, give us a far better understanding of the complex world around us than we could hope to get from any formally unified study.

All this is no disaster. It simply repeats what every working scientist knows about the difficulties of unifying knowledge: that there is a constant dialectic between the unifications of theory and the complexity of the facts, a dialectic which must never be allowed to degenerate into outright conquest. In fact, it only means that science is, after all, a human institution. That is why the dream of a tidy omniscience was unrealistic and never needed. That is why, as Rose puts it, "far from being determined, or needing to invoke some non-material concept of free will to help us escape the determinist trap, it is in the nature of living systems to be radically indeterminate, to continually construct their – our – own futures, albeit in circumstances not of our own choosing" (p. 7).

We live, then, in a world too complex to be investigated by any single method, one in which no single form of order determines what will happen. Though living things are in many ways orderly and intelligible to us, each of them is a unique individual with its own tendencies and is also subject to the constant play of chance. Complete predictability is not, even in principle, possible or necessary. "It is just this combination of predictability and unpredictability that distinguishes living systems and processes from the much simpler events that form the terrain of the sciences of physics and chemistry" (p. 154). That is why we must use "a perspective on biology which transcends genetic reductionism, by placing the organism, rather than the gene, at the centre of life" (p. x).

Rose is surely right. This conceptual shift is indeed overdue.

NOTES

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2. Karl Popper (1972) Of clocks and clouds. In: *Objective knowledge, an evolutionary approach.* Oxford University Press.

Biological determinism versus the concept of a person

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Abstract: Rose presents an important critique of the determinism and reductionism of modern biology. However, such trends are probably temporary aberrations in the development of science. Another form of determinism which has deeper roots is emerging from modern studies of brain dynamics. To reconcile this evidence with the concept of a "person" will require more radical rethinking of our received notion of natural law.

Steven Rose's *Lifelines* is a critique of modern biological sciences, aimed particularly at the reductionism and determinism which prevails today in many areas of research and popular science writing. Rose's underlying philosophy is Marxist and therefore materialist. Although I do not share either of these perspectives (Miller 1995), I have much sympathy for his critique of modern biology.

Amongst the areas of research emphasised in Rose's book is psychiatric genetics. I recently studied the evidence on the genetics of schizophrenia and was shocked by the naivete of molecular genetic studies of schizophrenia. Traditional psychiatric genetics has produced abundant evidence that mental disorders such as schizophrenia are in part determined genetically. This evidence points strongly to the view that many (perhaps very many) genetic factors are involved. Probably the genetic aspects of the disorder are relatively rare combinations of many individual genes, all of which are common, and in themselves probably quite benign. In molecular psychiatric genetics, however, the goal is usually to find "the gene" or "genes of major influence." This search, on which vast resources have been lavished, seems to assume that schizophrenia is a clearly defined category, so that it can be defined genetically in terms of one or a very few genetic factors. This betrays gross ignorance – both of the large body of evidence about schizophrenia favouring a dimensional rather than a categorical definition for the disorder, and of the traditional psychiatric genetics. Its implicit assumption "one gene – one psychological characteristic" or "one gene – one diagnosis" seems very similar to the underlying assumption of phrenology, and is not much more sophisticated conceptually. This work certainly fits well into Rose's critique of reductionism and determinism.

Rose also suggests that the trends in modern biology have an ideological basis. My views on this are complex, but in partial agreement. On the one hand, there are many contradictions in modern times which suggest that modern biology does not have any coherent ideological driving force. For instance, biology now tends to emphasise determinism, in curious contrast with other aspects of the contemporary world view that emphasise individuality, free enterprise, and personal responsibility. On the other hand, prevailing ideas about Darwinism or ultra-Darwinism do have their main impact not in terms of any obvious technological advance, but in promoting a view of human beings as genetic machines in competition. This view could be regarded as ideological.

I take all this to represent the fundamentalisms of our times; in thirty years time (one hopes) we will look back on the 1990s with amazement that scientists could have fallen for such crass attitudes. Thus the reductionism and determinism Rose criticizes may be a temporary aberration, not part of the main stream of scientific thought going back to the Renaissance.

In passing, Rose also regrets the fact that the physical sciences are taken as models for biology. In this he may be mistaken. The achievements of Galileo and Newton cannot be simply regarded as intellectual developments determined by the socio-cultural demands of their time. They provided us with the historic hypothesis that natural laws are a more fundamental description of the world than those based on final causes; they also provide a framework for quantitative description of the world based on mass,

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length, and time (with some elaborations added later). This descriptive language is quite fundamental to the scientific enterprise.

The notion of natural law was at first a tentative hypothesis. However, in the centuries after Newton, it has become a dogma, to which it seems (especially in biology) there can nowadays be no exception. This leads to two major points, one relating to reductionism, the other to determinism.

The term "reductionism" refers to two rather different types of explanatory arguments. The type criticised by Rose arises in part from a certain type of experimental design. The explanatory arguments which follow, such as they are, are often unsatisfactory because the higher-level phenomena to be explained are simplified to the point of caricature, rather than being considered in more realistic detail. Rose's book stresses the dynamic complexity of living things, which is not seen until one manages to avoid the oversimplification produced by reductionist experimental designs.

Nevertheless, within this complexity, a second, much better type of explanatory argument can be constructed, crossing between levels of description, such as some of those found in physics. Examples from physics include the hypothesis of atoms, based on the weight- or volume-ratios of combining substances, or the explanation of the gas laws in terms of the dynamics of colliding gas molecules. Similar kinds of explanation in biophysics are those of Hodgkin/Huxley relating the action potential to ionic fluxes. Explanations of this types are now also becoming possible for higher brain functions, thereby crossing between the neurobiological and the psychological levels, even for the human brain. Admittedly, in brain biology, such explanations often need to be framed in a somewhat different manner. They rely more on informal arguments and comprehensive scholarship than on elegant mathematical demonstrations. In addition, it is necessary to "build up" from what is actually known at the lower level, as well as to "build down" from what is known at the higher level, in order to frame explanations which cross between levels. Nevertheless, the principle of cross-level explanation is the same as in physics. When it is successful in brain biology, it is at the same time reductionist and holistic and does more justice to the complexity of the high-level phenomena to be explained than accounts which simply look for lower level explanations of what one observes, or the simplistic "reductionism" criticized by Rose.

Arising from this second type of explanation, is a variety of neural determinism based on much more robust science; the growing field of forebrain neurodynamics. This offers a much more solidly based determinism, deriving directly from the biophysics of single nerve cells, and their coming together as cell assemblies. This work is likely to be more enduring, and more difficult to shrug off as a temporary aberration. If we take the concept of natural law as a dogma, a principle to which there can be no exceptions, this work on neural dynamics is implicitly undermining the concept of a person in a far more fundamental way than does the science Rose decries. The concept of a person, though not a scientific one, is very important, arguably more so than the scientific enterprise itself. Hence we should try to find a way to escape from the apparent determinism of modern brain dynamics. To do this, we need to re-examine our notion of causality, and to reclaim the idea of natural law as a tentative hypothesis, rather than as a rigid law to which there can be no exceptions.

The implications of this would be profound. In the early days of Renaissance science, when the idea of natural law was a tentative hypothesis, there were no attempts to provide scientific explanations of history. This only came later (in the years leading up to the French revolution). As natural law became a dogma, theories became more confident and attempted to explain history as well as the here-and-now, or what was seen in the laboratory. If we are to reclaim the idea of natural law as a tentative hypothesis rather than the Orthodox Dogma of our present Brave New World, to which we must all subscribe, we will need to re-examine the status of arguments purporting to provide scientific explanations of history. This includes both Darwinism and Marxism.

Facing complexity: Against scientific oversimplification

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Abstract: Steven Rose's book is essentially a plea for considering the variety and complexity of life and against simplistic reductions of human and animal behavioral phenomena to single genetic causes.

Lifelines by Steven Rose is interesting and entertaining to read. It covers many areas of biology ranging from molecular biology to genetics and development. But first and foremost, the author has a definite case and argues for it. The main targets of the book are (1) simple genetic determinism, according to which our prospects in life are determined by our genes, and (2) a simplistic understanding of reductionism according to which all of biology and even human sociology can ultimately be reduced to the physics of the molecules and atoms we are composed of. I strongly sympathize with most of the arguments in the book, but I am less happy with some of the philosophical overtones and some exaggerations which show that the author is sometimes a little too eager to have an argument with other scientists. For example, I must admit that I enjoyed reading Dawkins's "The selfish gene" about as much as this book; I did not see Dawkins as guilty of the scientific oversimplifications about "gay genes" and the like that are the main target of Rose's book and that are apparently gaining ground in some scientific communities and even in the prestigious unspecialized journals *Science* and *Nature.*

I was also unaware of the magnitude of the danger to our human self-esteem and the potential sociopolitical consequences of these ideas. Being trained in mathematics and cybernetics, I used to consider it obvious that assuming that biological phenomena can eventually be reduced to chemical or even to physical phenomena does not mean reducing biology to chemistry and chemistry to physics, simply because of the enormous increase in complexity that one faces in trying to explain higher level phenomena in lower level terms. It is mainly in order to deal with the enormous complexity of living beings that we still need all the scientific disciplines from physics to biology. Hence, I would still defend reductionism (and also determinism) in properly construed form. I also think simplicity is perhaps the most important criterion for good science. Trying to see how far we can get by assuming the simplest laws to hold everywhere and always and allowing for exceptions and complications only when forced by the facts has brought us far and has proved to be a sound methodological principle. But its success may primarily be due to the simplicity of our minds rather than the world we are trying to understand. Thus a statement like "nature is simple" may be right as a methodological principle, but it may be false as a factual statement about "nature."

I used to believe that all of this was more or less equally clear to many if not most scientists, but after reading the examples given by Rose (in particular in the second half of the book) I have changed my opinion on this point. Moreover, I have myself experienced how hard it is to convey these basic ideas to a journalist who firmly beliefs in the ultimate truth of science and wants a spectacular story. Having read this book one realizes the danger of this process and the potential damage it can do to our self-image and thereby to our social relations. The public misconception of the underlying philosophy of science regarding the tentativeness of scientific hypotheses tends to lead to an overestimation of scientific results and an almost religious belief in science. This is particularly dreadful in scientific statements concerning human nature and social affairs. It is rather a methodological scientific assumption than a scientific result, for example, that we have no freedom of will. More concrete examples of such overinterpretations concern the localization of schizophrenia or homosexuality in one brain region or in one gene. The most imminent danger of

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false beliefs in such simple "scientific" explanations that reduce social, environmental, and biological complexities to the safe playground of single causes and physical laws is that they prevent us from facing complexity. And it is high time to face it, that is, to develop methods and intuitions to deal with complex situations.

Extrapolated lifelines

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Abstract: Extending lifelines still further, beyond determinism leads to doubt about females' ability to select mates with "good genes," and to a scenario for troop selection and to a link with questions of scale in ecology.

My first, and main, response to *Lifelines* is to raise a cheer; any counterattack against the overwhelmingly fashionable "a gene for everything" paradigm is to be welcomed. That it should come from Rose, an undeniably successful reductionist in his research methods, should give it more power and durability. We can but hope that the accessible style of the book will allow its message to be taken into the political arenas where decisions are now being made on the basis of the attractively simple-seeming molecular genetical explanations of life.

I should like to follow up and extend a couple of lines of thought started by reading *Lifelines.* The first led me to reconsider mate choice, recently an extremely fashionable topic in behaviour journals, and brought up by Rose in Chapter 7. [See also Buss: "Sex-Differences in Human Mate Preferences" *BBS* 12(1) 1989; Kenrick & Keefe: "Age Preferences in Mates Reflect Sex Differences in Human Reproductive Strategies" *BBS* 15(1) 1992; Rushton: "Genetic Similarity, Human Altruism, and Group Selection" *BBS* 12(3) 1989, Ed.] On what basis might a female mammal select a mate, given that, in most cases, he offers no parental help, but merely genes? Recent studies have assumed that females "select for good genes," but what does that mean? The males offering themselves for selection have survived to maturity, which means, in elephant seals and many monkeys, for example, that they are 10–12 years old. Clearly none of their genotypes is dysfunctional, simply because they have survived so long. But, is that splendid specimen evidence of a lucky couple of good seasons while he was a juvenile, or of some genetic advantage? And conversely, is that rather bedraggled male one who, because of his superior genotype, has been able to survive challenges which would have killed a lesser being? The phenotypes among which the female should choose are the result of such a lengthy interaction with the environment that reading the genetic contribution is surely impossible.

In any case, on offer is a more or less random selection of half the male's genes, which may or may not find a congenial environment in the genetic and cytoplasmic contribution of this particular female. Logically, it seems very doubtful that a valid selection "for good genes" can be made. A female, to be on the safe side, should avoid a sick-seeming male, because it might be infectious. She should on the whole prefer older males, because they have survived longer. And it would be as well to mate with several males, in case the first is infertile and so causes her to waste a whole breeding season. Apart from those considerations, and hurtful though it may be to the male ego, whom she mates with is a matter of practical indifference to her.

There is some evidence that females may in fact follow these simple rules-of-thumb. Most male secondary sexual characters become more pronounced with age: the antlers of deer get larger each mating season, the tail of a peacock becomes longer and the eyes more numerous (Petrie et al. 1991). Females thus have evidence of relative age, and are frequently observed to choose older males (while on a longer evolutionary time scale, of course there is pressure on the males for earlier development of these traits and hence for their increase in ultimate size). Careful observation of oestrous females under natural conditions, even those that seem at first sight to be controlled or sequestered by males, frequently reveals promiscuous mating (e.g., Cords et al. 1986).

The second line of thought concerns emergent properties of interacting systems, introduced by Rose in Chapter 4. While Rose only took this to the level of the emergent properties of the 4-dimensional individual organism, it can be carried further. Best known, perhaps, is Hinde's (1974) description of how the exchange of a series of communicative gestures between two individuals makes a social interaction; from a series of interactions over time emerges a relationship between the pair; and from the network of pairwise relationships between a group of animals emerges an organised social group. [See Bernstein: "Dominance" *BBS* 4(3) 1991, Ed.] Such a group, or herd, or troop interacts as a unit with its environment. Appropriate questions can be asked of the system at each of these levels, and they cannot be answered at lower or higher levels – they simply make no sense except at their appropriate level.

The level of cooperation and coordination among members of a troop will, to a large extent, determine its success at exploiting its environment and defending itself against predators. A predator perceives the troop as an entity which, depending on the degree of coordination within it, is either worth stalking, or better abandoned in favour of less well defended neighbours. Although it will be an individual that the predator eats, selection thus acts on the behavior of the troop, to which each individual contributes. The selection on the individual is for cooperative behaviour within its troop. Note that this troop selection, which acts on animals living together who need not be related, nor are a closed interbreeding unit, is not the same as group selection, which is a construct of population genetics concerned with closed, or almost closed demes. [See Wilson & Sober: "Reintroducing Group Selection to the Human Behavioral Sciences" *BBS* 17(4) 1994, Ed.]

This line of thought also leads on beyond intra-specific interactions to ecological questions: Allen and Starr (1982) consider hierarchy of scale in space and time, showing that questions are only relevant, and can only be asked, at the appropriate level; reductionism can in this case lead quite literally to failing to see the wood for the trees. Their book makes an excellent companion to the one under review.

Psychology and sociology: Beyond neither determinism nor science

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Abstract: While agreeing with Rose's reasoning about why the causes of organisms' behaviors cannot be reduced to the solely biological and molecular, this review questions Rose's uses of the terms "determinism" and "contingency"; his occasional seemingly cavalier acceptance as fact of unproven hypotheses about social and psychological phenomena; and his general disdain for the psychometric tradition and its causal modeling extensions.

To someone whose research crosses the macro-level, molar fields of sociology and psychology, it is gratifying to see a distinguished researcher on molecular mechanisms of cognition write a book that presents strong arguments against solely reductionist views of behavior. Rose's arguments stressing the importance of different levels of phenomena in causing behavior should give pause to the apparent stampede to explain all human function in extreme reductionist terms. I find Rose's critique of ultra-Darwinists convincing, although my relevant expertise is limited. Where I am more entitled to an opinion, I generally agree with his criticism of

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sociobiology and find Rose's much needed description of the limitations of heritability estimates generally on target. His admonition constantly to bear in mind the full implications of gene/environment interaction are well taken.

One is left with two kinds of qualms:

(1) There can be some concern that all of Rose's views should not be summarily dismissed because some of them are taken as soft-headed or overstated;

(2) Rose's occasional display of scientific arrogance toward psychology and sociology in what may be a byproduct of doing reductionist science (even if for methodological rather than ideological reasons).

