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Extended Phenotypes and Extended Organisms

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Abstract. Phenotype, whether conventional or extended, is defined as a reflection of an underlying genotype. Adaptation and the natural selection that follows from it depends upon a progressively harmonious fit between phenotype and environment. There is in Richard Dawkins' notion of the extended phenotype a paradox that seems to undercut conventional views of adaptation, natural selection and adaptation. In a nutshell, if the phenotype includes an organism's environment, how then can the organism adapt to itself? The paradox is resolvable through a physiological, as opposed to a genetic, theory of natural selection and adaptation.

Oh! let us never, never doubt
What nobody is sure about![1]

Introduction

The twentieth century was not, for biologists at least, a doubt-filled century. In one of the most thorough-going intellectual revolutions in history, biologists, along with like-minded allies in chemistry and physics, revealed, among other things: the material basis of heredity; how heritable information is encoded; how genes are replicated; how this information is translated into functional machines; where genetic variation comes from; how genes behave in populations, and how fidelity of genetic replication can be reconciled with genetic variation and natural selection. Like Sherman's triumphant march through Georgia, the new biology was a juggernaut that swept everything before it, leaving the inhabitants stranded in a landscape that was transformed beyond all recognition.

The Extended Phenotype (Dawkins 1982) was very much a product of this era of genetic triumphalism. The book's sub-title says this clearly – *The gene as the unit of selection* – and the book's message is no less clear,

consistent and insistent. What drives evolution is natural selection among replicators, genes essentially. The organisms that make replication possible, the "vehicles" of heredity, no matter how wonderful, no matter how intricately contrived, no matter how subtle or Machiavellian their machinations, all pass away, leaving only the enduring gene to be naturally selected. As Dawkins himself put it, his aim in writing *The Extended Phenotype* was "not to praise the vehicle, but to bury it" (Dawkins 1994), to move it into its proper role in the background so that we might think clearly about how the vehicle came to be.

The end of the twentieth century has not been nearly so kind to the genetic triumphalist. As we have come to know more about how genes are put together, how genes actually work, how they fit into the "society of proteins" within the cell, how they may be modified by cellular environments, how the coding in genes is transformed into function, the existence of alternate forms of heritable memory like prions, the bright line that divided genes from the vehicles that carry them has dimmed considerably. Consequently, we have come to see how the phenotype is more than simply a reflection of genotype, but rather a thing unto itself, playing by its own rules, even controlling genes to an extent inconceivable in the 1980's. Along with this, we have seen the erosion of many other of the "bright lines" that were drawn through biology during that era. No longer can a reliable distinction be made between genome and organism, between replicator and vehicle, between structure and function.

In retrospect, what makes *The Extended Phenotype* a truly inspired work was not its robust defense of the supremacy of genes in evolution, but how it breached what is probably the brightest of biology's bright lines: the distinction between organism (vehicle, if you wish) and the environment in which it functions. This idea opened up eyes everywhere to an entirely new way of thinking about adaptation and evolution, but it also, I would argue, contained at its heart a contradiction that undermined the very point Richard Dawkins strove so eloquently to make. If the fate of great ideas is to be wrong in a fruitful way, *The Extended Phenotype* stands out as one of biology's greatest ideas.

The contradiction is rooted in the notion that organisms are embedded in environments, to which they and their progeny either adapt or die. In this sense, the environment is like a sieve, through which each generation's multifarious candidates for selection either pass or are blocked: "the organism proposes, and the environment disposes", to quote Stephen Jay Gould (Gould 1989). *The Extended Phenotype* turns this conventional Darwinian dogma on its head. When organisms can modify environments to beneficial ends, they are liberated from being simply slaves at the mercy of the environment, and

become, in a profound sense, its masters. And therein lies the contradiction. If adaptation is the progressively harmonious fit between organism and its environment, and if the environment is part of the organism's phenotype, where does this leave George William's famous assertion of another bit of Darwinist conventional wisdom: "organisms always adapt to their environments, never the other way around" (Williams 1992)? In blurring the bright line that divides organism from environment, Dawkins made it possible to think, even embrace, what was unthinkable to George Williams: that environments can adapt to organisms, that environments can have fitness, and that environments can even evolve.

The blood and guts of the extended phenotype

I am a physiologist, not an evolutionary biologist. Nevertheless, *The Extended Phenotype* was as much of an inspiration to me as it has been to an entire generation of my more evolution-minded colleagues. Physiology, like most of the other biological disciplines, gazed relentlessly inward for most of the twentieth century. *The Extended Phenotype* opened the possibility that interesting physiology could also be found "out there", beyond the organism's skin (Turner 2000a). Interestingly, the notion of an "extended physiology" requires less of a leap of imagination than does the extended phenotype. Indeed, to the physiologist, extended physiology is virtually inevitable. Physiology is fundamentally a science of how matter, energy and information flow through and between organisms, the physical forces driving them and the mechanisms that control or modulate them. There is no reason why such things need be confined to within organisms' bodies. In fact, the laws of thermodynamics and conservation of mass and energy demand that they not be.