The first set of qualms center on Rose's use of the term "determinism." Given Rose's theoretical reasoning and empirical examples, there seems to be no problem if what is meant by the book's subtitle "*Biology beyond determinism*" is that an organism's development is not solely determined by its genes and its behavior by its biological characteristics. On the other hand, if one truly believes, as Rose appears to, that other levels of phenomena also affect an organism's behavior, arguing that not all is biology does not make that organism's behavior any less fully determined. Nor do the effects of contingency make it any less determined. For Rose, "contingency" has several meanings. One is that causal patterning may be extremely complex and diffuse. Another concerns our difficulty in measuring a particular event or process whose effects are relevant. A third is the spatio-temporal conjunction of previously separated processes that then jointly affect the organism. None of these forms of contingency makes the way the organism changes or responds any less determined.

Differences in connotation between the terms "change" and "respond" suggest another questionable way Rose sees organisms as somehow being beyond determinism. "Change" implies that the object or organism being acted upon is relatively passive, that is, the movement of a billiard ball after being hit is calculable as a direct function of the forces acting upon it. The way of striking the cue ball, however, is a function of the player's response to the demands of the situation, a response that involves not only physical abilities and state, but also motivation, knowledge of the game, and ability to plan for the future. Still, these characteristics are a direct function of the player's past history as embodied at the instant of striking the ball. Since it is not completely determined by the immediate external environment, this active, and indeed proactive, response might be seen as beyond determinism. Nevertheless, it is completely determined by the player's history.

This description of the determinacy of the billiard player's response probably accords with Rose's own views. He sees "biology as history" (p. 18). When he describes humans as free agents it is in the "Marxist sense of the freedom of necessity" (p. 18). Unfortunately, to a non-Marxist "freedom of necessity" may seem an oxymoron, likely to provide only cold comfort to someone who bought Rose's book believing that the subtitle promised a release from determinism to free will.

Grounds for my qualms over reductionist arrogance come from the way Rose lists, without citing a single relevant empirical study, a whole host of psychological and sociological explanations of attention deficit hyperactivity disorder (e.g., relations with parents, size of class in arguing that the theory that the disorder's cause is "'inside' the child's brain . . . is almost certainly wholly fallacious" (p. 56). Carrying out the relevant studies may be more difficult than counting the pecks of a chick in a meticulously controlled environment, but doing so is necessary if our knowledge of human behavior is to go beyond ex cathedra assertions of what appears obvious to the asserter. In a similar vein, one is bemused by Rose's belief (discussion pp. 191–92 and n. 17, p. 206) that he would learn much by inspecting the primary data of Bouchard's (Bouchard et al. 1990) twin research. Only someone trained in the psychometric and related statistical techniques that Rose apparently disdains can appropriately appraise the research in question.

More generally, one is bothered by Rose's consistent linkage of racism, genetic studies, and the psychometric methodology. Granted, he has much history on his side. Nevertheless, doing twin studies is no proof of racism. Neither is respect for the accomplishments of the psychometric tradition and its extensions into causal modelling of non-experimental data. The one of Rose's objections to heritability studies that is off-the-mark is his contention that the relevant psychological and environmental variables are not measurable. Mine is admittedly the perspective of someone who, with the aim of assessing the psychological effects of environmental conditions, has spent much of his career developing rigorous psychometric measure of such psychological characteristics as job satisfaction, self-esteem, and intellectual functioning. These measure are valid indicators of the individual's psychological functioning in a particular time and situation. By themselves they say nothing about what the individual would be like in other times and situations. They do, however, provide a means of modeling development, stability, and change in the individual's psychological functioning.

I have been equally involved in developing statistically rigorous measures of environmental conditions, such as the substantive complexity of work. On mathematical grounds, it is even more important to get accurate measures of independent environmental variables than dependent psychological ones, especially if we want to compare the magnitudes of the psychological effects environmental variables with the magnitudes of biological ones (presumably measured accurately). If we are to take seriously Rose's concern for how individuals are affected by different aspects of their environments, the measurement of environmental variables and their effects should not be left to armchair speculation.

Why twin studies really don't tell us much about human heritability

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Abstract: The derivation of heritability from human twin studies involves serious methodological flaws. Heritability is consistently overestimated because of biological confounds of twinning, consistent and often gross underestimation of the environmental variance, and nonadditive genetic influences that can hugely exaggerate heritability values. Despite this bad research design, behaviour geneticists continue to publish results implying that their heritability results are valid.

Genetic factors clearly influence human development. What is contested is the nature of this influence (the focus of *Lifelines*) and its extent. In theory, one can obtain a measure of the degree to which variations in a trait across individuals are attributable to genetic variations in the population. Twin studies provide the strongest support for genetic sources of this variation in traits. The commonly used metric is heritability, which is derived from the increase in correlation in the trait of interest in monozygotic twins (MZ) compared with dizygotic twins (DZ). Rose dismisses this methodology because the assumptions of the heritability model are not met in human studies (pp. 188–93). There are several good reasons for this dismissal, although he singles out primarily statistical assumptions. Unfortunately, behavioural scientists are in the habit of ignoring problems of statistical assumptions, and assume that replication and large sample sizes will validate problematic results in the end. The research design problems of twin studies are much more severe than this, however, being ones of systematic bias, inflating heritability values. These overestimates of heritability are due to at least three research design faults:

1. Biological confounds. In an MZ–DZ twin study, the only difference between the twin types is supposedly the extent of genotypic similarity. However, two-thirds of MZ twins also share the same placenta and chorion whereas no DZ twins do (Phelps et al. 1997). MZ twins who share a chorion are significantly more

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similar on a variety of personality and cognitive measures than those that do not (Sokol et al. 1995). This should not be surprising, for by sharing a chorion, MZ twins also share blood supply, a variety of early hormonal influences, and even infections and immune status (which may account for higher concordance rates of schizophrenia in MZ compared to DZ twins; Phelps et al. 1997). Thus, MZ twins share other long-lasting developmental factors besides DNA similarity. This reinforces the notion of early biological factors influencing psychological development, but the genetic attribution is exaggerated.

2. Consistent underestimation of environmental variance. In twin studies, environmental variation *between the members of the twin pairs* is always an underestimate of society's range. If every individual were to receive exactly the same environment, then all variation in a trait would have to be attributed to genotypic differences (and genotype × environment interactions). To the extent that environmental separation of adoptive twins is less than that between twin pairs, the heritability is higher. But adoption agencies never give children to families in abject poverty (because their mandate is the best interests of the child), much less separate twins across such an advantaged/impoverished spectrum. They have even matched adoptive home religion for twin pairs. Thus, the cultural distance between twin members must be considerably less than that available in the society as a whole. Add to this the fact that the large twin studies (those in Minnesota and in Sweden) follow up adopted twins in relatively homogeneous societies (where well-developed health agencies and medical records are available). The thought experiment of separating twins at birth to widely different settings – for example, one to urban New York, the other to rural Sahara; one to an affluent home in London, the other to a poor family in the third world – illustrates how heritability is artificially raised by restrictions of environmental variance. Indeed, Bronfenbrenner (1975) showed that the correlation in IQ between twins, which is normally .85, drops to .26 with twins whose adoptive families live in different communities (e.g., mining versus agricultural). Thus, potential environmental variance is vastly underestimated and therefore heritability is necessarily overestimated.

3. Nonadditive genetic influences. Heritability is mathematically based on the notion of *additive genetic components* – the more similar genes two individuals have the more similar their trait outcome will be. What happens, however, if genetically influenced traits are nonadditive, such as when a *complete pattern* of genes influences the trait rather than the genes individually? We are used to nonadditivity in traits, such as the attractiveness of a face. Having a nice nose and chin with the wrong mouth and eyes does not bring the face halfway to beauty. In twin studies, when MZ twins are so much more similar to each other than are DZ twins that heritability comes out to over 100%, which is nonsensical, we conclude that the genetic contribution is nonadditive (Lykken et al. 1992). [See also Wahlsten: "Insensitivity of the Analysis of Variance to Heredity–Environment Interaction" *BBS* 13(1) 1990.] In the extreme case, DZ can have a zero correlation and MZ a 100% correlation, making heritability 200%. In such a case, the heritability model must be rejected. Unfortunately, every trait measured by a behaviour geneticist can be conceived as being a high-level product of many subtraits, some of which may have nonadditive genetic aspects, others not. Nonadditive factors will add enormously exaggerated heritability components (up to the maximum of 200%). Since we have no independent measures of the heritability for the subcomponents, there is no way to quantify the overestimation of heritability.

For these three reasons (and others), heritability derived from twin studies is always inflated, and replicating the twin studies will only replicate the error. It is not surprising that composite traits such as IQ have a high heritability–gene similarity in twin studies is confounded with prenatal health and environment, reduced environmental variation in twin-adoptive families, and some of the subtraits related to IQ probably have nonadditive components. Thus, we cannot find a value for the genetic heritability of traits from twin studies, although we clearly accept that genes influence (in some complex way) nervous system growth. The most that twin studies can tell us is the upper limit of true heritability, not an absolute value that is to be believed.

Hierarchical approach to replication and selection

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Abstract: The major merit of Rose's book is the elaboration of the idea of multilevel causation in different explanatory languages. Yet Rose's critique of "ultra-Darwinism" is not convincing. Rose argues that activity and self-replication are properties of organisms rather than genes, which contradicts his idea of multilevel causation. Also, Rose fails to develop the concept of multilevel selection.

Contemporary biology is essentially reductionistic in its attempt to collapse the entire hierarchy of living systems to the molecular level. Rose defends the idea of hierarchy. In particular, he criticizes the view of Dawkins (1976; 1978), labeled "ultra-Darwinism," that evolution occurs at the level of genes rather than organisms.

Rose considers systems at each level as semi-independent agents. The interaction between hierarchical levels occurs through the process of development. Hence the title of the book, *Lifelines*, which means developmental trajectories of organisms. The novelty of Rose's approach is that each level of the hierarchy is viewed as a distinct *language* for describing things at a specific scale. One language can be partially translated into another, but there is always an untranslatable residual. This thesis is formulated as "one world, many ways of knowing" (p. 304). Notions used in one area of biology change their meaning or become useless when applied to another area. For example, the notion of gene changes its meaning as we change scale from organism to chromosomes and then to DNA molecules.

Living systems do not exist in isolation from their environment. But isolation is necessary to perform experiments. This contradiction is resolved by using a *hierarchy of isolations.* We can isolate an RNA molecule and determine the sequence of its nucleotides. But in isolation from the cell we will never observe the function of this molecule. Thus, boundaries of living systems are fuzzy.

According to Rose, ultra-Darwinism is based on the idea that "the purpose (*telos*) of life is reproduction, reproduction of the genes embedded in the 'lumbering robots' which constitute living organisms" (p. 209). But Dawkins (1986) denies any purpose of life as well as any other purposes in Nature; this is the major idea of his "blind watchmaker" metaphor. The next statement of ultra-Darwinism, according to Rose, is that "Every observable aspect of the phenotype of an organism . . . is in some way adaptive" (p. 210). As evidence of the role of chance in evolution he mentions neutral evolution, strange fossils found at the Burgess Shale, and the role of developmental constraints (photoreceptors in the eye have a backward orientation). Unfortunately, Figure 8.2 shows *Hallucinogenia* (an animal from the Burgess Shale) upside down. The second row of its legs was initially missing, and thus, first reconstructions were misleading (Ramskold 1992). After flipping the image, it becomes similar to Onychophora. But Dawkins never denied the role of chance in evolution. He wrote about neutral evolution and photoreceptors in the eye (Dawkins 1986). The only difference is that he considers neutral evolution a "boring part" (p. 303) of evolutionary theory.

The line of demarcation between the views of Dawkins and Rose lies in their answer to the question: Which are active, genes or organisms? Dawkins (1986) thinks that the real agents are genes who collectively build an organism, which is a mechanism for gene

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duplication. In contrast, Rose thinks that organisms are active, whereas genes are passive tools used by organisms to transfer information. He writes that DNA is not a self-replicating molecule as claimed by Dawkins because it cannot build its copy in isolation from the cell. Not even viruses are considered self-replicating or active because they require a host cell to replicate.

This discussion appears pointless if we think in terms of multilevel causation. If hierarchical levels are partially independent, then activity can be found at various levels, including genes *and* organisms. Self-replication is impossible in any isolated system, not only in the isolated gene. Somebody might argue then that any material thing can be considered a self-replicator simply because it can be copied by humans. This logic is based on extreme cases (complete isolation or no isolation) and ignores the hierarchy of isolations.

One of Rose's key statements is that "the individual gene is not the only level at which selection occurs" (p. 215). It is not clear how this is compatible with his claim that genes do not self-replicate. Although I entirely accept the idea of multi-level selection, the arguments presented by Rose are not convincing. He considers the fact that "any individual gene can be expressed only against the background of the whole of the rest of the genome" (p. 216) as evidence of selection at the level of organisms. But Dawkins (1986) did not deny interaction between genes. He wrote: "the whole process of embryonic development can be looked upon as a cooperative venture, jointly run by thousands of genes together" (p. 170). He then adds: "But from each gene's point of view, perhaps the most important part of its environment is *all the other genes that it encounters* . . . Each gene is selected for its capacity to cooperate successfully with the population of other genes that it is likely to meet in bodies" (p. 170). All effects of genes at various levels (proteins, cells, organisms, societies, and ecosystems) are easily explained by Dawkins (1986) as cooperation among genes. Thus, Rose fails to formulate the concept of multiple levels of selection. His major mistake is that he treats selection at the level of genes as something ontologically different from selection at the level of organisms. Instead, these are *different languages* for describing basically the same phenomenon. Thus, the selection of cooperating genes is a legitimate way of describing natural selection, but it is not the only possible way. Although these languages are mostly compatible, there may be a preferred language for handling each particular case. Complete genetic descriptions exist only for a few species of organisms such as the fly *Drosophila.* To analyze the evolution of other species, we have no choice but to remain at the organism level.

Cognitive and psychiatric science beyond determinism

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Abstract: Many of Rose's criticisms of determinism in biology have clear relevance to modern cognitive and psychiatric science; too narrow a focus on the brain as an information processing machine runs the risk of neglecting the context in which information processing takes place, and too narrow a focus on the neuroscience of psychopathology runs the risk of neglecting other levels of explanation for these phenomena. It should be emphasized, however, that animal and genetic studies of phenomena of interest to cognitive and psychiatric science (e.g., Alzheimer's disorder, schizophrenia, attention deficit/hyperactivity disorder, and violence), while perhaps only providing a partial perspective, may be useful in understanding these phenomena and in leading to appropriate psychiatric interventions.

In *Lifelines,* Steven Rose asserts that in contrast to physics and chemistry, biology "is different" (p. 68). "Not only is the living world much more complex and less predictable than the inanimate world studied by physicists and chemists, but biology . . . lays claims to be in a position to tell us, as humans, who we are, where we came from, where we are going, how we must live" (p. 68). Thus, a central theme of the volume is the folly of reductionism in biological science, and the importance of multiple levels of explanations of biological phenomena.

Among the most "different" (most complex and least predictable) fields within biology are the study of human psychology and, perhaps even more, of psychopathology. Indeed, as a psychiatrist, I could not help but relate many of Rose's concerns to developments in this field. (Rose too uses a number of examples from the psychiatric literature.) Modern psychiatry, particularly in the United States, has seen the replacement of a predominantly psychodynamic perspective with a "biological" approach, which emphasizes the importance of neuroscience in understanding psychopathology, with pharmacotherapy as an appropriate intervention. Among the risks of this approach is failing to see that psychiatry is itself a social practice.

Many of Rose's criticisms have bearing upon the aspirations of biological psychiatry; too narrow a focus on the neuroscience of psychopathology runs the risk of neglecting other levels of explanation for these phenomena. Several authors, such as McHugh and Slavney (1983), have emphasized the importance of different kinds of explanation in psychiatry; an important strand of explanation must, as Rose advises for biology in general, focus on history and development. Clearly, any philosophy of science and biology must address not only mechanism but also meaning (Stein 1991). Thus, a sudden episode of decompensation in a patient with schizophrenia may require both a biological perspective (the patient discontinued medication) and a more psychological one (the decision to stop medication took place in the context of family conflict over the expense of medical care).

Rose's criticisms also have bearing on the aspirations of much of cognitive science; too narrow a focus on the brain as an information processing machine runs the risk of neglecting the context in which information processing takes place. Several authors have emphasized the importance of situated cognition as opposed to simply symbolic cognition (Norman 1993); once again, the emphasis of situated cognitivists is, as Rose advises, on matters such as agency, meaning, and history. As Rose succinctly puts it (p. 9), "Not everything is capable of being captured in a mathematical formula." Indeed, more sophisticated approaches in cognitive science (e.g., Lakoff 1987) go well beyond the narrow focus on Platonic essences (p. 42) and digital algorithms (p. 121) that Rose criticizes. Certainly, when it comes to explaining psychopathological phenomena, a purely algorithmic approach (for example, PARRY, the digital model of paranoia; Colby 1981) would do only partial justice to the complexity of the matter at hand.

Rose goes one step further, though, arguing that biological reductionism is ascendant, and that it entails an ideology that needs to be fought (p. 273). While genetic findings in psychology and psychiatry have undoubtedly received a great deal of attention, and while the pharmaceutical industry is clearly a powerful one, I am unconvinced that neuroscientific approaches to these fields are always very widespread (certainly laypersons continue to view mental disorders as "different") or necessarily very evil. Let us consider some specific examples from psychiatry which Rose himself raises: Alzheimer's disorder, schizophrenia, attention deficit/hyperactivity disorder, and violence.

Rose notes that he himself is a neuroscientist interested in memory. He puts forward an argument about the necessity of (simplified) animal models of memory, and the possibility that they may have implications for understanding human memory and for intervening in disorders like Alzheimer's (p. 32). He notes also that in Alzheimer's only 5% of cases are clearly associated with a specific genetic dysfunction (p. 293). Similarly, any specific mechanism that emerges from a study of animal models may well require extension and elaboration before it can be applied to human memory dysfunction. It hardly seems controversial to assert that

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a good explanation of Alzheimer's is going to require an understanding of the interactions between multiple factors (neuroscientific, psychosocial) involved in the pathogenesis of this disorder; it makes sense for a funding agency interested in Alzheimer's to fund studies on the genetics of Alzheimer's and on animal models (such as Rose's, as well as a range of other approaches). The recent introduction of specific medications for Alzheimer's has by no means provided a cure, but the very fact of their existence provides promise for a neuroscientific approach to the disorder.

How different is the study of schizophrenia? Rose asserts (p. 306) that schizophrenia is a contested area where crucial determinants may occur at several levels (e.g., neuroscientific, psychosocial). Certainly, there is good evidence that psychosocial variables influence the course and expression of schizophrenia. Nevertheless, the field of schizophrenia has moved far beyond the outdated notion of the schizophrenogenic mother. As in the study of memory and its dsyfunctions (e.g., Alzheimer's), genetic findings and animal models may be extremely helpful in understanding this disorder; an important remaining challenge is to integrate these findings with those from research on psychosocial factors (Portin & Alanen 1997; Weinberger 1996). Furthermore, a focus on the neuroscience of schizophrenia does not necessarily fall prey to Rose's sociopolitical criticisms of reductionism (p. 297) – "blaming the victim" and misallocation of resources. It is notable that families of schizophrenia patients have increasingly supported neuroscientific research on the disorder. Furthermore, the introduction of antipsychotics (and more recently of the atypical antipsychotics) have represented dramatic steps forward in the treatment of this major disorder.

What about the study of attention deficit/hyperactivity disorder (ADHD)? Rose (p. 56) asserts that ADHD is not appreciably known outside the United States and that it is a disorder that may disappear on weekends and holidays. Furthermore, he seems to indicate that it is erroneous to think of the disorder as brain-based; it is better explained by the relationships of the child to the outside world. Here his argument, which sounds similar to literature produced by the anti-psychiatry movement, runs the risk of a reductionism of a different sort (reduction to the psychosocial). While psychiatric diagnosis is of course a particular kind of social practice (Stein 1991), ADHD is in fact widely recognized and treated outside of the United States (World Health Organization 1992). Furthermore, although ADHD is clearly complex, genetic and biological studies have in fact shed important light on its pathogenesis (Castellanos 1997). The research demonstrates clearly that for a child with ADHD, medication and psychotherapy interventions are extremely helpful (Shaywitz et al. 1997). While the theoretical possibility of replacing such individual interventions with universal changes in school systems no doubt exists, such a solution runs the risk of "fatuity" (cf. Rose p. 299). (This is not of course, to deny that in some cases, where a primary underlying problem is in fact simply poor parenting or poor schooling, children may unfortunately be misdiagnosed as having ADHD.)

What about the study of violence? Rose appears to be scathing about the possibility of a neuroscientific approach to human violence; he picks out Brunner's study (p. 281) for criticism for arbitrarily lumping together different kinds of aggressive behavior, is withering about Raine's studies of PET scanning in murderers (p. 290), criticises the extrapolation of animal studies of aggression to humans (p. 294), and argues that biological interventions for violence would be fatuous (p. 298). Brunner (1996) himself, however, has clearly acknowledged the complexity of aggression and violence. Similarly, Raine (1993; whose work is not listed in *Lifelines*' references), has emphasized that multiple factors contribute to criminal behavior, and rather than seeing Prozac as a panacea (cf. Rose p. 291) has argued for the importance of psychosocial interventions. Rose is no doubt right in stressing the complexity of violent behavior and in noting that interventions need to be primarily psychosocial; and it is certainly important not to blame the victim but to allocate funding appropriately (p. 297); nevertheless, there is no prima facie reason to avoid animal or genetic studies of aggression as useless or harmful. In addition, pharmacological interventions are useful for some impulsive and aggressive psychiatric patients (Coccaro & Kavoussi 1997). Finally, although Rose provides a trenchant critique of the excessive claims of some evolutionary psychology (p. 246), evolutionarily informed approaches to aggression can be sophisticated and relevant to human behavior (de Waal 1996).

If biology is able to succeed in being a good explanatory science, then a "biological psychiatry" which includes multiple levels of explanation is also possible, and will extend this field beyond the all too common connotation of "biological psychiatry" as necessarily entailing a reductionistic perspective. Rose notes (p. 302) that much of his thesis is not new; nevertheless, his synthesis is an attractive and important one. Certainly, when it comes to complex psychopathology, there is much evidence à la Rose, that understanding both genetic and historical factors is crucial (Schaffner 1998); that soma and psyche are interlinked (with successful psychotherapy resulting in functional brain changes; Baxter et al. 1992); that biological interventions having multiple psychological effects (Kramer 1994); and that explanations of psychopathology are themselves rooted in social practices (Kleinman 1988). Any oversimplification in this area (for example, characterizing psychiatric disorders solely in terms of neurotransmitter dysfunction, or depicting psychiatric disorders as representing simply a "normal" response to an "abnormal" environment; Conrad & Schneider 1980) is bound to be problematic.

Lifelines to a sinking cause

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Abstract: Rose's attack on ultra-Darwinists' and evolutionary psychologists' accounts of human behavior fails, largely because he does not offer a viable alternative. His "lifelines" view of organismic development is essentially an epigenetic one that few sociobiologists would disagree with, Rose seeks to disempower genes where human behavior is concerned, despite contrary neurogenetic evidence and at odds with the implications of his own lifelines view. He attempts to discredit biological determinist accounts by suggesting that their research program is politically motivated, while ignoring the scientific viability and novel insights such research has yielded.

Much of what Rose says I agree with, so why am I not in his camp, a defender of his cause, which is to reject the current "enthusiasm for biologically determinist accounts of the human condition"? It is because Rose does not offer a viable alternative. I want to illustrate and support this point further by examining Rose's arguments concerning (1) what genes do; (2) appropriate levels of causal analysis; and (3) science as a mirror of reality.

What genes do. A central tenet of Rose's anti-determinist argument rests on the indeterminate nature of an organism's genetic instructions, be they coding for the eventual form of proteins, neural circuitry, or behavior. In Rose's view, the reason for the genome's indeterminacy lies in the dynamic, and essentially unpredictable interaction between the developing organism and its environment, from the cellular environment of the DNA strands themselves to the extracellular environment of developing tissues, to the external, postnatal environment of the developing organism. This dynamic developmental and maturational trajectory of an organism Rose calls its "lifeline," a process throughout which "organisms . . . and the environment – all relevant aspects of it – interpenetrate" (p. 140); in other words, an inextricable joining of nature and nurture. Thus, although according to Rose, lifelines as such are "not embedded in genes" (p. 171), an organism's imperviousness to "developmental and environmental buffeting" is "in the genes" (p. 306), ensuring normal developmental outcomes in

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the face of chance environmental perturbations. To Rose, genes "are no more and no less than an essential part of the toolkit with and by which organisms construct their *own* futures" (p. 137, emphasis mine). It is, then, in viewing the organism as free to self-construct, as an active agent in building its lifeline, that Rose parts company with those who see the organism as a passive entity (as in Dawkins's genetic vehicles) whose structure, form and function are predetermined by its DNA.

Because Rose's "lifelines" view of organismic development is essentially an epigenetic one – the recognition of the fundamental interdependence between organism and environment – I doubt that any sociobiologist would disagree with him. Rather, disagreement revolves around whether epigenesis excludes a genetic influence on behavior or how epigenesis can be said to provide "freedom" from genetic influence. Rose's final sentence leaves his interpretation of epigenesis unambiguous: "And it is therefore our biology that makes us free" (p. 309). Aside from its rhetorical intent, how are we to interpret this declaration of independence? Does he mean "free" in the sense of "unconstrained"? Surely not. He does, after all, recognize the process of genetic canalization, and, as mentioned earlier, the genome's contribution to developmental buffering. The argument then boils down to the meaning of constraint.

There is nothing in Rose's "lifelines" argument that precludes the standard sociobiologist's requirement for a genetic influence (or constraint) on behavior. We need only pursue two aspects of his "lifelines" view to make this clear. One concerns initial conditions (or constraints) provided by the genome, the other concerns the "interpenetration" between organism and environment. Regardless of the "distance" between genes and behavioral expression, genes (minimally) do specify the initial conditions for the organism's lifeline, thereby constraining all subsequent organism-environment interactions. I assume that Rose would agree that, for the reliable appearance of species-typical characteristics (e.g., bipedality, human language, play), the initial conditions provided by the genome (e.g., cellular components and products) must somehow constrain all subsequent organism–environment interactions. Indeed, it is precisely the departure from such canalized development, as in the fluctuating asymmetry of normally symmetrical morphology, that interests evolutionary biologists, because it suggests (and in some cases has been linked to) genetic variation – and hence fitness – in the organism's ability to withstand the effects of Rose's "developmental buffeting" (Polak & Trivers 1994).

How the cascade of developmental events constrains developmental outcomes is inherent in Rose's own notion of "interpenetration" between organism and environment. I'm sure that as a biologist he would not disagree that from conception to death, each organism defines its environment by seeking out or identifying and responding selectively to stimuli within it. It follows that each organism's experience is *not* random (contrary to Rose), and that its genome (by initially constraining and setting in motion the developmental trajectory) defines which stimuli are salient, and in this sense constrains the organism's interaction with its environment.

The field of neurogenetics, of which Rose is critical, supports this view of developmental constraints. Myriad experimental studies show that the mammalian brain has evolved to both expect and depend on information from the environment not only for its preadult ontogeny, but also for its adaptive modification in adulthood (Greenough 1986). The nervous system appears to "anticipate" experiential events that have been reliable environmental features of the species' evolutionary history – its past "lifelines" (e.g., the primate mother–infant bond is an evolutionarily reliable environment that provides "information" for the normal development of brain structure, function, and behavior; Hofer 1981). A species' evolutionary history thus defines which experiences are salient, at what time, and in what manner (e.g., see Gottlieb's 1976 useful distinction between inductive, facilitative, and maintenance effects of experience on behavioral and neural maturation). Because the timing and importance of experience for phenotypic expression vary among different components of the phenotype, it is meaningless to think of entire organisms, rather than particular phenotypic traits, as either "plastic" or "predetermined" (e.g., dependent on or independent of learning). In humans, for example, the capacity for spontaneous facial expression of emotion emerges independent of visual experience, suggesting that its normal emergence is highly constrained. This, however, does not rule out the influence of other forms of experience (e.g., sensory feedback from facial muscles) on the development of this ability.

If Rose pursued the full implications of his concept of organism–environment "interpenetration" he would not argue, for example, that because the family environment of siblings is similar, "similarities detected between them are inextricably the result of both genes and environment" (p. 190); in other words, that any genetic contribution to the behavioral phenotype will be masked by the shared environment. A wealth of studies show that fraternal twins, despite being raised in the same family environment, often turn out to be very different in personality and behavior (Plomin & Daniels 1987). Indeed, from a comprehensive review of the research literature, Harris (1998, p. 37) concludes that "growing up in the same home does not make siblings alike." The likely reason for the failure of the shared environment to produce shared traits is that the genetically constrained lifelines of siblings lead them to construct their "shared" household environments differently. From the perspective of the self-constructing organism, the environment is never truly random or homogeneous.

Appropriate levels of causal analysis. Rose unjustly condemns "ultra-Darwinists" for choosing the wrong level of causal behavioral analysis. In discussing human violence, for example, he not only disagrees with neurogeneticists who aver that violence is caused by "'violent' or 'criminal' genes," but also insists that "neurogenetics is the wrong level . . . at which to find answers to many of the problems confronting us" (p. 276–77).

Like Rose's early example of causal explanations why a frog jumps, each level of explanation is equally valid and appropriate, providing insight into causation at that level (biochemical, anatomical, evolutionary). A complete causal analysis combines all levels, which is basically what the classical ethologists have advocated all along in recognizing the complementarity of proximate and ultimate causes of behavior. In the case of human violence, the sociological level of causal analysis is no more appropriate than the genetic or biochemical one; all are equally appropriate for a full understanding of why (to use Rose's example) in the same situation some individuals murder while others do not. The sociological level of causation would include social stimuli or triggers (e.g., poverty or a history of marital infidelity), while the physiological level could identify the responsible brain mechanisms (e.g., low serotonin). One cannot agree more when Rose writes that "the phenomena of human existence and experience are always simultaneously biological and social" (p. 279). Does it not follow, then, that solutions to reduce "violence in the streets" are to be sought in both the social and biological domains?

Where the complexity of human behavior is concerned, Rose considers the biological domain fundamentally irrelevant, because he does not believe that genetics can usefully explain, for example, why some individuals murder while others do not. Although there may not be "aggression genes" per se, it is hard to deny that genetic variation contributes to individual differences in temperament, sensation seeking (Zuckerman 1984) and other personality features that predispose some individuals to act violently, or to show impaired judgment of what is socially appropriate. Moreover, evolutionary psychology has yielded powerful insights into the social stimuli that reliably trigger human violence (e.g., Daly & Wilson 1988). Thus, while culture defines when violence is appropriate, it does not follow, as Rose would have it, that either the capacity or the prepotent nature of stimuli for violence are cultural.

Science as a mirror of reality. Rose suggests that research in the bio-behavioral sciences is at the mercy of political whim and

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"dominant social expectations" rather than being a "true reflecting mirror" of nature" (p. 274). This allows him to dismiss as politically motivated the research program of the ultra-Darwinists and evolutionary psychologists. To be sure, science as a cultural activity is not immune to influence from other cultural domains. Would Rose not agree, however, that in order to succeed, science must provide the best possible mirror of reality? Scientific predictions about how nature works either come out or they do not.

Why not let science do its work where evolutionary psychology is concerned? If the ultra-Darwinist approach to humans turns out to be a poor mirror of reality, it will have to be abandoned in favor of a more productive (predictive) program. The last quarter century of sociobiological research has not given any indication that its premises are flawed or its predictive powers diminished. Rather, the reason for the program's continued success is its power to identify novel research directions and to yield deep insights into the human condition, whereas the traditional consideration of purely socio-cultural factors in human behavior has stagnated. Rather than ignoring "the historical and anthropological evidence of variation in social practices across time and space, and instead treat current Western norms . . . as if they were human universals" (p. 198), many evolutionary psychologists' accounts have fully encompassed "the rich diversity of human experience" (e.g., see Buss 1994). Rose's caricatures of the application of reciprocal altruism and parental investment theory to human behavior fail to do justice to the corpus of genuine insights these theories have provided. Regardless of our opinion of their worth, only data and the continued predictive power of theory, rather than rhetoric or political motivation, will decide whether sociobiology or evolutionary psychology survive or fail.