The extended phenotype's physiological inevitability

Conservation of mass and energy ensures that an organism will inevitably affect the environment that surrounds it. Consider a closed system consisting of two environments, one living, and one supposedly inanimate, separated by a boundary (Figure 1). The two environments could be, say, a cell interior and extracellular fluid separated by a cell membrane, or the interior of a body segregated by an integument from its surroundings. Living environments are divided from inanimate surroundings by so-called adaptive boundaries that manipulate flows of mass and energy across them. Generally, adaptive boundaries manage two types of flux. Thermodynamically favored fluxes (TFFs) are driven by gradients in potential energy (pressure, temperature, concentration, voltage) across the membrane, and these are mediated by devices embedded

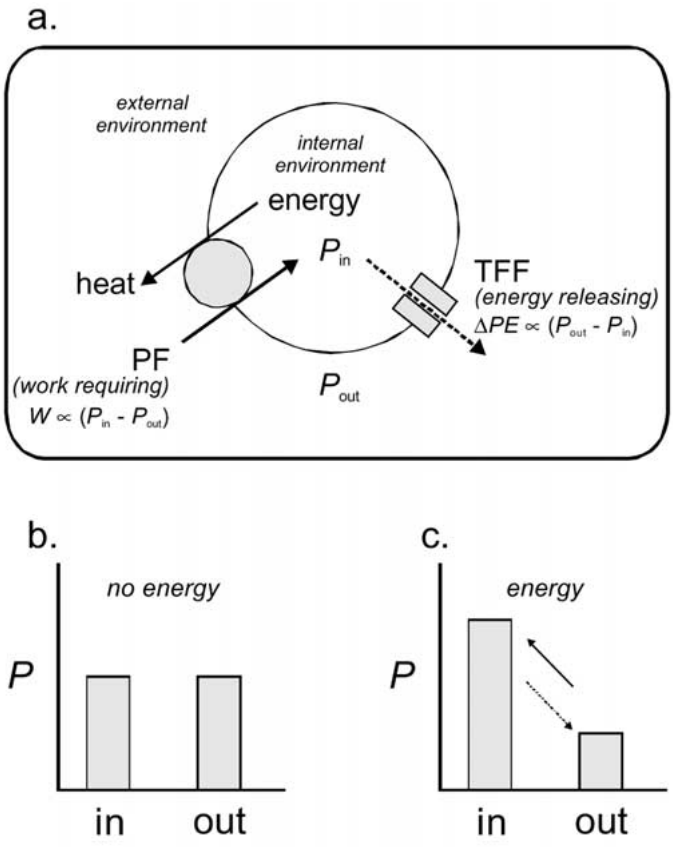

Figure 1. A simple physiological model of an organism, consisting of an internal environment separated from an external environment by a boundary. *a*. Embedded within the boundary are channels that permit thermodynamically favored fluxes (TFF, dashed line), driven by differences of potential energy between inside (P_{in}) and outside (P_{out}) the boundary, and physiological machines that use energy to do the work of maintaining a physiological flux (PF, solid line), which maintains the potential energy difference. *b*. Distribution of potential energy, *P*, in the absence of an energy flow through the system. *c*. When the PF is powered, it maintains a persistent PE disequilibrium between organism and environment, maintained by opposing PFs (solid line) and TFFs (dashed line).

in the boundary which facilitate these fluxes. Physiological fluxes (PFs) are driven against these potential energy gradients, and powering them requires work to be done. A living environment is characterized by a disequilibrium, which persists as long as energy to run physiological fluxes is available. However, because mass and energy are conserved, any increase of either on one side of the boundary necessitates its removal from the other. By the very

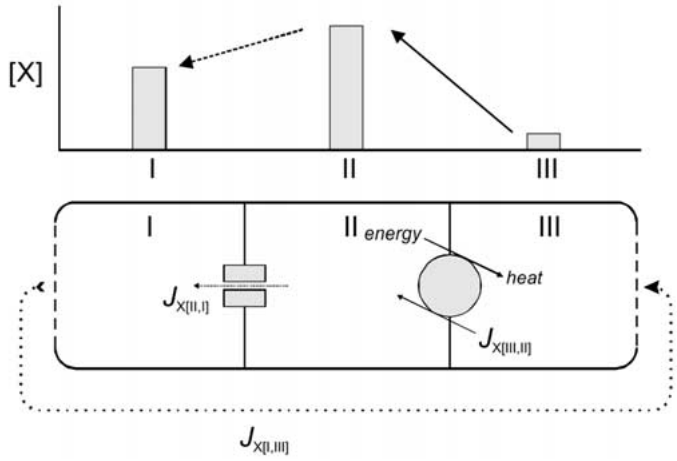

Figure 2. Energetics of a putative open system, consisting of three compartments through which matter can flow driven by energy. The operation of a physiological machine imposes potential energy differences, embodied in differences in [**X**] between compartments. Exchange of matter is ultimately a closed system, ensuring there will be some pathway for return of matter from compartment I to compartment III (indicated by dotted arrow).

act of living, organisms alter the environments in which they live. Extended physiology is inevitable.

Does this make extended phenotypes inevitable? I believe it does, but three likely objections come to mind. These happen to be incorrect, but why they are will be important for the arguments to follow.

The first objects that living systems are not closed but open, with different rules and patterns of behavior. Assume a putative open living system through which matter and energy flow (Figure 2). Activating physiological fluxes puts the system into a dynamic disequilibrium, accompanied by a change of property in all environments. The putatively open system of Figure 2 differs from the "closed" system of Figure 1 only in the number of compartments – it does not nullify the inevitability of an extended physiology.

The second objection, which might be dubbed the fly-in-the-soup problem,[2] goes something like this. Conservation of mass or energy surely applies, but the capacities of environmental sources and sinks are typically so vast that any physiological draw-downs or build-ups by organisms will affect these environments only negligibly. For example, how much will the respiration of, say, a fish, alter the oxygen concentration of, say, Lake Ontario? The fly-in-the-soup objection is a red herring, if I may mix metaphors. Environments are not always capacious, and there are many instances where physiological activity indeed has significant effects on them, as in the environment of a burrow inhabited by a respiring rodent. Furthermore, even

capacious environments can be filled by expanding populations of organisms, as in the physical properties and physiological functions in tracts of soil.

The third, and more subtle, problem is in the nature and origin of the sources and sinks in any putatively "open" system. The biosphere is open only with respect to energy: with respect to matter, the biosphere is essentially closed (Barlow and Volk 1990). Physiology carried out in a materially finite and closed Earth means any sources and sinks must be linked somehow in an ultimately closed loop, by some physical process, or mediated by systems of living environments (Figure 2). Thermodynamically, these complexities generalize to the simple model outlined in Figure 1.

Homeostasis

Replication of replicators, whatever those might be, requires matter to be supplied and organized in a particular way, powered by energy channeled through physiological machines. Those organisms that "work well" will produce more copies of their replicators than those that work less well. Darwinism is indifferent to, or at least agnostic on, how good function works: it is sufficient that good function is achievable. Nevertheless, distinguishing good function from poor requires attention be paid to three important principles. One, conservation of energy and mass, has already been mentioned. Another, the second law of thermodynamics, dictates that orderliness is transient, and that work must be done to maintain it. The last is the phenomenon of homeostasis, which is a fundamental property of any living thing.

Homeostasis is often misunderstood to mean precisely the word's literal translation, steadiness of some property of a system, like body temperature, acidity of the blood or the voltage inside a cell. Homeostasis cannot be so narrowly defined, however. A property can be steady through time without homeostasis being at work, as in the steady temperatures of deep soil horizons, abyssal oceans, or the interiors of massive rocks. Nor does homeostasis necessarily indicate steadiness of a particular property. Mammals and birds, for example, regulate their body temperatures more precisely than, say, barnacles. One cannot draw from this, however, the essentially chauvinist conclusion that homeostasis among mammals and birds is more highly-developed than in barnacles. Barnacles live in environments which impose different physiological demands than those in which mammals and birds live. Yet, they maintain function, just as mammals and birds do. This persistence of function is the essence of homeostasis. All organisms exhibit homeostasis in one form or another.

Homeostasis, like extended physiology, is a physically-constrained inevitability. Physiological function requires an orderly environment that specifies

particular pathways for flows of mass and energy. Persistent cellular metabolism, for example, requires an elaborately constructed *milieu* of catalytic shapes within the cell, embodied in proteins encoded by genes. The second law of thermodynamics asserts that this orderly environment inexorably degrades, and along with it the physiological function it specifies. Function persists only if work is done to restore this orderliness as rapidly as it degrades. This is homeostasis at its most fundamental level.

The problematic boundary between living organism and inanimate environment

The environments on both sides of an adaptive boundary, like a cell membrane, are linked by conservation of mass and energy. Any flux of mass and energy that mediates homeostasis on one side of the boundary will impose, to a degree, homeostasis on the other. This renders problematic any designation of the environment on one side as "living", while designating the environment on the other as "inanimate". Practically, our designations of living and inanimate environments turn more on what is the predominant driver of the respective mass and energy balances (Figure 3). In a putative inanimate environment, any physiological draw-down or build-up is minuscule compared to fluxes driven by other factors: the fly-in-the-soup condition. Meanwhile, it is the physiological flux that dominates the properties of the putative living environment. If neither dominates, it is harder to distinguish a living environment from a supposedly inanimate one: the distinction is only of degree rather than kind. Any sharp distinction is thermodynamically indefensible.