Autopoiesis and *Lifelines:* The importance of origins

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Abstract: *Lifelines* provides a useful corrective to "ultra-Darwinism" but it is marred by its failure to cite its scientific predecessors. Rose's argument could have been strengthened by taking greater account of the theory of autopoiesis in biology and of enactive cognitive science.

We agree with all ten major theses of *Lifelines* (pp. 303–309) and wish to congratulate Steven Rose for his useful corrective to the excesses of "ultra-Darwinism." Especially important is his ninth thesis "The past is the key to the present" (p. 309), or, as he states it at the beginning of the book: "Nothing in biology makes sense except in the light of *history* . . . the history of life on Earth . . . the history of the individual organism . . . the history of our own subject, biology" (pp. 15–16). Unfortunately, in the case of the history of biology, Rose neglects to acknowledge the scientific origins of one of his central concepts, that of *autopoiesis,* and fails to cite and credit its originators. Furthermore, he could have strengthened his argument had he drawn from subsequent developments in biology and cognitive science inspired by the autopoietic perspective.

The idea of autopoiesis – that living systems produce themselves continuously through their own activity – forms part of the backbone of *Lifelines.* This is as it should be, for the idea of autopoiesis places the organism at the heart of biology. As Rose writes when he first introduces the idea:

> To put the organism and its lifeline back at the core of biology . . . means replacing the static, reductive, DNA-centred view of living systems . . . with an emphasis on the dynamics of life . . . Instead we must speak of the dialectic . . . through which the living organism constructs itself. The central property of all life is the capacity and necessity to build, maintain and preserve itself, a process known as *autopoiesis* [Rose's emphasis]. (p. 18)

What he neglects to tell the reader is that the very coinage of the word, its meaning, and the many consequences it entails, did not arise *ex nihilo,* without a history, to populate the world of ideas anonymously ("a process known as"). It has an origin that is clearly inscribed in the scientific record of publications: the notion and many of its consequences for biology (see below) were introduced by Humberto R. Maturana and Francisco J. Varela, first in 1973 in a Spanish monograph (Maturana & Varela 1973), which appeared in English in 1980 (Maturana & Varela 1980), and then in a paper in English, complete with cellular automata modes, in 1974 (Varela, Maturana & Uribe 1974). A book treatment concerning autopoiesis as an exemplar of the autonomy of living systems appeared in 1979 (Varela 1979). Why does Rose not cite the actual origins of this idea and present its precise formulation, as he does for metabolic complex graphs (p. 164) or autocatlytic sets (p. 265)?

Since 1974 a large body of literature on autopoiesis has been produced in experimental and theoretical biology. Let us mention just two examples, both concerned with the origin of life, and therefore of direct relevance to Rose's discussion of "Origin myths" (pp. 250–71). First, Lynn Margulis has explicitly acknowledged (with proper citation) the importance of autopoiesis in her work on symbiosis and the origins of sex (Margulis & Sagan 1986), and in her popular book with Dorian Sagan *What is life?* (Margulis & Sagan 1995). Second, some of the most interesting experimental advances in research on the origins of life have been inspired by the theory of autopoiesis. Thus, Bachman et al. (1992) constructed a minimal autopoietic system using autocatalytic self-replicating micelles. The self-replication of these micelles occurs as a direct consequence of their autopoiesis or self-production, and the latter occurs without DNA and proteins. Thus these chemical systems tangibly reinforce Rose's argument in Chapter 9 that life probably originated with autopoietic protocells rather than naked molecular replicators. As Bachman et al. observed:

> Within this framework, there is no mention of DNA or proteins, but the present autopoietic system can be considered as a cell that metabolizes low-molecular-weight components. A true 'protocell' will require DNA, RNA and proteins to encode and transfer genetic information; but it provides a useful new perspective to recognize that a primitive mechanism for self-replication and metabolism can exist without them. (Bachman et al. 1992, p. 59)

If the notion of autopoiesis had originally been introduced only to address the unitary organization of life at the cellular level, then our point about Rose's failure to acknowledge his predecessors would stop there. As the original publications make clear, however, the main motivation of the theory of autopoiesis was, from the beginning, to provide a new approach to the biology of cognition in which the organism and its space-time pathway occupy center-stage (Maturana & Varela 1980; see also Varela 1996). Indeed, Maturana and Varela (1987) unfold these implications for our understanding of evolution and cognition, in terms that Rose's book closely follows. Other more detailed implications have been presented for our understanding of the immune system (Varela & Coutinho 1991), evolution (Varela et al. 1991, Ch. 9), biological individuality (Varela 1991), and artificial intelligence and artificial life (Thompson 1997; Varela 1997; Varela & Bourgine 1992).

An especially important point concerns the consequences of autopoieis for reinterpreting the operation of the nervous system in cognition. The central revision was to discard the idea that the nervous system encodes the external environment in an internal representation, so as to ensure the optimal adaptation of the organism. The nervous system was instead seen as a unitary recursive network, situated within an autonomous (self-governing or "homeodynamic") organism, itself engaged in a constant flow of action embedded in its surroundings. Cognition via the nervous

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system is not the "picking up" of prexisting information "out there," but rather the bringing forth of meaning in ontogenetic and phylogenetic "lifelines" – the laying down of a path in walking, to use our metaphor (Varela et al. 1991, Ch. 9). One of the best illustrations of this idea, both for its biological and cognitive scientific detail, and for its philosophical wealth, comes from comparative color vision and ecological perception: colors do not exist out there, waiting to be represented internally; they are brought forth in ecologically situated, perceptually guided action (Thompson 1995; Thompson et al. 1992). In cognitive science, this nonadaptationist and non-representationist perspective on life and cognition is now known as the *enactive* approach (Chiel & Beer 1997; Clark 1997; Varela et al. 1991).

Rose refers to none of this work. Perhaps we should feel pleased that a distinguished biologist weaves as a central part of his argument ideas that others have toiled to develop: it must mean that these ideas have become part of our shared intellectual heritage, for all to use and enjoy. Nevertheless, citing one's sources for key concepts and ideas is an essential part of scientific practice. Had Rose done so, his book would have been more accurate; had he made use of the literature mentioned above, his argument would have been stronger. And we would have been able to give his arguments the attention they deserve, rather than voice this call for fairness.

High purpose, low execution

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Abstract: In reasserting the primacy of the individual in biological analysis, Rose directs attention away from the crucial insights of the developmental/structuralist perspective that he advocates. In presenting his advocacy as a diatribe, he brings disrespect down upon that very tradition.

On Steven Rose's own account, his goal in writing *Lifelines* is to "offer a coherent alternative frame work [to contemporary Darwinism] within which to interpret living processes." The promise of such a framework was enticing because of recent works that suggest that the Darwinian paradigm may be due for at least a thorough overhaul. The works I have in mind include Waldrop's (1993) *Complexity,* Goodwin's (1994) *How the leopard changed his spots,* Sapp's (1994) *Evolution by association,* Sober and Wilson's (1998) *Unto others,* and Depew and Weber's (1997) *Darwinism evolving.* Rose's *Lifelines* falls far short of its high purpose in both substantive and rhetorical ways.

Substance. Metaphors are essential to science, but every scientific metaphor incurs costs as well as benefits. The costs of using a metaphor arise from disanalogies between the metaphor and its object. These costs are weighed against the power of the metaphor to bring order to what we know and to suggest other discoveries we might make. The useful life of a scientific metaphor is brought to a close when the discoveries it stimulates reveal too many disanalogies.

The metaphor of natural selection is rooted in Darwin's correspondence with breeders, who taught him that the composition of a flock can be changed by selecting desirable individuals for mating. The metaphor thus focuses attention on the differential reproduction of individuals. But many of the findings described in the works cited above seem to suggest that in focussing on the individual, we miss much of what is essential in biology that is going on at levels of organization above and below the level of the individual.

These problems with the metaphor of selection are not going to be solved by frame shifting the metaphor to levels above or below the individual because at whatever level we care to look, objects seen as good entities from higher levels of organization appear as diaphanous webs of interaction when examined closely at the next level down. Thus, my hope for *Lifelines* was that it would provide a way of looking at biological organization that would broaden our focus beyond the individual as a privileged level of organization. Regrettably, Rose grabs the other end of the stick.

Rhetoric. Not only is Rose's vision of the future of biology disappointing, his arguments for that vision often seem incomplete, ill-founded, and even mean-spirited. Consider, for example, the passage that opens the Preface.

> The rise of the present enthusiasms for biologically determinist accounts of the human condition date to the late 1960s. They were not initiated by any specific advance in biological science, or powerful new theory, but harked back instead to an earlier tradition of eugenic thinking which . . . had been eclipsed and driven into intellectual and political disrepute in the aftermath of the war against Nazi Germany and its racially inspired Holocaust.

The passage is a good example of a rhetorical technique known as the Big Lie. Big Lies work for two reasons. First, they are so vague and all-encompassing that the evidence necessary to refute them is difficult to conceptualize, let alone marshal. Second, they suppress rebuttal by pre-stigmatizing it. Let us examine each of its components in detail:

> The rise of the present enthusiasms for biologically determinist accounts of the human condition date to the late 1960s.

If what the author is talking about here is what he later identifies as "ultra-Darwinism," then that movement dates from the publication of Wynne-Edwards (1962) voluminous, *Animal dispersion in relation to social behavior,* which asserts that much social behavior has been selected at the level of the population. At the core of this influential work was a dramatic analogy between animal social systems and the conventions that limited fishing fleets in the North Atlantic. This vivid concretization of the population-selection argument, which had been immanent in the literature for many years, prompted immediate individual-level responses from William Hamilton, George Williams, and Robert Trivers, followed only belatedly by E. O. Wilson and Richard Dawkins. In short, the intellectual developments that Rose ascribes to the late sixties actually evolved over a 20-year period from the early sixties to the late 70s.

> They were not initiated by any specific advance in biological science, or powerful new theory.

By any objective standard the period of which Rose speaks here was a time of breathtaking theoretical development and empirical achievement in the study of behavior and evolution. All over the U.S.A. and Europe, new graduate programs in behavior and evolution were started, and the deluge of new information about animal behavior was overwhelming. Two theoretical innovations that distinguish this period are often confused. The first might be called "biological individualism"; the interpretation of animal social organizations in terms of the interests of individuals. The second might be called "biological genism"; the interpretation of animal social organizations in terms of the interests of abstract units of inheritance called genes. Although the failure to discriminate these two positions has led to some important confusions (Sober & Wilson 1997; Thompson 1998; Thompson & Derr 1995; Wilson & Sober 1994), the general paradigm has proven to be extraordinarily productive, leading literally to thousands of investigations of the details of animal communication, reproduction, and social life.

> . . . but harked back instead to an earlier tradition of eugenic thinking.

The words "harked back" are the giveaway here. How can one ever hope to refute a "harking back"? I can only say, speaking as somebody who lived through that time and knew many of the participants, that I and my colleagues were interested in explanation, not in ideology.

> . . . had been eclipsed and driven into intellectual and political disrepute in the aftermath of the war against Nazi Germany and its racially inspired Holocaust.

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Here, is a reduction, indeed: a generation of field and laboratory investigations by hundreds of hard-working people reduced to a political epithet! My colleagues and I thought we were exploring the strengths and limitations of the Darwinian paradigm through experiment, observation, modeling and theory. As a consequence of Rose's perceptive socio-reductive analysis we now see that we were only engaged in a revanchist plot to restore the Third Reich! How silly of us not to have seen it before!

Were I wrapping up this review in Professor Rose's rhetorical style, I would dismiss his work as just another manifestation of the Cult of the Individual that has thrived in Post Cold War Britain. But such socio-cultural *reductios* have no place in responsible scientific writing.

How to alienate your natural allies and attract your enemies

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Abstract: Rose erroneously believes that the sciences of human behavior are being swept with a wave of genetic determinism. Actually, sociologists and psychologists remain predominantly hostile to any genetic influence on behavior. They will love Rose. The few behavior geneticists and sociobiologists in these disciplines are marginalized and looking for a little respect. Rose impugns their motives and ridicules their science.

Rose proclaims that his purpose is to promote "a vision which recognizes the power and role of genes without subscribing to genetic determinism" (p. 7). From his reading of Dawkins and the Sunday newspapers, he sees ultra-Darwinism (sociobiology) sweeping the sciences of human behavior. Rose and the "radical science movement" (as he calls it) have been fighting this battle for a long time. He sees it as a continuation of the battle against eugenics. Implicit racism, Rose charges, is the fundamental motivation of those who hold for either the evolutionary foundations of human nature or for the genetic explanation of individual variance in behavior (behavior genetics).

How big is the sweep of this takeover of the sciences by genetic determinism? As a biologist looking at the social and behavioral sciences, Rose seems to believe genetic determinism has carried the day. I live and work as a sociologist among other social scientists. From here the view is less clouded by the perceptions of journalists about the big science news. In sociology, sociobiologists are pariahs trying to claw a little toehold in the discipline. In psychology, what is now called evolutionary psychology may be big with the newspapers, but in the discipline as a whole it is a speck on the public image.

So it is humorous to think that sociology and psychology are being swept by a wave of genetic determinism. Generally we social scientists, like Rose, hold very strong views against it. But this is not because we want to be politically correct. We can't help ourselves. Our shared disciplinary immune systems recognize biological explanation of behavior as an infection, and reject it. (This is a metaphor, not a homology.) Rose would immediately recognize that our arguments against *any* biological explanation of behavior are his against genetic determinism. His arguments even contain the same innocent mistakes as ours do (for example, confusing the causes of secular trends with the causes of variance among individuals).

Rose says he wants us all to recognize the power of genes while rejecting genetic determinism. Maybe, because he says *Lifelines* is written to be a within-biology argument, his battle is with other biologists (he says it is mainly with those who don't *do* biological science, or at least not any more). His argument with the social science disciplines today does not need to focus on fighting genetic determinism so much as to help us accord any power to the genes at all when it comes to behavior. The only genetic determinists I can find are the rhetorical bogymen conjured up by Rose. Even the most radical behavior geneticists are theorizing (and finding) environmental effects on genetic process, and genetic influence on social processes.

In the real world of scholars today, let us concede to Rose (who must know) that there are genetic determinists among biologists. Perhaps he will reach them with his message that the organism shapes its own development. In the world outside biology there are scholars who believe human behavior in part evolved genetically (that is, that there is a human nature), and those who do not (that is, that there is no human nature). No one believes that human behavior is completely controlled by evolution. No one believes that variance in (any) human behavior is determined completely by genetic differences; but many believe that there is no genetic influence. From the social/behavioral sciences, *Lifelines* will appeal to those who reject evolutionary and genetic influence. How will this help Rose achieve his goal?

Rose's homeodynamic perspective is not an alternative to neo-Darwinism

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Abstract: *Lifelines* discusses two approaches to biology, "ultra-Darwinism" which Rose criticises, and the "homeodynamic perspective," which he offers as an alternative. This review suggests that ultra-Darwinism is a caricature of the theoretical positions Rose wishes to oppose and that the homeodynamic perspective is not an alternative, but is complementary to so-called ultra-Darwinism.

Lifelines discusses two approaches to biology. Rose calls one "ultra-Darwinism," and the other the "homeodynamic" perspective. Ultra-Darwinism, says Rose, has, among others, the following flaws:

1. It is biologically and "neurogenetically" determinist (pp. *viii, ix*).
2. It uses misleading information theoretic metaphors (pp. 121, 137).
3. It claims that the individual gene is the "unit of life" (p. 209).
4. It uses a "billiard-ball" model of the gene (p. 210).
5. It claims that every observable aspect of the phenotype of an organism is adaptive (pp. 210, 230).
6. It is preformationist (p. 212).
7. It entails mind-body dualism (p. 214).
8. It assumes a direct, unmodifiable link between gene and adult phenotype (pp. 215, 221).
9. It excludes processes of development and "the internal physiological processes which constitute the organism" (p. 215).
10. It claims that organisms are inherently passive (p. 244).

The homeodynamic perspective, by contrast, is said to be free of these major flaws and is offered as "a coherent alternative framework" (p. *ix*) which transcends genetic reductionism and places the organism at the centre of life.

Looking at the list of shortcomings above, one might be forgiven for thinking that ultra-Darwinism, as Rose presents it, is intended to portray an extreme view that no one actually holds. Thus its purpose would be essentially heuristic. But although in places Rose writes as though this were so (e.g., pp. *x* 209), he also assures the reader that there are many real ultra-Darwinists. In the last decade, he claims, "the stream of ultra-Darwinist and biologically determinist claims has become a torrent" (p. *viii*). Richard Dawkins and Dan Dennett are singled out early on as arch ultra-Darwinists, and E. O. Wilson merits a number of specific mentions. Moreover, there is a suggestion (p. 176) that ultra-Darwinism is just another name for neo-Darwinism, in which case one ought, presumably, to count among the ultra-Darwinists peo-

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ple like George Williams, Bob Trivers, Bill Hamilton, and John Maynard-Smith.

It is manifestly incorrect to attribute to neo-Darwinian theorists the catalogue of errors of which Rose wishes to convict them. His case does not stand up even to a cursory reading of the works of those he criticises. Nor are his arguments compelling. (See, for example, the clumsy attempt to justify the charge that Dawkins imports dualism into his explanatory framework, pp. 213–14.) One possible explanation for his claims is that Rose has simply failed to make a key distinction between the capacity to change behaviour and the capacity to change genetic structure.

It is a central tenet of neo-Darwinism that acquired characteristics are not passed on from one generation to the next by the biological process of reproduction. This understanding is encapsulated in the so-called "central dogma," originally expounded by August Weismann, who argued that information flow goes one way only from germ plasm to soma, or, in molecular terminology, from DNA to protein. Bodies cannot modify their genomes by practice or effort. The blacksmith cannot bequeath to his offspring the effects of his craft on his biceps. There are exceptions to the central dogma, but they are few in number and their significance is hard to assess. Exceptions are rare because most phenotypic changes are not adaptive. A hereditary mechanism that allowed the transmission of such changes would not be favoured by natural selection. Thus, in general, we do not have the capacity to change our genetic structure.

It does not follow from this that neo-Darwinists believe behaviour to be unmodifiable or that they think developmental processes are unimportant and can be ignored. If genes were only able to build reflex mechanisms then the human behavioural repertoire would indeed consist of nothing more than a set of fixed action patterns. But there is no reason at all to suppose that genes are limited in this way and every reason to suppose that they structure decision making mechanisms which are highly sensitive to environmental contingencies of many kinds. As Rose rightly says, an individual lifetime involves complex interactions between genes and environments. Neo-Darwinists are in full agreement with this proposition.

Rose's apparent inability to understand that "genes for" psychological mechanisms do not preclude behavioural flexibility may be related to his evident distaste for computational thinking. The significant point about computational thinking is that it has banished the last vestiges of dualism from contemporary psychology and shows precisely how to avoid the infinite regress of representational systems that plagued early attempts to achieve a satisfactory materialist basis for mental processing. It is just because information can be encoded in physically realisable structures that behavioural flexibility is possible and explicable in a non-circular way.

It is regrettable that Rose chose to present the homeodynamic perspective as an alternative to the neo-Darwinian perspective when in fact they are complementary. There are, for example, many evolutionary psychologists and behavioural ecologists who count themselves as neo-Darwinists, whose interests emphasise development and other ontogenetic issues which Rose claims are precluded by neo-Darwinism. Rose's treatment of sexual selection and kin selection theory (pp. 197–203) is an instance of this false opposition. He claims without argument that sexual selection theory "simply cannot encompass the rich diversity of human experience" (p. 198) and that kin selection theory suffers from a "relative lack of experimental support" (p. 202). To argue thus is to ignore a large and growing body of empirical evidence that illustrates the fruitfulness of both of these theories for the framing of testable hypotheses in psychology and behavioural ecology. Parent–offspring conflict, familial violence, and the nature of mate choice are all aspects of individual lifelines which are being illuminated by neo-Darwinist thinking.

Rose suggests (p. *viii*) that the views of those he criticises are based on shaky empirical evidence and unexamined ideological presuppositions. There is a certain irony in this claim because it is hard to avoid the conclusion that the ultra-Darwinism which Rose excoriates throughout *Lifelines* is essentially a product of his own theoretical proclivities and ideological assumptions. As a result, *Lifelines* fails as a critique of contemporary neo-Darwinian thinking in biology and also in psychology because there is no one out there who thinks as he claims they do.

Author's Response

Biological determinism lives and needs refutation despite denials

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Abstract: Commentators are divided between those who welcome and creatively extend the agenda of *Lifelines* and those who defend what it criticises. My response covers style; history, politics, and ethics; concepts of freedom, active organisms, and determinism; the uses of metaphor; reductionism and levels of analysis; Darwin and Darwinists; heritability and intelligence; human universals and biological determinism.

Reading reviews of one's book is always an interesting experience, and where the reviews are multiple as in the *BBS* format it can be particularly challenging. This mixed set of reactions has been instructive, and I have been forcefully reminded that the attempt to communicate by writing, a sort of contract between author and reader, requires mutual effort. The book a reader reads may very well not be that the author believes he has written. Indeed, in the case of some of the reviews of *Lifelines*, I have had a strong sense of *dejà vu* that the reviewers really felt they were reviewing the book I co-authored some 15 years ago with Richard Lewontin and Leo Kamin, *Not in our genes* (Rose, Lewontin & Kamin 1984). This is notably the case with **Alcock,** who even bolsters his rather adjective-laden argument by quoting, substantially out of context, a review of that book written by my colleague Pat Bateson. So I make no apology for restating here what *Lifelines* was intended, from this writer's perspective, to be about, and then replying in more detail to the specific points raised. In doing so I will also refer to two books which had not appeared when *Lifelines* was written but which illustrate some of its points rather clearly: Steven Pinker's *How the mind works* (1997) and E. O. Wilson's *Consilience* (1998) (see also the comments by **Hirsch** – whose defence of Loeb, by contrast, encourages me to re-evaluate that early mechanist).

Before even doing that, however, I owe one unreserved apology. **Thompson & Varela,** whilst generously commending the theses of the book concerning autopoiesis, rightly charge me with failing to credit the term to its originators, Maturana and Varela (1980). I was shocked to discover on rereading my own text that I had omitted this elementary scholarly responsibility, especially as I have always been conscious of the intellectual debt I owe to their work, and agree with them entirely that an appreciation of the origins of a concept is essential for a full grasp of its significance. I'll come back to the defence of autopoiesis

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against some of its other critics among the reviews later, but want to make it clear up front that the omission of credit, in every sense of the term, has now been fully rectified in any more recent edition of the book.

R1. What *Lifelines* is about

Contrary to the impression that one might get from reading some of the reviews of the book, *Lifelines* is not a political tract, nor a detailed discussion of the social origins or implications of biological determinism, least of all an affirmation of "Marxist philosophy," if indeed that protean epithet has any single meaning. The ideological and sociopolitical framework of the debate occupies only the preface and Chapter 10. Nor is it primarily a critique of sociobiology, although many of the presuppositions of sociobiology (these days rebranded as evolutionary psychology) are indeed discussed. The book is addressed to that mythical lay audience for which those of us concerned with the public understanding of science see ourselves as writing. It follows my previous book *The making of memory,* (1992) in attempting to lay out how my sort of biologist tries to extract scientific meaning from the world, the problems of objectivity in science, and of designing experiments. Some reviewers dismiss the need to do this on the grounds that everyone knows about such issues already. I wish that were true, but in my experience it just isn't, and even where it is, it is considered slightly indecent to mention it outside the charmed circle of active researchers. To appreciate the problem, one need only read books by distinguished biologists explaining their subjects with sublime indifference to such questions.

Having set out these themes, I turn my attention to the nature of explanation in biology, and confront the question of why many (including reviewers I will respond to more specifically below) seem to regard reductionist explanations as the only ultimately legitimate ones. At the core of the book are five chapters discussing genetics, development, evolutionary mechanisms, and life's origins and developing the framework within which I believe these need to be conceptualised: homeodynamics, autopoiesis, and complexity. These chapters argue for placing cells and organisms rather than nucleic acids at the centre of living processes. There is little enough about ecology and population dynamics in the book, which is to be regretted, as some reviewers point out, but this reflects my own perspective and limitations as a biochemist turned neuroscientist (*pace* **Cornish-Bowden** I don't just "claim" to be a biochemist). I confess to some surprise to discover that these core themes occupied so few of the reviewers, who preferred to respond to the periphery rather than the centre. Indeed almost the only person who did address them was indeed Cornish-Bowden, who sadly totally misunderstands the argument. He claims that I first provide a simplistic account of rate-limiting reactions in biochemistry, then correct that account, and finally argue that this means that genes are irrelevant to behaviour. In fact I do nothing of the sort; the discussion of rate-limiting reactions is, as my text makes very clear, a self-critical comment on early editions of a teaching text of mine, *The chemistry of life* (1991). The second is a description of the Kacser-Kauffman approach to metabolic complexity, which forms part of the explanation of why gene function and action can only be understood in the context of the metabolic web within which they are enmeshed; it is only remotely connected with the discussion of genes and behaviour, and nowhere in *Lifelines* or anywhere else have I ever made the absurd statement that genes have nothing to do with behaviour. Cornish-Bowden must read more carefully. But so be it: let me turn to the themes that did concern most of those who responded.

R2. On style

Some reviewers (**Economos, Charlton,** and **Crusio** amongst them) take exception to a certain robust style of writing, and my specific choice of Dawkins and Dennett as foils for some of my arguments. This is perhaps an excuse for the *ad hominem* approach of some of the reviews, but I make no apologies. The reasons for my choice are that these are amongst the most clear-cut defenders of the positions I criticise (Pinker [1997] and Wilson [1998] would also have served in this regard) and my use of them could almost be taken as a compliment. However, I have a strong feeling of double standards being applied. All four authors have been responsible for some pretty cheap shots aimed at, for instance Gould and Lewontin (and me) personally, and assorted but unspecified "leftists and feminists" in general, and I cannot recall their ever being rebuked – indeed, rather the reverse – for this.

R3. On history, politics, and ethics

Several reviewers assume that my criticism of biological determinism and excessively reductive, gene centred thinking is based on either political or ethical principles, rather than being grounded in a within-biology critique, and they take me to task accordingly. Thus **Charlton** states, without evidence, that I conflate ethical with pragmatic thinking, and that I regard oversimplification as a sin. *Au contraire,* I regard it as poor science, though I recognise that simplification is often a useful heuristic device, whether, to use his term, it is "accidental" or not. For instance, both Mendel's laws and the Central Dogma are, as we now know, simplifications, but they were both enormously potent and useful in their time. Both, however, have long outlived their usefulness, and to continue to claim them as "truth" is a sort of wickedness – though only to science and the scientific ethic, not to morality in general. I can't think for one moment why Charlton goes on to suggest that *Lifelines* attempts to provide a way of determining *in advance* whether a biological theory is oversimplified and therefore produces a general purpose short cut; I can't recall ever thinking such a thing, let alone writing it.

N. S. Thompson disputes the brief history of the rise of biological determinist thought that forms the Preface. I trace the recent history through the late 1960s to a eugenic past which I am sure all the reviewers regard as being as shameful as do I. My reference to the late 1960s relates specifically to the publication by Jensen (1969) on race and IQ and its aftermath. As this history has often been recounted (e.g., Rose et al. 1984), I didn't feel it was necessary to spell it out in detail. I certainly was not referring to Wynne-Edwards, group selection, and the debates within evolutionary theory which were going on, as Thompson notes, both before and after 1969. I am quite astonished that anyone could misread me as doing so, especially as later

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in the book and in a historically internalist contest I refer specifically and in detail to the history of the group selectionist/kin selectionist debate! If there is a Big Lie here it is certainly not mine. And it is absurd to suggest that I – or indeed any practicing biologist – could regard evolutionary biology as in some sense evil. If *Lifelines* says nothing else, it over and over again quotes Dobzhansky (1973) on the centrality of evolutionary thinking to understanding the living present. However, anyone amongst the commentators who doubts that these internal debates have political resonance need only refer to, amongst other things, the use of sociobiological claims by conservative writers (Ridley 1997), racist groups (Brunn 1978; Verrall 1980), antifeminists (Goldberg 1975; Wright 1994) and many other examples too tedious to list. This doesn't mean, *pace* **Schooler** and **Economos,** that I see a simple link between racism, genetics, and psychometry – although too often in the quite recent history of these sciences such links have indeed been present and painfully documented (e.g., Gould 1997). To claim that these issues are above "mere human politics" as Dawkins (1981) once did in an exchange with me in *Nature* is to bury one's head in the sand.

R4. On freedom, active organisms, and determinism

Lifelines uses the concepts of freedom and of determinism in a number of ways, and my failure to spell them out and distinguish between them more clearly is one of the weak points in my account; I have learned from all the reviewers who focus on this theme. The commentary by **Freeman** (see also Freeman 1999) is particularly helpful in emphasising intentionality and agency. It is the active part played by all organisms in shaping their own futures which I understand by the wonderful term autopoiesis. **E. Thompson & Varela** explicate (and cite) this term more adequately than I did in the book, and also point out that its utility extends to the cognitive sphere as well, and I am grateful to them. **Alcock**'s suggestion that I – or Varela and his colleagues – avoid a "detailed test" of autopoiesis merely confounds an empirical method with a theoretical framework.

Both **Fuller** and **Midgley** usefully clarify some of these issues, though from rather different perspectives. Fuller emphasises the distinction between subjective and objective senses of the term freedom – I may act in a way that "feels" free, for instance to buy this or that product, or to have or not have sex, but "objectively" I am serving the needs of capitalism or striving to propagate my selfish genes. This use of "objectively" parrots (I suspect Fuller's usage is deliberate, as he is no innocent in such matters) the ways that both vulgar Marxists and evolutionary psychologists talk. My point is much closer to Midgley's that the existence of explanations and actions at multiple levels means that there are many different forms of such "objectivity," and that freedom in part depends on the existence of this multiplicity. Fuller explicates the concept of "the freedom of necessity," commonly if crudely exemplified by the observation that we are all free to dine at the Ritz – provided we have the money to do so. Yet I mean more than just this. Of course we are all, as **Economos** points out, subject to the laws of gravity and chemistry (I won't dispute here the use of the concept of law) – an aspect of the argument about human "universals" to which I will return below. But I wish to argue that the processes of development and of evolutionary change are radically underdetermined in quite a different way (as I believe **Steklis** appreciates in his review). This isn't, let me assure **Leslie,** in his disappointment at the coda to the book, because I wish to sneak in vitalism or mentalism. It is rather because historical change comes at the meeting point of many "determinisms," physical, biological, social, and technological. Evolutionary mechanisms – natural selection amongst them – can only respond to the here and now; they cannot predict the future, and hence are constantly tracking moving targets – targets that move precisely because all living forms are constantly modifying them as well as themselves. This is the radical indeterminacy of evolution.

R5. On metaphors

Hull, Bradie, and **N. S. Thompson** comment on my concern over the power of metaphors in biology. They (and indeed others who had reviewed the first edition of the book) point out that metaphors are an essential scientific tool; we all do try to explain otherwise un-understandable phenomena by analogising them to things or processes we do understand – or at least that we believe we understand. I accept this – and indeed have tried to clarify my views in the preface to the paperback edition of the book. But I continue to maintain that metaphors are dangerous devices which must be handled with care and responsibility, because they seduce one into thinking that the phenomenon one is metaphorising is indeed in some way *the same as* that to which it has been likened. Dawkins in particular has a gift for metaphor which contributes to the brilliance of his writing but is often totally misleading, as in examples I quote in *Lifelines.* Furthermore, we seem to draw our metaphors in biology either from the behaviour of humans ("rape" in mallard ducks or "harems" amongst baboons, for instance) or from human technologies (e.g., brain memory as the same as computer memory). Similarly, the Darwinian metaphor has spread into realms in which it has no business, in my view – for instance, Hull's use of it to discuss the ways in which theories in science "compete" or Dennett's view of natural selection as a "universal acid." This is hazardous, not only because of the ideological and conceptual baggage that such transfers from one domain to another inevitably carry with them.

Of course **Steklis** is right that we do as scientists try to hold a mirror to reality, but it can never be a plane mirror; it is always shaped by – distorted by – our framing assumptions, metaphors, expectations. If the last three decades of debate amongst sociologists and philosophers of knowledge has taught us nothing else, it has certainly made clear that this is the *best* we can hope for – and there are many who would not even allow us that. I agree entirely therefore with **Bradie**'s points that science can never be free of ideology – noting that neither he nor I have paused to unpack the multiple meanings that that word itself has – and that metaphors serve useful heuristic and epistemic purposes along with their rhetorical function. **Miller** worries that, by pointing out that metaphors drawn from physics and technology may do disservice to biology, I appear to reduce Galileo's achievements to mere historico-social products. Nothing could be further from my intentions. They are indeed historico-social products, but not *merely* this;

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they also told us something important and at least partially transcendent about the way nature is. I'm not with the social constructionists in this debate.