Organisms comprise numerous environments, organized more-or-less hierarchically, and the question then becomes: which of these are living, and which are inanimate? The cell is an obvious example, an intracellular environment separated from an extracellular environment by the adaptive boundary of the cell membrane. Within the cell's membrane are other environments, enclosed themselves within membrane-bound organelles like mitochondria, chloroplast, endoplasmic reticulum and nucleus. All would agree these comprise living environments, but the organization of environments-within-environments also extends outward, and here is where difficulties emerge. One could, for example, draw a reasonably sharp distinction between, say, a single-celled protist and the environment in which it swims. However, organisms comprise complex environments that are themselves delimited and regulated by adaptive boundaries. Epithelia, for example, divide the body into various compartments, which can include enclosed environments, like coeloms, or infoldings of an organism's "innards", like livers, kidneys, and systems for distribution of blood or hemolymph. Are the cells within these

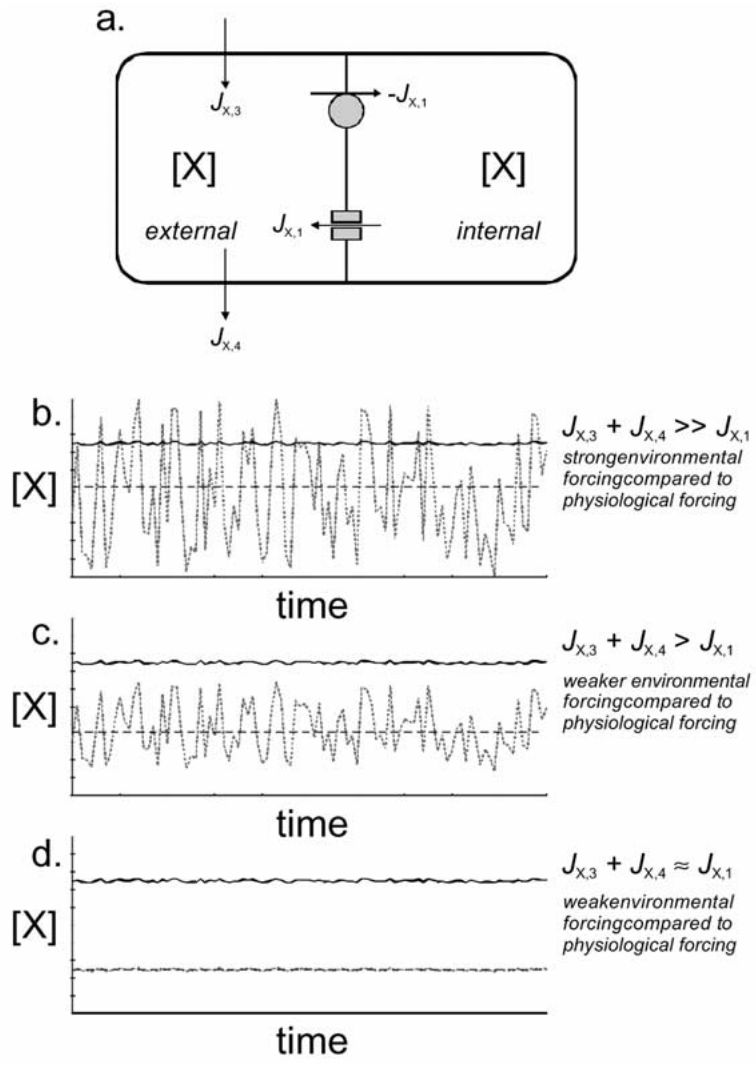

Figure 3. Imposition of homeostasis upon a supposedly inanimate environment. *a.* A simple physiological system with a boundary separating an internal environment from an external environment. Concentrations of **X** in each compartment are determined by balances of fluxes into and out of each compartment. [**X**] in the internal compartment are determined by a balance between a physiological flux ($-J_{X,1}$) and a thermodynamically favored flux ($J_{X,1}$). Homeostasis in the internal environment ensures that PF + TFF = 0. [**X**] in the external environment is determined by the balance of fluxes $J_{X,1}$ and environmental fluxes $J_{X,3}$ and $J_{X,4}$. If the environmental fluxes are much larger than the physiological flux, external [**X**] will be driven by variations in the external flux. If environmental fluxes are small comparted to the physiological flux, external [**X**] will be regulated to the same degree as internal [**X**].

environments, or for that matter a protist swimming through them, inhabiting living environments or inanimate ones? Even infoldings of organisms' outer skins, as in lungs or intestines, create internal regulated environments, even though these are topologically outside the organism. The gas composition of air in the alveolus is as tightly regulated as the blood gases "inside". Is the lung interior one of the organism's living environments? And what of extensions of physiology and homeostasis beyond the organism's skin? Are these inanimate environments, and if so, by what distinction?

Extended phenotype or extended organism?

Let me illustrate these thoughts with one of the more spectacular examples of extended physiology on the planet (Figure 4): the colonies of the mound-building termites of the Macrotermitinae (mostly of the genus *Macrotermes*). The macrotermitines, like other termites, rely on symbiosis with cellulolytic microrganisms, cultivated in controlled environments within the intestine to digest their woody food (Martin 1987). Unlike other termites, the macrotermitines have "outsourced" cellulose digestion to fungi, cultivated on elaborately folded structures, called fungus combs, constructed by the termites from macerated wood brought back to the nest by foragers. A variety of fungal spores are mixed with the macerated forage as it passes through the workers' intestines, inoculating the comb. Once in the nest, spores of only one type, of the basidiomycete genus *Termitomyces*, germinate and spread hyphae throughout the comb (Thomas 1987a, b). The fungi digest the macerated forage to a variety of simpler sugars, which the termites then reconsume and digest.

Macrotermes and *Termitomyces* together make a metabolically effervescent combination. Outsourcing cellulose digestion liberates *Macrotermes* from constraints on digestion rate that face termites that rely solely on intestinal digestion (Penry and Jumars 1986). *Macrotermes* and *Termitomyces* also each bring different cellulases and lignases to the chore, and these together digest cellulose faster than the enzymes of each alone (Martin 1987; Rouland and Civas et al. 1988). Consequently, *Macrotermes-Termitomyces* colonies are able to liberate energy from cellulose at much faster rates than other termites, which influences all aspects of their biology and life history. For example, macrotermite colonies have larger collective biomass than other termites (Darlington and Dransfield 1987; Darlington 1990, 1991), both through larger body sizes (roughly three times larger than other types of termite), and very populous colonies (on the order of millions of individuals, as opposed to the tens of thousands or hundreds of thousands found in nests of other termites). Macrotermites' high rates of energy liberation confer upon

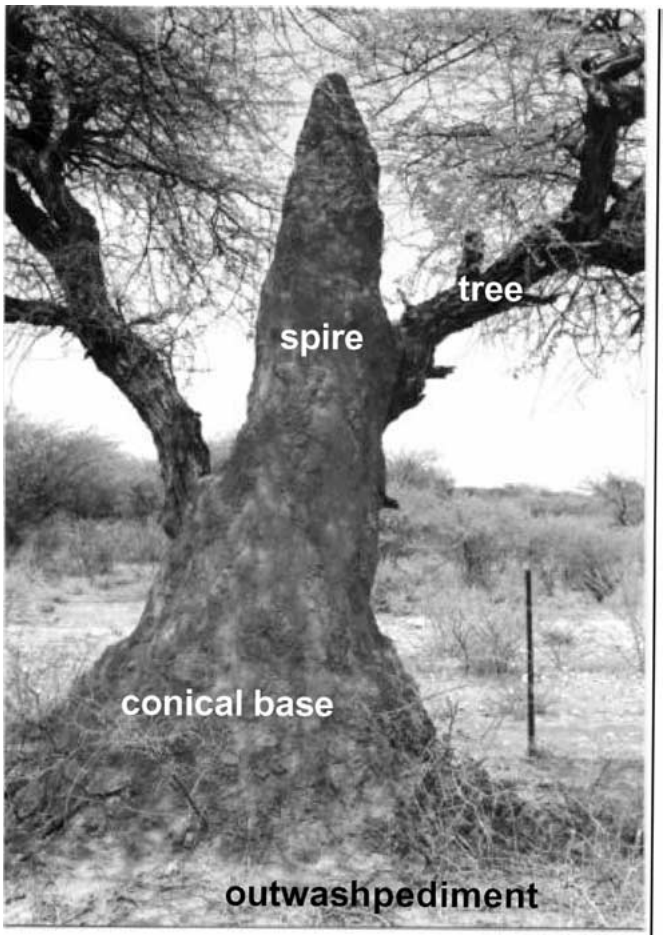

Figure 4. A mound of *Macroterme michaelseni* in northern Namibia.

their colonies impressive capabilities for doing the work of environmental engineering (Dangerfield and McCarthy et al. 1998), manifest in their most visible attribute, the massive mounds that dot the landscapes of sub-Saharan Africa at densities of roughly 1–4 colonies per hectare.

The mound constitutes an impressive engineering project, yet the termites do not live in it (Figure 5). The queen and king, the sterile workers, fertile alates and fungal gardens are housed in a subterranean nest just below the mound, a roughly spherical structure about 1.5–2 meters in diameter. The mound itself, and soils immediately surrounding the nest, contains an elaborately constructed network of tunnels which serve as conduits for flows of air (Turner 2000b). These flows are driven both by the heat produced by the colony's considerable metabolism, estimated to be 50–250 watts (Darlington