Hull goes on to distinguish between homologies and homoplasies – a term I am not familiar with, but in his example of torpedo shapes for efficient swimming it sounds like a description which might relate either to my reference to constraints of structure and/or to convergent evolution. In any event I am happy with it. I am less happy with his assertion that no one still takes seriously the conflict between ovists and spermists originally discussed in Needham's classical history of embryology (for an up-to-date history of this episode, see Pinto-Correia 1997). Incidentally. Hull also calls attention usefully to the doubts over the "classic" natural selection story of industrial melanism and its reversal in peppered moths – doubts which strengthen rather than weaken my arguments. A further important point he raises concerns my critique of the concept of "natural kind" in biology (one of several in which I part company from, for instance, Webster & Goodwin [1997]). Agreeing with my doubts over whether species, organisms, or macromolecules can be regarded as natural kinds, he nonetheless claims that carnivores or founder populations can be so regarded. I disagree, though I don't feel strongly on the point; the functional definition is, it seems to me, even more in the eye of the researcher than the structural one. What is the logic of grouping together dogs and wasps on the grounds that they both feed on flesh? But whether after so much agreement Hull's final sentence follows, others must judge.

R6. On reductionism and levels of analysis

In *Lifelines* I distinguished between several meanings of the term reductionism, including theory reductionism (which did not concern me), methodological reductionism (which I regard as an essential aspect of science as it has developed historically in the West), philosophical, and ideological reductionism. These distinctions have not always been recognised by the reviewers. I ask why reductionism should be regarded as *the* explanatory goal. **Michel** responds by emphasising, rightly I think, that research pragmatics supports reductionism because it is easy, whereas what he calls "developmental research" is difficult to do and, because its results are often not simple, harder to get supported and published.

However, as in my fable of the frog, I insist that in biology there are multiple legitimate ways to describe phenomena, of which the reductionist way is but one, and any of these may be appropriate, depending on the purposes for which it is intended. **Miller,** whilst sharing my concern, suggests that reductionism is merely a "temporary aberration" which will in due course be transcended. I would like to think he is right, but I respond in the shadow of Wilson's *Consilience* (1998), which is an unblinking claim not merely for the necessity but also for the ultimate triumph of physical reductionism in eventually bringing even art and ethics into line (see my review, Rose 1999 and **Hirsch**). The doyen of philosophy of science, Thomas Nagel, insists that whereas a non-reductive account – for instance of the physiology of nerve transmission – merely "describes" the phenomenon, a reductive one – presumably in this case in terms of ions and membrane properties – "really explains" it (Nagel 1998, pp. 3–14). In my view, the reverse is often the case. I suspect that **Palm,** with his courteously expressed search for simplicity in scientific explanation, would share Nagel's view, which is the bedrock to the work of many distinguished mathematical modellers and indeed mirrors the criticism that nineteenth century physicists laid at Darwin's door when they called his proposed evolutionary mechanism "the law of higgledy-piggledy." If I dissent from it, it is because to an experimental biologist – as Palm himself reflects – the world is indeed inherently messy and contingent. Miller suggests that neurodynamics may be a more dangerous form of reductionism in what he sees as an assault on the concept of the person, because, presumably, it may "explain away" intentionality. I'm not sure I agree with him here. Freeman (1999) has made a substantial contribution to our understanding of the way that "meanings" and "intentions" may be translated into neurodynamic terms, but I don't think he would see this type of language as "explaining" rather than "describing," to use Nagel's terms. Think of the phrase "I am in love" and then consider reducing it to statements about neurodynamics, nerve impulses, hormonal pulses, and so on. Most of us, I suggest, would find the former a satisfying explanation of my behaviour (despite the disparagement of "folk psychology" by some computational neurophilosophers), the latter merely a description.

Economos argues the that denying reductionism means believing in the supernatural or denying any laws or adding "new primitive terms" to "quarks." I am, she says, an emergentist (though apparently not "a scholar and a gentleman" – but then I've never laid claim to the second of these terms). What I do argue is that terms and concepts relevant to one level of organisation of the world are often not meaningfully translatable into others. To take some examples from Midgley (1998), how would Economos translate "money" or "justice" into chemical or even biological terms? It is in this context that I understand **Rowell**'s unpicking of the complexities of mate selection and her reference to cooperative phenomena in troops of social animals. I appreciate the reference to the need to consider hierarchies in space and time; this is surely right and not at all fully dealt with in *Lifelines.*

My discussion of reductionism also raises issues about levels of description and introduces the term translation rather than causality to describe their relationships. **Sharov** agrees, but then goes on to claim that I am mistaken to argue that "selection at the level of genes [presumably he means genomic not individual genes] as something ontologically different from selection at the level of organisms" – these, he says, using my own metaphor, are different languages for describing the same thing. I don't think he is right; we are dealing here with a nested hierarchy of selection processes, not a mere translation.

Several commentators point to the dangers of replacing genic or physical reductionism with sociological reductionism, and I entirely agree. **Stein** is an example, and although he expresses important worries about the dominance of neuroscientific explanations in psychology and psychiatry, he goes on to argue that there are nonetheless important insights to be gained from such an approach. He cites Alzheimer's as an example, and I am especially pleased to concur, as our own lab is heavily involved in such research at present (Lancashire et al. 1999; Mileusnic et al. 1999). He points out too that Brunner (1996) has dissociated himself from some of the uses made of his study of a Dutch

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pedigree with an apparent linkage between MAOA and "violence." Indeed he has, and very helpfully too, though as I point out below, this hasn't prevented others from interpreting it very crudely indeed.

I must emphasise again, as I do repeatedly in the book, that the point is not to deny that there are likely to be particular features of the brain of a person who repeatedly kills or is violent to others in a civilian rather than a military context, and indeed that there may be specific genes associated with the development of such features, but that this reductionist description may not help us in trying to understand or limit the level of violence in society. The task of good science and of effective social policy is to try to identify determining levels of causation and potential sites of intervention. In this context, **Fuller** argues for the need to distinguish "genuinely efficacious" interventions from merely "convenient" ones. I couldn't agree more, but would point out that these very terms are not neutral. Efficacious for whom? Convenient to whom? What might be socially both convenient and efficacious, for instance, transportation for sheep stealing to British landowners in the eighteenth century, might not be either for the transportees.

It is precisely here that I argue, *contra* **Schooler,** that the use of ritalin to treat children given an ADHD diagnosis is – to put it no more strongly – frequently misguided: efficacious and convenient perhaps for schools authorities, but distinctly less so for the child given the drug, granted its many less than desirable pharmacological effects (Breggin 1999). In this context I am quite puzzled by Schooler's claim that I am disrespectful of sociology and want to colonise it for biology. **Anderson** makes a similar claim: that I want biology to have a veto over psychology. Some mistake here surely. Wilson (1998) explicitly and Pinker (1997) certainly implicitly make such claims, but nothing could be further from my intentions or my arguments; one of the points of *Lifelines* is to argue (*contra* Wilson, Pinker, and evolutionary psychologists in general) for the relative autonomy of both psychological and sociological levels of description. Anderson goes on to claim an even more damaging veto, in asserting that behaviour genetics can determine the general phenomena that a psychological theory must accommodate. Indeed, other reviewers, such as **Economos,** criticise me for apparently going too far in this direction. My attempt to resist such biological colonisation could scarcely be otherwise, as I have lived and co-written with a sociologist for the best part of forty years! Perhaps Anderson and Schooler misinterpret me because I am as unhappy about the psychometrising of sociology as I am about psychometry's reductive approach to psychology, and because I have an (outsider's) preference for these disciplines' more dynamic and qualitative rather than quantitative and functionalist traditions. Despite Schooler, the antithesis of "rigorous psychometric measures of such psychological characteristics as job satisfaction" is not necessarily "armchair speculation," as a multitude of sociologists and psychologists of less reductive schools would speedily attest.

R7. On Darwin and Darwinists

In their anxiety to stamp out what they see as heresy, a number of reviewers imply that my critique of ultra- or fundamentalist Darwinism is equally a critique of the neo-Darwinian synthesis of the 1930s, or of Darwin himself. Thus **Wells** claims that Darwin's central tenet is Weissmanism. Is it? Darwin himself knew better, as later editions of *The origin* attest, and Weissmanian dogma is itself, and always has been, under challenge from a number of sources, especially biochemical, developmental and microbiological, several of which are cited in *Lifelines,* notably Bonner (see also Dover 1992; 2000). And consider the recent flurry of interest in prions, or in protein splicing (e.g., Cooper & Stevens 1995). I'm afraid Wells's review illustrates the mess that nonbiologists often get into in their enthusiasm for over-simple philosophically rather than data-driven inputs into this debate. **Charlton** claims that I ignore good work done by evolutionary biologists and cites in particular Szathmary and Maynard Smith's (Maynard Smith & Szathmary 1995) work on major transitions in evolution to defend Maynard Smith from a charge I never make: that he is himself an ultra-Darwinist in the Dawkins/Pinker sense. There are many interesting themes in that book, especially in its early chapters, and Maynard Smith is far too subtle a thinker ever to be so branded, despite his association with the "fundis." As I point out in *Lifelines,* his models of evolutionary stable strategies are quite consonant with population level selection.

R8. On heritability and intelligence

Crusio asks what grounds I have for believing that intelligence is a more complex phenotype than milk yield, where I agree that heritability measures may be of relevance. I dispute two points. The first is that "intelligence" is definable in a way that makes it a phenotype at all, unless it is reduced to a fixed lump in the head called IQ. For a review of why intelligence is not reducible to IQ, see Richardson (1998; 1999). Here I agree with **Anderson,** who states that whether intelligence is quantifiable or not depends on whether we have a theory that allows for the quantification of its constructs. I deny that such a theory could be valid, but claim that intelligent behaviour, thought, and action are emergent interactions, socially defined for the most part, between individuals and their social and physical environment. They are only partially "embedded" within the brain and body of the person concerned. Of course there are brain processes, of many sorts, involved in this interaction, as **Martindale** points out; it would be highly surprising to any neuroscientist if there were not. "Speed of processing" or some EEG parameters may be among them. But before we get too carried away with such relatively crude correlations, let me point to the difficulties that 40 years of good neuroscientific work have encountered in attempting to define the molecular, cellular, and physiological correlates of even such a seemingly tractable problem as simple association learning in animal models (see, e.g., Rose 1993, for a review).

I appreciate the richer discussion of these issues by **Depue** and the attempt to ground such measures as extraversion in neurobiological parameters. His discussion of the capacity – surely a relevant biological process – by which stability may be generated through plasticity is important, though I feel as queasy about these psychometric constructs as I do about IQ. More fundamentally, for all the reasons specified in *Lifelines,* I challenge the obsession that psychometricians and behaviour geneticists have with the fundamentally meaningless statistic called heritability. I

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thank **Hirsch** for his further development of this point (and for pointing to several minor errors in the text of the hardback, most of which have been caught and corrected in the paperback). But even if one grants behaviour geneticists their use of the term, its application in twin and related studies is highly problematic, as **Segalowitz,** following in a long tradition, points out. For **Anderson** to suggest that "measuring two identical twins [reared apart] is just about indistinguishable from measuring the same twin twice" suggests to me that he has never spoken to twins, even those reared together. Martindale also refers to my regret that Bouchard was unwilling to provide access to his primary data – an unwillingness that he has also shown to date in response to requests from researchers undertaking meta-analyses of published twin studies – on the grounds of confidentiality. There are, of course, easy ways to protect such confidentiality via coding, and I remain unconvinced.

R9. On human universals and biological determinism

Unsurprisingly, granted the readership of *BBS,* these are the themes which have attracted mostly hostile responses. **Broude** sets the issue up by providing an entire paragraph of apparent human universals, derived from a bizarre list attributed to the ancestral positivist anthropologist G. P. Murdock (1945), long abandoned in the anthropological community, though clearly still an influence amongst some psychologists. *Of course* there are such universals; the societies we create are profoundly shaped by the length of the human lifespan and the neoteny of our offspring, to say nothing of our size (as Haldane [1985] pointed out many years ago in the marvellous sentence which forms the epigram to one of my chapters [Ch. 8], or the fact that we breathe air not water, and are omnivorous. As Ahouse (personal communication) points out in a comment on Wilson's view about human art, expressed in *Consilience,* no one would expect dogs to paint in colour, or I would add, humans in infra-red or ultra-violet – until the arrival of technologies that made these wavelengths "visible" to us. But to say that gestures, cooking, tool-making, trade, or ethics are human universals is to say absolutely nothing other than that humans are social animals who have developed technologies for living, and it entirely ignores the diversity of forms that these assumed Platonic natural kinds may take. To add kin groups to these might imply that the social definition of kin is coterminous with the biological one used in definitions of inclusive fitness, yet of course it is nothing of the sort; social definitions of kin are remarkably complex and varied (the *reductio ad absurdum* being to discover that young children often include their pets within their definitions of their family!).

The serious theoretical issue lies in our understanding of how such commonalities as there are emerge. The argument of evolutionary psychologists is that they are genetically built into our behaviour as a result of evolutionary selection pressures. This is the line taken by Pinker (1997) following for instance, Cosmides and Tooby (Barkow et al. 1992). For them, behaviour and the mind are constructed from genetically fixed modules – Cosmides and Tooby's famous Swiss army knife analogy. Of course this doesn't mean – and *pace* **Alcock, Wells,** and several other reviewers, I never for one moment imply that it does – that such behaviour is unmodifiable; clearly the "modules" are supposed to permit flexibility. (Incidentally, I did not invent the story of pink-feathered flamingos – it first surfaced in an account by Thayer in 1909! I am grateful to Bateson (Bateson & Martin 1999) for the account of this piece of proto-adaptationism). There is no space to deal with this argument at length here (see Rose & Rose 2000). Suffice it to say first that the innate modular argument (whose history stretches back to Plato) ignores both development (Karmiloff-Smith 1992) and the constructivist alternatives offered by Oyama (1986) and Ingold (1986; 2000; see also Bateson 2000 for a clarification of the multiplicity of meanings given to the term instinct), or even the connectionist approach of Elman et al. (1996). It is precisely the crudity of this view of innate modularity that the concept of autopoiesis counters.

It is the evolutionary psychologist's sense of biological determinism, not some fantasy of **Alcock**'s imagining, that *Lifelines* is concerned to counter, on both empirical and theoretical grounds. **Udry** believes that there isn't enough biological determinism about, though this claim to be a persecuted minority is clearly belied by the evidence of the continuing mass sales of many of the authors I have mentioned. But the defenders of biological determinism want to have it both ways. Thus even though he, **Steklis, Alcock,** and others maintain that insofar as they do exist, evolutionary psychologists do not make the determinist claims I attribute to them, one only has to read **Broude**'s shopping list to get a contradictory sense, though even she doesn't go so far as offering, as some contemporary Murdockians have, children's alleged dislike and adults' liking for spinach, or early morning sickness or "food cravings" in pregnancy, as evolutionarily honed adaptations. Consider too Pinker's (1997) and Wilson's (1998) rooting of an alleged human preference for green landscapes to our evolutionary origins in the savannah. Their accounts often read like a scientific version of The Flintstones – a projection of idealised US suburban life circa 1955 back into the neolithic.

The empirical evidence for such claims is often extremely shaky, and where it exists it is often much better explained on proximate rather than distally causal grounds. **Wells** refers to familial violence as a parent–offspring conflict. But what is an evolutionary psychologist to make of the (empirical) fact that violence by men against their female partners is particularly high during pregnancy, when the women are carrying their partner's child-to-be? Yet evolutionary psychologists, ignoring the huge differences in scale, prefer instead to dwell on the tiny number of murders by step-fathers of their offspring. I believe, along with anyone familiar with such evidence and not constrained by a fundamentalist need to shoehorn all aspects of human existence into a rigidly defined adaptationist framework, that proximal explanations at the social, economic, and personal life-history level are likely to give us much more purchase on understanding such phenomena than half-baked pseudo-Darwinism.

I cite many examples of such biological determinist claims in *Lifelines,* but here are some more for good measure (and see also Brown 1999): "our belief in morality is merely an adaptation put in place to further our reproductive ends" (Wilson 1975, quoted by Dennett 1995); "[the human mind is] equipped with a body of genetically determined information specific to universal grammar" (Smith & Tsimpli 1995); "the mind is likely to contain blueprints for

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grammatical rules . . . and a special set of genes that help wire it in place" (Pinker 1995); "From pregnancy complications, to the stress response, to the beauty in symmetry, to the attraction of money, to the historical tendency of the rich to favor firstborn sons, everything we think, feel and do might be better understood as a means to the spread of our own – or of our ancestor's – genes" (Betzig 1997).

Or consider Hamilton's romantically racist eugenicism, expressed in a paper delivered to a recent conference at the Vatican Academy (Pontifical Academy 1998), where he explained, to the distress of most of the other participants, that he would willingly sacrifice "a hundred unknown Chinese" to save a panda from destruction. Claiming as **Maxson** and **Steklis** do, that such biological determinists allow for epistasis and epigenetics scarcely addresses the point: any genetic effect has to work via epigenetic processes. When I turn to the question of behaviour genetics and neurogenetic determinism, I am not suggesting either that genes are irrelevant to an understanding of human behaviour or that determinists believe their effects are unmediated. Nor has Maxson any grounds for claiming that I "believe that most if not all [adaptations] involve selection at the group rather than the individual level" or that I believe that heritability estimates are only suitable for nonhumans (but see Sober & Wilson, 1998, for an extremely useful reevaluation of the dogmatic anti-group selectionist approach of many neo- as well as ultra-Darwinists; see also Wilson & Sober: "Reintroducing Group Selection to the Human Behavioral Sciences" *BBS* 17(4) 1994). Maxson is of course right when he points out that *Lifelines* makes no reference to *C. Elegans* – however he misquotes and therefore makes a nonsense of my statement on p. 4 that "*biochemical and* genetic generalisations are still derived from just three organisms" by omitting the two words italicised. This distortion comes poorly from someone who claims that I quote out of context.

The reductive sequence I describe in Chapter 10 of *Lifelines* begins with questioning the validity of reifying and objectifying the "behaviour" (in my major example, "violence" or "aggression") and ends with a critique of the assumption of a genetic explanation as "the" determining cause. As both **Fuller** and **Midgley** recognise, the search for causal explanations is also a search for efficacious sites of intervention, and it is in this sense that the attempts to explain "violence" in society in terms of genes or MAOA inhibitors is at best irrelevant and at worst positively harmful, for the reasons that chapter set out. **Crusio** defends the conclusions of the Cases et al. (1995) paper against my critique before describing an interesting experiment of his own with whose modest interpretation I have no major problems. My objection to the highlighting of "aggression" in the Cases et al. paper is that the genetically modified mice the authors describe as aggressive also had so many other phenotypic abnormalities as to make this particular feature a rather trivial aspect of their profoundly disabled lives, and the insouciance of the authors' response, described in *Lifelines*, emphasising this particular aspect of their mice's behaviour as a deliberate attempt to draw attention to their findings. Well indeed it has, and hence the rhetorical transmogrification to which Crusio refers must be seen as an interpretation (albeit metaphorical) of the authors' intentions.

In sum, I thank those many reviewers of *Lifelines* who have reflected so thoughtfully on the issues it raises, and in doing so have pushed me to think further and harder about several of its themes. As for the hornet's nest of behaviour geneticists, I cannot say that I am surprised that I have stirred them up, but conclude that despite their buzzing, my book has remained largely immune to their stings.

References

Letters "a" and "r" appearing before authors' initials refer to target article and response, respectively.

Ahmed, S. & Koob, G. (1998) Transition from moderate to excessive drug intake: Change in hedonic set point. *Science* 282:298–300. [RAD]

Alexander, R. (1987) *The biology of moral systems.* Aldine de Gruyter. [JA]

Allen, T. F. H. & Starr, T. B. (1982) *Hierarchy: Perspectives for ecological complexity.* The University of Chicago Press. [TER]

Bachman, P. A., Luisi, P. L. & Lang, J. (1992) Autocatalytic self-replicating micelles as models for prebiotic structures. *Nature* 357:57–59. [ET]

Barkow, J. H., Cosmides, L. & Tooby, J. (1992) *The adapted mind: Evolutionary psychology and the generation of culture.* Oxford University Press. [SCM, rSR]

Bateson, P. (1985) Sociobiology: The debate continues. *New Scientist* 105:58–59. [JA]

Bateson, P. P. G. & Martin, P. (1999) *Design for life.* Cape. [rSR]

Baxter, L. R., Jr., Schwartz, J. M., Bergman, K. S., Szuba, M. P., Guze, B H., Mazziotta, J. C., Alazraki, A., Selin, C. E., Ferng, H. K., Munford, P. & Phelps, M. E. (1992) Caudate glucose metabolic rate changes with both drug and behavior therapy for OCD. *Archives of General Psychiatry* 49:681–89. [DJS]

Betzig, L. (1997) *Human nature: A critical reader.* Oxford University Press. [rSR]

Bonner, J. T. (1974) *On development: The biology of form.* Harvard University Press. [aSR]

Borgia, G. (1994) The scandals of San Marco. *Quarterly Review of Biology* 69:373–75. [JA]

Bouchard, T. J. (1983) Do environmental similarities explain the similarity in intelligence of identical twins reared apart? *Intelligence* 7:175–84. [JA]
(1994) Genes, environment, and personality. *Science* 264:1700–701. [RAD]
(1997) Experience producing drive theory: How genes drive experience and shape personality. *Acta Paediatrica* 86 (Suppl. 422):60–64. [aSR]

Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L. & Tellegen, A. (1990) Sources of human psychological differences: Minnesota study of twins reared apart. *Science* 250:223–28. [SCM]

Bouchard, T. J., Jr. & McGue, M. (1990) Genetic and rearing environmental influences on adult personality: An analysis of adopted twins reared apart. *Journal of Personality* 58:263–93. [SCM]

Bouchard, T. J., Segal, N. L. & Lykken, D. T. (1990) Genetic and environmental influences on special mental abilities in a sample of twins reared apart. *Acta Genetica Gemellologica* 39:193–206. [CS]

Bradie, M. (1997) Explanation as metaphysical redescription. *Metaphor and Symbol* 2:125–39. [MB]
(1999) Models and metaphors in science. In: *After the received view,* ed. G. Preyer, G. Peter & A. Ulfig. A special issue of *Protosociology* 12. http://www.rz.uni-frankfurt.de/protosociology. [MB]

Breggin, P. R. (1998) *Talking back to Ritalin.* Common Courage Press. [rSR]

Bronfenbrenner, U. (1975) Nature with nurture: A reinterpretation of the evidence. In: *Race and IQ,* ed. A. Montagu. Oxford University Press. [SJS]

Broude, G. J. (1994) *Marriage, family, and relationships.* ABC-CLIO. [GJB]

Brown, A. (1999) *The Darwin wars: How stupid genes became selfish gods.* Simon and Schuster. [rSR]

Brunn, J. (1978) *La nouvelle droit.* Oswald. [rSR]

Brunner, H. G. (1996) MAOA deficiency and abnormal behavior: Perspectives on an association. In: *Ciba Foundation Symposium 194: Genetics of criminal and anti-social behaviour,* ed. G. R. Bock & J. A. Goods. Wiley. [rSR, DJS]

Brunner, H. G., Nelen, M., Breakefield, X. O., Ropers, H. H. & van Oost, B. A. (1993) Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science* 262:578–80. [WEC]

Buss, D. M. (1994) *The evolution of desire.* Basic Books. [HDS]

Carlier, M., Roubertoux, P. L. & Pastoret, C. (1991) The Y chromosome effect on intermale aggression in mice depends on the maternal environment. *Genetics* 129:231–36. [WEC]

Caryl, P. G. (1994) Early event-related potentials correlate with inspection time and intelligence. *Intelligence* 18:15–46. [CM]

Cases, O., Seif, I., Grimsby, J., Gaspar, P., Chen, K., Poumin, S., Muller, U., Aguet, M., Babinet, C., Shih, J. C. & De Maeyer, E. (1995) Aggressive behavior and altered amounts of brain serotonin and norepinephrine in mice lacking MAOA. *Science* 268:1763–66. [WEC, rSR]

https://doi.org/10.1017/S0140525X99442207 Published online by Cambridge University Press

Castellanos, F. X. (1997) Toward a pathophysiology of attention-deficit/hyperactivity disorder. *Clinical Pedriatrics* 36:381–93. [DJS]
Chiel, H. J. & Beer, R. D. (1997) The brain has a body: Adaptive behavior emerges from interactions of nervous system, body and environment. *Trends in Neurosciences* 20:553–57. [ET]
Clark, A. (1997) *Being there: Putting brain, body, and world together again.* MIT Press/A Bradford Book. [ET]
Clayton, N. S. (1994) The influence of social interactions on the development of song and sexual preferences in birds. In: *Causal mechanisms of behavioral development,* ed. J. A. Hogan & J. J. Bolhuis. Cambridge University Press. [GFM]
Coccaro, E. F. & Kavoussi, R. J. (1997) Fluoxetine reduces impulsive aggressive behavior in personality disordered patients: Results from a double blind placebo controlled trial. *Archives of Clinical Psychiatry* 54:1081–88. [DJS]
Colby, K. M. (1981) Modeling a paranoid mind. *Behavioral and Brain Sciences* 4:515–60. [DJS]
Collins, C. (1995) Letter to Professor R. Plomin, 22 June, 1995. In: *J. Hirsch Papers,* University of Illinois Archives, No. 15/19/22. [JH]
Collins, P. & Depue, R. (1992) A neurobehavioral systems approach to developmental psychopathology: Implications for disorders of affect. In: *Developmental psychopathology, vol. 4,* ed. D Cichetti. University of Rochester Press. [RAD]
Conrad, P. & Schneider, J. (1980) *Deviance and medicalization: From madness to sickness.* Mosby. [DJS]
Cooper, A. A. & Stevens, T. H. (1995) Protein splicing - self-splicing of genetically mobile elements at the protein level. *Trends of Biochemical Science* 20:351–56. [rSR]
Cords, M., Mitchell, B., Tsingalia, H. M. & Rowell, T. E. (1986) Promiscuous mating among blue monkeys in the Kakamega forest, Kenya. *Ethology* 72:214–26. [TER]
Costa, P. & McCrae, R. (1994) Stability and change in personality from adolescence through adulthood. In: *The developing structure of temperament and personality from infancy to childhood,* ed. C. Halverson, G. Kohnstamm & R. Marten. Erlbaum. [RAD]
Crusio, W. E. (1990) Estimating heritabilities in quantitative behavior genetics: A station passed. *Behavioral and Brain Sciences* 13:127–28. [WEC]
(1996) The neurobehavioral genetics of aggression. *Behavior Genetics* 26:459–61. [WEC]
(1999) An introduction to quantitative genetics. In: *Neurobehavioral genetics: Methods and applications,* ed. B. C. Jones & P. Mormède. CRC Press. [WEC]
Daly, M. & Wilson, M. (1987) Evolutionary psychology and family violence. In: *Sociobiology and psychology,* ed. C. Crawford, M. Smith & D. Krebs. Erlbaum. [JA]
(1988) *Homicide.* Aldine de Gruyter. [HDS]
Davidson, D. (1980) Actions, reasons, and causes. In: *Essays on actions and events.* Clarendon Press. [WJF]
Dawkins, R. (1976) *The selfish gene.* Oxford University Press. [SCM, JE, AAS]
(1981) Letter to *Nature. Nature* 289:528. [rSR]
(1982) *The extended phenotype.* Oxford University Press/Freeman. [SCM, aSR]
(1986) *The blind watchmaker.* Longman. [aSR, AAS]
(1995) *River out of Eden.* Weidenfeld & Nicolson. [aSR]
Dennett, D. C. (1995) *Darwin's dangerous idea: Evolution and the meaning of life.* Allen Lane. [arSR]
Depew, D. J. & Weber, B. H. (1997) *Darwinism evolving.* MIT Press. [NST]
Depue, R. & Collins, P. (1999) Neurobiology and the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences* 22(3):491–568. [RAD]
De Waal, F. (1996) *Good natured: The origins of right and wrong in humans and other animals.* Harvard University Press. [DJS]
Dobzhansky, T. (1968) Genetics and the social sciences. In: *Biology and behavior: Genetics,* ed. D. C. Glass. The Rockefeller University Press. [SCM]
(1973) Nothing makes sense except in the light of evolution. *American Biology Teacher* 35:125–29. [arSR]
Dover, G. (1992) Observing development through evolutionary eyes: A practical approach to molecular coevolution. *Bioessays* 14:281–87. [rSR]
(2000) *Dear Mr. Darwin. . . .* Weidenfeld and Nicholson. (forthcoming). [rSR]
Dunn, L. C. (1965) *A short history of genetics: The development of some of the main lines of thought, 1864–1939.* McGraw-Hill. [JH]
Edelman, G. (1987) *Neural Darwinism.* Basic Books. [aSR]
Elman, J. L., Bates, E. A., Johnson, M. H., Karmiloff-Smith, A., Parisi, D. & Plunkett, K. (1996) *Rethinking innateness: A connectionist perspective on development.* MIT Press. [GFM, rSR]
Endler, J. (1986) *Natural selection in the wild.* Princeton University Press. [JA]
Fisher, R. A. (1930) *The genetical theory of natural selection.* Oxford University Press. [SCM]
Freeman, W. J. (1999) *How brains make up their minds.* Weidenfeld and Nicholson. [rSR]
(in press) Consciousness, intentionality, and causality. *Journal of Consciousness Studies.* [WJF]
Gehring, W. J. (1998) *Master control genes in development and evolution: The homeobox story.* Yale University Press. [SCM]
Goldberg, S. (1975) *The inevitability of patriarchy.* Morrow. [rSR]
Goodwin, B. (1994) *How the leopard changed its spots.* Weidenfeld & Nicolson. [NST]
Goodwin, F. K. & Jamison, K. R. (1990) *Manic-depressive illness.* Oxford University Press. [CM]
Gottlieb, G. (1976) The role of experience in the development of behavior and the nervous system. In: *Neural and behavioral specificity,* ed. G. Gottlieb. Academic Press. [HDS]
Gottlieb, G., Wahlsten, D. & Lickliter, R. (1998) The significance of biology for human development: A developmental psychobiological systems view. In: *Handbook of child psychology, vol. 1: Theoretical models of human development, 5th edition,* ed. R. M. Lerner. Wiley. [GFM]
Gould, S. J. (1997) *The mismeasure of man.* Penguin. [rSR]
Gould, S. J. & Lewontin, R. C. (1979) The spandrels of San Marco and the Panglossian paradigm: A critique of the adaptationist programme. *Proceedings of the Royal Society of London B* 205:581–98. [aSR]
Gray, J. (1992) Neural systems, emotion and personality. In: *Adaptation, learning and affect,* ed. J. Madden, S. Matthysee & J. Barchas. Raven Press. [RAD]
Greenough, W. T. (1986) What's special about development? Thoughts on the bases of experience-sensitive synaptic plasticity. In: *Developmental neuropsychology,* ed. W. T. Greenough & J. M. Juraska. Academic Press. [HDS]
Haier, R. J., et al. (1988) Cortical glucose metabolic rate correlates of abstract reasoning and attention studied with Positron Emission Tomography. *Intelligence* 12:199–212. [CM]
Hamilton, W. D. (1996) *Narrow roads of gene land: The collected papers of W. D. Hamilton.* Freeman. [rSR]
Harris, J. R. (1998) *The nurture assumption.* The Free Press. [HDS]
Heinisch, J. (1986) Isolation and characterisation of the two structural genes coding for phosphofructokinase in yeast. *Molecular and General Genetics* 202:75–82. [AC-B]
Hesse, M. (1966) *Models and analogies in science.* University of Notre Dame Press. [MB]
Hinde, R. A. (1974) Interactions, relationships, and social structure in non-human primates. *Symposium of the 5th Congress of the International Primate Society, Nagoya, Japan,* 13–24. [GFM]
Hirsch, J. (1973) Introduction to J. Loeb (1918) *Forced movements, tropisms, and animal conduct.* Dover (reprint). [JH]
(1997) Some history of heredity-vs-environment, genetic inferiority at Harvard (?), and The (incredible) bell curve. *Genetica* 99:207–24. [JH]
(1999) The pitfalls of heritability: Can all tangible phenomena really be reduced to the laws of physics? Review of E. O. Wilson's *Consilience: The unity of knowledge* (1998). *The Times Literary Supplement,* February 12, 1999, p. 33. [JH]
Hofer, M. (1981) *The roots of human behavior.* Freeman. [HDS]
Ingold, T. (1986) *Evolution and social life.* Cambridge University Press. [rSR]
(2000) Explaining walking. In: *Coming to life,* ed. H. Rose & S. Rose. Crown/Random House. (forthcoming). [rSR]
Jacquard, A. (1983) Heritability: One word, three concepts. *Biometrics* 39:465–77. [JH]
Jensen, A. (1969) How much can we boost IQ and scholastic achievement? *Harvard Educational Review* 39:1–23. [rSR]
Jensen, A. R. (1982) Reaction time and psychometric g. In: *A model for intelligence,* ed. H. J. Eysenck. Springer. [CM]
Jensen, A. R. & Sinha, S. N. (1993) Physical correlates of human intelligence. In: *Biological approaches to the study of human intelligence,* ed. P. Vernon. Academic Press. [CM]
Kacser, H. & Burns, J. A. (1979) Molecular democracy: Who shares the controls? *Biochemical Society Transactions* 7:1149–61. [AC-B]
Karmilloff-Smith, A. (1992) *Beyond modularity: A developmental perspective on cognitive science.* MIT Press. [rSR]
Kempthorne, O. (1978) Logical, epistemological and statistical aspects of nature-nurture data interpretation. *Biometrics* 34:1–23. [JH]
Kleinman, A. (1988) *Rethinking psychiatry: From cultural category to personal experience.* New York Press. [DJS]
Kramer, P. D. (1994) *Listening to Prozac.* Fourth Estate. [DJS]
Krebs, J. R. & Davies, N. B. (1993) *An introduction to behavioural ecology. Third edition.* Blackwell. [SCM]
Lakoff, G. (1987) *Women, fire and dangerous things: What categories reveal about the mind.* University of Chicago Press. [DJS]
Lancashire, C., Mileusnic, R. & Rose, S. P. R. (1998) Apoliprotein antibodies affect the retention of passive avoidance memory. *Neural Plasticity* 6:29–40. [rSR]
LeDoux, J. (1992) Brain mechanisms of emotions and emotional learning. *Current Opinion in Neurobiology* 2:191–97. [RAD]

https://doi.org/10.1017/S0140525X99442207 Published online by Cambridge University Press