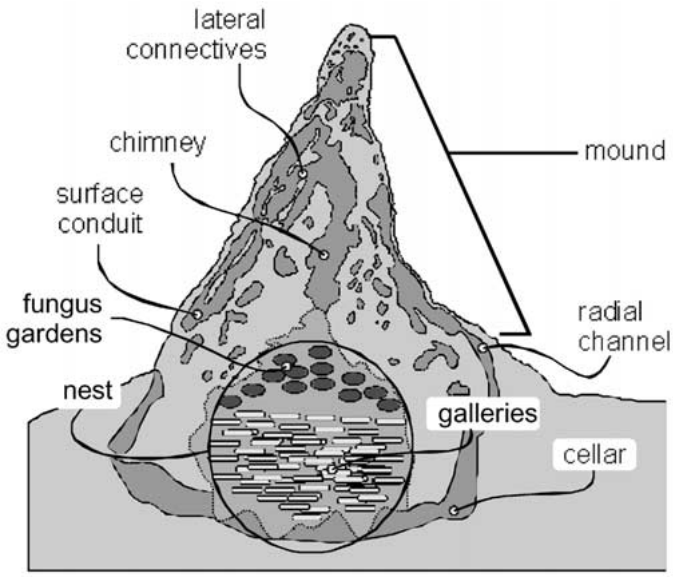

Figure 5. A representative cross section through a mound of a *Macrotermes michaelseni* nest, showing respective position of nest, fungus gardens and the tunnel network in the mound and surrounding soils. After Turner (2000a).

and Zimmerman et al. 1997), and by the capture of wind energy by the tall mound (Turner 2001). The combination of the two produces a vigorous ventilation of the air spaces around the nest: 95% of the nest air exchanges with the atmosphere roughly once every twenty minutes. The nest environment also appears to be regulated. Oxygen partial pressures in the nest are maintained at roughly 1–2 kilopascals below the atmosphere, despite substantial variation in respiratory demand for oxygen (Turner 2001).

The nest atmosphere can be regulated because the mound is a dynamic structure. Roughly a cubic meter of soil cycles through the mound each year, eroded soil being replaced by new soil brought up into the mound by the termites (Pomeroy 1976). Consequently, the mound's architecture can change through time, and the changes are directed, in part, by how well ventilation is matched to respiration. Take, as an example, the regulation of oxygen partial pressure (pO_2) in the nest (Figure 6). Nest pO_2 is the balance between consumption rate by the colony ($J_{M,O2}$) and its rate of replacement by ventilation ($J_{V,O2}$). Any perturbation of the nest environment indicates a mismatch between the two fluxes, which the termites resolve by modifying the architecture of the mound. For example, an inordinately low nest pO_2 signifies that ventilation is not keeping up with respiration. This is rectified by termites opening up new pores in the surface, extending the mound upwards into more

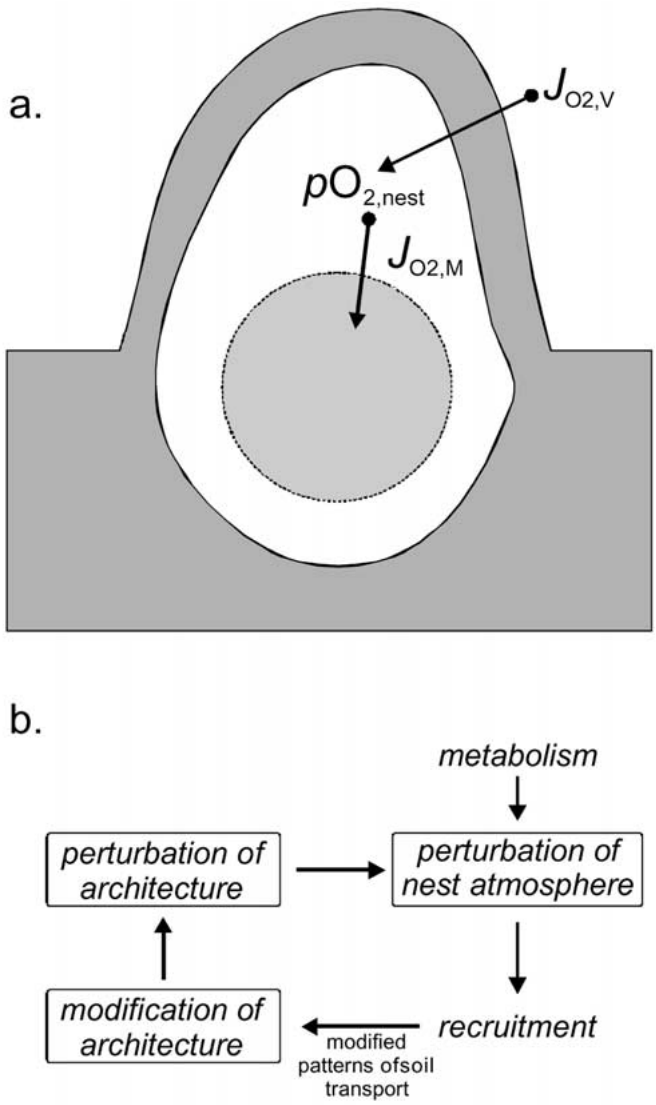

Figure 6. Homeostasis of the nest environment in a *Macrotermes* colony. *a*. Maintenance of oxygen partial pressure ($pO_{2,nest}$) as a balance between a metabolic consumption rate ($J_{O2,M}$) and a ventilation rate ($J_{O2,V}$). *b*. General scheme of relationship between mound morphogenesis and homeostasis of the nest atmosphere.

energetic winds, etc. Thus, the mound is an adaptive structure, similar in principle to the adaptive boundaries that separate cells or organisms or any of a number of other living environments from their surroundings (Turner 2000a). Is the nest environment, therefore, a living environment? Clearly, it is, maintained by an organ of extended physiology, the mound.

Is the mound, therefore, an extended phenotype, that is a product of genes that control the processes whereby termites build mounds? Certainly, it could be, but a closer look at the nest's physiology muddies the picture a bit. It is a bit of a misnomer to call the nest and mound a termite colony. The fungi account for roughly 85% of the colony's collective metabolism, which makes the fungi, not the termites, the major perturber of the nest environment (Darlington and Zimmerman et al. 1997). Nevertheless, the burden of regulating the nest atmosphere is left solely to the termites. The question thus arises: for whose benefit is the work of homeostasis being done, the termites or the fungi? There is clearly a very finely balanced mutualism at work. The fungi benefit for obvious reasons – termites are mobile organisms with sensory systems to seek out food, collect it and gather it in a massive cache in the nest. Termites benefit for other obvious reasons – by using fungi to carry out digestion, they can mobilize energy at much higher rates than their competitors.

It is not public-spiritidness that drives the evolution of this mutualism, however. The nest's cache of cellulose is a prize for any fungus that can exploit it, and the potential competitors are numerous and diverse, evidenced by the presence of roughly two dozen other types of fungal spores in the nest, fungus combs on and within the termites' bodies. *Termitomyces* always prevails, though, albeit not from any inherent competitive advantage (Thomas 1987c). For one thing, *Termitomyces* is slow-growing compared to other fungi, presumably because it takes up its extracellularly-digested food slower than other, faster-growing fungi would. This is what makes *Termitomyces* a desirable partner for the termites – slow rate of fungal uptake of digested sugars means more is available for consumption by the termites. However, it also ensures that *Termitomyces* would lose in any head-to-head (if I may use the simile) contest with faster growing, greedier fungi that take up their nutrients more quickly. What gives *Termitomyces* the upper hand (another poor simile) over its fungal competitors, rather, appears to be some quality of the nest environment, provided them courtesy of the termites. Within the nest, the spores of all of *Termitomyces*' potential rivals remain dormant (Thomas 1987c). A fungus comb removed from the nest, however, is quickly taken over by an aggressive fungus, *Xylaria*, which germinates and quickly overwhelms the comb's population of *Termitomyces*. This is the outcome even if worker termites are allowed to manage the removed comb. What suppresses

the growth of these other spores is unknown, but an intriguing hypothesis suggests it is the very properties of the nest that are regulated by the termites (Batra and Batra 1966). *Termitomyces* does well in high-CO_2 environments and the acid conditions that go along with this, while *Xylaria* is less tolerant. This raises the intriguing hypothesis that it is the fungi that are cultivating the termites, using their tendency to build regulated environments as a way to suppress the growth of *Termitomyces*' fungal competitors.

Now, precisely whose extended phenotype is the mound? Is it the termites' that build it, or is it the fungi's that perturb the termites' home? The imprecision, is, of course, one of Richard Dawkins' principal points – that it is futile to point to any organism as being the object of selection. Rather, what is being selected is a massive coalition of genes distributed amongst two organisms, *Macrotermes* and *Termitomyces*. The coalition is maintained by punishment of any gene that seeks to break away. For example, one can easily envision the emergence of "greedy" strains of *Termitomyces* that have elevated uptake rates that let them keep more of the cellulose digestate for themselves. Greedy *Termitomyces* will ultimately be counter-productive, though, choking off energy to the termites that convey cellulose to the fungi. Similarly, any termite genes that seek to liberate themselves from their fungal symbionts will find themselves literally eating wood and straw rather than the rich compost provided by the fungi.

But I want to ask a broader question. Does the notion of extended phenotype really encompass the essence of the *Macrotermes-Termitomyces* system? As a physiologist, I look at this system and see a massive conspiracy (in the literal sense of the word) of living environments, driven by a powerful agent of homeostasis which extends physiology well beyond the bounds of the organisms themselves. In this sense, the colony and mound is not really a phenotype – a reflection of genes that encode the building of a particular type of mound. Rather, the colony and mound constitute an extended organism, with all the attributes – integrity, persistence, homeostasis – the designation implies (Turner 2000a). What makes the *Macrotermes* mound distinctive is the termites' homeostalic response to the extraordinary metabolic perturbation imposed by the adopted *Termitomyces*.

Furthermore, the *Macrotermes-Termitomyces* assemblage has evolved not so much as a selection between alternate genomes, but between alternate extended physiologies – a "closed loop" pathway involving *Macrotermes* $\rightarrow$ *Termitomyces* $\rightarrow$ *Macrotermes* $\rightarrow$ waste, and an "open loop" pathway involving *Macrotermes* $\rightarrow$ *Xylaria* $\rightarrow$ waste. The first is self-sustaining compared to the latter, and so prevails. Key to the one prevailing and not the other is the homeostasis of the nest environment wrought by *Macrotermes*,

the restraint on nutrient uptake by *Termitomyces*, the complementarity of cellulose digestion, etc.