Le Moal, M. & Simon, H. (1991) Mesocorticolimbic dopaminergic network: Functional and regulatory roles. *Physiological Reviews* 71:155–234. [RAD]
Lewontin, R. C. (1998) Theoretical population genetics in the evolutionary synthesis. In: *The evolutionary synthesis,* ed. E. Mayr & W. B. Provine. Harvard University Press. [SCM]
Lewontin, R. C., Rose, S. & Kamin, L. (1984) *Not in our genes.* Random House. [JA]
Lykken, D., McGue, M., Tellegen, A. & Bouchard, T. J. (1992) Emergenesis: Genetic traits that do not run in families. *American Psychologist* 47:1565–77. [SJS]
Lykken, D. & Tellegen, A. (1996) Happiness is a stochastic phenomenon. *Psychological Science* 7:186–89. [RAD]
Margulis, L. & Sagan, D. (1986) *Origins of sex.* Yale University Press. [ET]
(1995) *What is life?* Simon and Schuster. [ET]
Maturana, H. R. & Varela, F. J. (1973) *De máquinas y seres vivos: Una teoría de la organización biológica.* Editorial Universitaria. [ET]
(1980) *Autopoiesis and cognition: The realization of the living.* Boston Studies in the Philosophy of Science, vol. 42. D. Reidel. [arSR, AAS, ET]
(1987) *The tree of knowledge: The biological roots of human understanding.* Shambala/New Science Library. [ET]
Maxson, S. C. (1996) Issues in the search for candidate genes in mice as potential animal models of human aggression. In: *Genetics of criminal and antisocial behaviour,* ed. G. R. Bock & J. A. Goode. Wiley. [SCM]
Maynard Smith, J. (1997) Commentary. In: *Feminism and evolutionary biology,* ed. P. A. Gowaty. Chapman and Hall. [JA]
Maynard Smith, J. & Szathmary, E. (1995) *The major transitions in evolution.* Freeman. [BGC, rSR]
McGue, M., Bacon, S. & Lykken, D. (1993) Personality stability and change in early adulthood: A behavior genetic analysis. *Developmental Psychology* 29:96–109. [RAD]
McHugh, P. & Slavney, P. (1983) *The perspectives of psychiatry.* Johns Hopkins University Press. [DJS]
Michel, G. F. & Moore, C. L. (1995) *Developmental psychobiology: An interdisciplinary science.* MIT Press. [GFM]
Midgley, M. (1998) One world, but a big one. In: *Brains to consciousness? Essays on the new sciences of the mind,* ed. S. Rose. Allen Lane. [rSR]
Mileusnic, R., Lancashire, C. & Rose, S. P. R. (1999) Antibodies and antisense to the amyloid precursor protein prevent memory consolidation: RERMS peptide rescues. (submitted). [rSR]
Miller, R. (1995) *Arguments against secular culture.* SCM Press. [GFM]
Moore, C. L. (1992) The role of maternal stimulation in the development of sexual behavior and its neural basis. *Annals of the New York Academy of Sciences* 662:160–77. [GFM]
Murdock, G. P. (1945) The common denominator of culture. In: *The science of man in the world crisis,* ed. R. Linton. Columbia University Press. [GJB]
Nagel, T. (1998) Reductionism and antireductionism. In: *Novartis Symposium: The limits of reductionism in biology.* Wiley. [rSR]
Nobel Foundation (1962) *Nobel: The man and his prizes,* ed. H. Schück, R. Sohlman, A. Österling, G. Liljestrand, A. Westgren, M. Siegbahn, A. Schou & N. K. Ståhle. Elsevier. [JH]
Norman, D. A. (1993) Cognition in the head and in the world: An introduction to the special issue on situated action. *Cognitive Science* 17:1–6. [DJS]
Oyama, S. (1986) *The ontogeny of information.* Cambridge University Press. [rSR]
Petrie, M., Halliday, T. & Sanders, C. (1991) Peahens prefer peacocks with elaborate trains. *Animal Behaviour* 41:323–31. [TER]
Phelps, J. A., Davis, J. O. & Schwartz, K. M. (1997) Nature, nurture, and twin research strategies. *Current Directions in Psychological Science* 6:117–21. [SJS]
Pinker, S. (1995) *The language instinct.* Allen Lane/Penguin Press. [rSR]
(1997) *How the mind works.* Allen Lane. [rSR]
Pinto-Correia, C. (1997) *The ovary of Eve: Egg and sperm and preformation.* University of Chicago Press. [rSR]
Platt, S. A. & Sanislow, C. A., III. (1988) Norm-of-reaction: Definition and misinterpretation of animal research. *Journal of Comparative Psychology* 102:254–61. [JH]
Plomin, R. & Daniels, D. (1987) Why are children in the same family so different from one another? *Behavioral and Brain Sciences* 10:1–60. [HDS]
Plomin, R., DeFries, J. C., McClearn, G. E. & Rutter, M. (1997) *Behavioral genetics. Third edition.* W. H. Freeman. [SCM]
Polak, M. & Trivers, R. (1994) The science of symmetry in biology. *TREE* 9(4):122–24. [HDS]
Popper, K. (1972) Of clocks and clouds. In: *Objective knowledge: An evolutionary approach.* Oxford University Press. [MM]
Portin, P. & Alanen, Y. O. (1997) A critical review of genetic studies of schizophrenia. I. Epidemiological and brain studies. *Acta Psychiatrica Scandinavica* 95:1–5. [DJS]
Provine, W. B. (1971) *The origins of theoretical population genetics.* University of Chicago Press. [SCM]
Raine, A. (1993) *The psychopathology of crime: Criminal behavior as a clinical disorder.* Academic Press. [DJS]
Ramskold, L. (1992) The second leg row of *Hallucigenia* discovered. *Lethaia* 25:221–24. [AAS]
Richardson, K. (1998) *The origins of human potential: Evolution, development and psychology.* Routledge. [rSR]
(1999) *The making of intelligence.* Weidenfeld and Nicholson. [rSR]
Ridley, M. (1997) *The origin of virtue.* Penguin. [rSR]
Rose, H. & Rose, S., eds. (2000) *Coming to life.* Crown/Random House. (forthcoming). [rSR]
Rose, S. (1991) *The chemistry of life* (1st edition, 1966). Penguin. [rSR]
(1992) *The making of memory.* Bantam. [arSR]
(1997) *Lifelines: Biology beyond determinism.* Oxford University Press. Also published as (1997) *Lifelines: Biology, freedom, determinism.* Penguin. [aSR]
(1999) Review of E. O. Wilson's *Consilience. Endeavour.* (in press). [rSR]
Rose, S., Lewontin, R. C. & Kamin, L. (1984) *Not in our genes.* Penguin. [JCL, rSR]
Roubertoux, P. L. & Capron, C. (1990) Are intelligence differences hereditarily transmitted? *Cahiers de Psychologie Cognitive/European Bulletin of Cognitive Psychology* 10:555–94. [WEC]
Roubertoux, P. L., Carlier, M., Degrelle, H., Haas-Dupertuis, M. C., Phillips, J. & Moutier, R. (1994) Co-segregation of intermale aggression with the pseudoautosomal region of the Y chromosome in mice. *Genetics* 136:225–30. [WEC]
Rowe, D. C. (1994) *The limits of family influence: Genes, experience, and behavior.* Guilford Press. [SCM]
Ryle, G. (1949) *The concept of mind.* Barnes and Noble. [WJF]
Sapp, I. (1994) *Evolution by association.* Oxford University Press. [NST]
Scarr, S. (1992) Developmental theories for the 1990s: Development and individual differences. *Child Development* 63:1–19. [MA]
Schaffner, K. F. (1998) Genes, behavior, and developmental emergentism: One process, indivisible? *Philosophy of Science* 65:209–52. [DJS]
Segal, N. L., Dysken, M. W., Bouchard, T. J., Jr., Pedersen, N. L., Ekert, E. D. & Heston, L. L. (1990) Tourette's disorder in a set of reared apart triplets: Genetic and environmental influences. *American Journal of Psychiatry* 147:196–99. [SCM]
Shaywitz, B. A., Fletcher, J. M. & Shaywitz, S. E. (1997) Attention-deficit/hyperactivity disorder. *Advances in Pedriatrics* 44:331–67. [DJS]
Shostak, M. (1981) *Nisa.* Harvard University Press. [GJB]
Singh, S. (1998) *Fermat's last theorem.* Fourth Estate. [AC-B]
Skinner, B. F. (1950) Are theories of learning necessary? *Psychological Review* 57:193–216. [JCL]
(1969) *Contingencies of reinforcement: A theoretical analysis.* Prentice-Hall. [JCL]
(1972) *Beyond freedom and dignity.* Bantam. [JCL]
Smith, L. B. & Tsimpli, I.-M. (1995) *The mind of a savant: Language learning and modularity.* Blackwell. [MA, rSR]
Sober, E. & Wilson, D. S. (1998) *Unto others: The evolution and psychology of unselfish behavior.* Harvard University Press. [rSR, NST]
Sokol, D. K., Moore, C. A., Rose, R. J., Williams, C. J., Reed, T. & Christian, J. C. (1995) Intra-pair differences in personality and cognitive ability among young monozygotic twins distinguished by chorion type. *Behavior Genetics* 25:457–66. [SJS]
Stamps, R. & Gurling, H. M. D. (1998) Identification of a British cohort of patients with a phenotype similar to that found to be associated with a premature stop codon in the monoamine oxidase A (MAOA) gene. *American Journal of Medical Genetics, Neuropsychiatric Genetics* 81:467–68. [WEC]
Stein, D. J. (1991) Philosophy and the DSM-III. *Comprehensive Psychiatry* 32:404–15. [DJS]
Sternberg, R. J. & Grigorenko, E. L., eds. (1977) *Intelligence, heredity, and environment.* Cambridge University Press. [CM]
Szathmary, E. & Maynard Smith, J. (1995) The major evolutionary transitions. *Nature* 374:227–32. [BGC]
Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K. J., Segal, N. L. & Rich, S. (1988) Personality similarity in twins reared apart and together. *Journal of Personality and Social Psychology* 54:1031–39. [RAD]
Ten Cate, C. (1994) Perceptual mechanisms in imprinting and song learning. In: *Causal mechanisms of behavioral development,* ed. J. A. Hogan & J. J. Bolhuis. Cambridge University Press. [GFM]
Thompson, E. (1995) *Colour vision: A study in cognitive science and the philosophy of perception.* Routledge. [ET]
(1997) Symbol grounding: A bridge from artificial life to artificial intelligence. *Brain and Cognition* 34:48–71. [ET]
Thompson, E., Palacios, A. & Varela, F. J. (1992) Ways of coloring: Comparative color vision as a case study for cognitive science. *Behavioral and Brain Sciences* 15:1–74. [ET]
Thompson, N. (1998) Some confusions at the core of evolutionary psychology's

https://doi.org/10.1017/S0140525X99442207 Published online by Cambridge University Press

resistance to multi-level selection theory. Talk given at the Human Behavior and Evolution Society in Davis, California. [NST]
Thompson, N. & Derr, P. G. (1995) On the use of mental terms in behavioral ecology and sociobiology. *Behavior and Philosophy* 23:31–37. [NST]
Trivers, R. (1971) The evolution of reciprocal altruism. *Quarterly Review of Biology* 4:35–37. [aSR]
Varela, F. J. (1979) *Principles of biological autonomy.* Elsevier/North Holland. [ET]
(1991) Organism: A meshwork of selfless selves. In: *Organism and origins of self,* ed. A. I. Tauber. Kluwer. [ET]
(1996) The early days of autopoiesis: Heinz and Chile. *Systems Research* 13:407–16. [ET]
(1997) Patterns of life: Intertwining identity and cognition. *Brain and Cognition* 34:72–87. [ET]
Varela, F. J. & Bourgine, P., eds. (1992) *Towards a practice of autonomous systems. Proceedings of the First European Conference on Artificial Life, Cambridge, Mass.* MIT Press/A Bradford Book. [ET]
Varela, F. J. & Coutinho, A. (1991) Second generation immune networks. *Immunology Today* 12:159–66. [ET]
Varela, F. J., Maturana, H. R. & Uribe, R. (1974) Autopoiesis: The organization of living systems, its characterization and a model. *Biosystems* 5:187–96. [ET]
Varela, F. J., Thompson, E. & Rosch, E. (1991) *The embodied mind: Cognitive science and human experience.* MIT Press. [ET]
Verrall, R. (1980) Sociobiology says racism is in our selfish genes. *The New Nation* 1, 2 (summer/autumn). [rSR]
Waldrop, M. M. (1993) *Complexity: The emerging science at the edge of order and chaos.* Touchstone Books. [NST]
Webster, G. & Goodwin, B. (1997) *Form and transformation: Generative and relational principles in biology.* Cambridge University Press. [rSR]
Weinberger, D. R. (1996) On the plausibility of the "neurodevelopmental" hypothesis of schizophrenia. *Neuropsychopharmacology* 14:1S-11S. [DJS]
Wilson, D. & Sober, E. (1994) Reintroducing group selection to the human behavioral sciences. *Behavioral and Brain Sciences* 17:585–654. [NST]
Wilson, E. O. (1975) *Sociobiology: The new synthesis.* Bellknap Press/Harvard University Press. [SCM, arSR]
(1978) *On human nature.* Harvard University Press. [SCM]
(1998) *Consilience: The unity of knowledge.* Little, Brown. [JH, rSR]
World Health Organization (1992) *The ICD-10 classification of mental and behavioural disorders. Clinical description and diagnostic guidelines.* World Health Organizaiton. [DJS]
Wright, R. (1994) *The moral animal: Why we are the way we are: The new science of evolutionary psychology.* Pantheon. [rSR]
Wright, S. (1968) *Evolution and the genetics of populations: Vol. 1. Genetic and biometric foundations.* The University of Chicago Press. [SCM]
Wright, W. (1998) *Born that way: Genes, behavior, and personality.* Knopf. [SCM]
Wynne-Edwards, V. C. (1962) *Animal dispersion in relation to social behavior.* Oliver and Boyd. [NST]
Yehuda, R. & McFarlane, A. (1997) Psychobiology of post-traumatic stress disorder. *Annals of the New York Academicy of Sciences, vol. 821.* [RAD]
Zuckerman, M. (1984) Sensation seeking: A comparative approach to a human trait. *Behavioral and Brain Sciences* 7:413–71. [HDS]

https://doi.org/10.1017/S0140525X99442207 Published online by Cambridge University Press

https://doi.org/10.1017/S0140525X99442207 Published online by Cambridge University Press