Do only the genotypes of extended phenotypes evolve?

Viewing the *Macrotermes-Termitomyces* partnership as an extended organism exemplifies a physiological perspective on extended phenotypes. It does not negate Richard Dawkins' principal point in *The Extended Phenotype*: that genes are the ultimate units of natural selection. Substitute the unwieldy phrase "superorganismal coalitions of genes" for the succinct "gene" in *The Extended Phenotype*'s subtitle, and the *Macrotermes-Termitomyces* partnership is a conventional, if sophisticated, extended phenotype.

Phenotypes evolve, though, and the question arises: do extended phenotypes evolve in any way differently than conventional ones do, that is as something other than assemblages of genes that are naturally selected? If, for the sake of discussion, we are to allow the possibility that they do, then our ideas of fitness and natural selection may differ depending upon whether the evolving phenotype is conventional or extended. On the other hand, phenotypes, whether they be conventional or extended, have to function well if they are to evolve at all, and the extended physiology that underlies extended organisms presents a common ground for analyzing the evolution of any kind of phenotype we might imagine.

Environments can live because flows of matter and energy are channeled through them in highly specific ways, accomplished by a complex structured environment. We designate this structured environment as a collection of *specifiers* (Figure 7). Within cells, genes clearly determine function through their ability to encode catalytic shapes in proteins, but many other molecules in the cell, including various lipids, RNA molecules and even snippets of DNA also can play catalytic roles. Specifiers can also be influenced by external environmental conditions, in which case they are epigenetic influences.

Living environments can evolve when they meet two criteria: function is heritable, so that good function persists, and function is variable, so that natural selection can occur. Phenotypes are, in a sense, devices created by living environments to minimize the epigenetic effects on function, making it more a reflection of what is encoded in genes than it otherwise would be. This is true for both conventional and extended phenotypes. If a termite mound can be thought of as a device for providing a comfortable environment for the termites and their fungal guests, can one not also think of, say, a circulatory system as the mitochondrion's way of ensuring itself a comfortable environment?

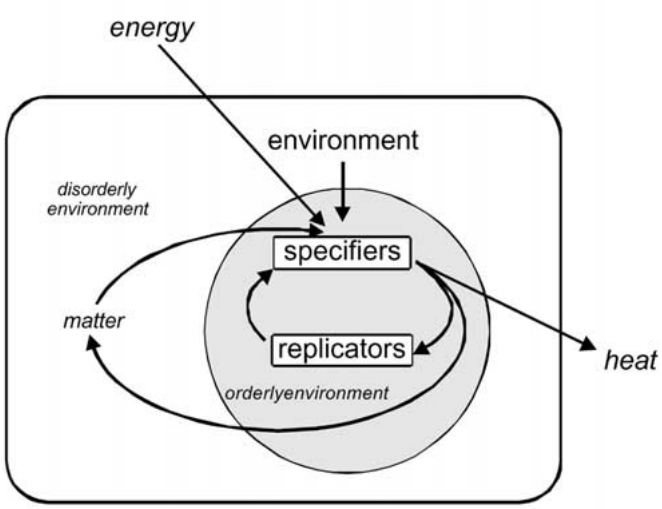

Figure 7. A general scheme of a living environment. Specifiers are the catalytic surfaces that specify particular types of chemical reactions. These can be affected both by translated information in replicators (genes) and by environmental conditions. The specifiers are maintained by a balance between structural degradation and structural regeneration, powered by coupled fluxes of matter and energy through the living environment.

Obviously, genes admirably meet the two criteria for evolvability. In 1982, however, genes' had a much stronger claim for being ultimate determiners of function than they now do. Then, genes' capabilities were defined by the central dogma, which asserted that function radiated like light from the central sun of DNA. As we have come to know more about how cells actually work, we are now coming to see the central dogma as fallacious. Cells' astonishing ability to adapt to varying environmental conditions can only be partly ascribed to "hard-wired" information in genotypes. Metastable proteins, like prions and prion-like proteins, embody a sort of heritable memory that can, as in the prion-like transcription factors of yeast, even specify the nature of genes (Prusiner 1998; Wickner and Taylor et al. 1999; True and Lindquist 2000). Metastable RNAs work in similar ways, behaving as sensors of environmental conditions and catalytic switches of function.

Although most would agree that the central dogma is essentially dead, genes nevertheless have a special role to play. The appropriate questions to ask are: just what is it that enables genes to be arbiters of evolution, and do these capabilities appear in other forms in living environments, be they conventional or extended? Here is my answer. The gene's special nature derives not from its ability to encode function, or to replicate, or to accumulate mutations, but from its *longevity* as a determinant of future functional

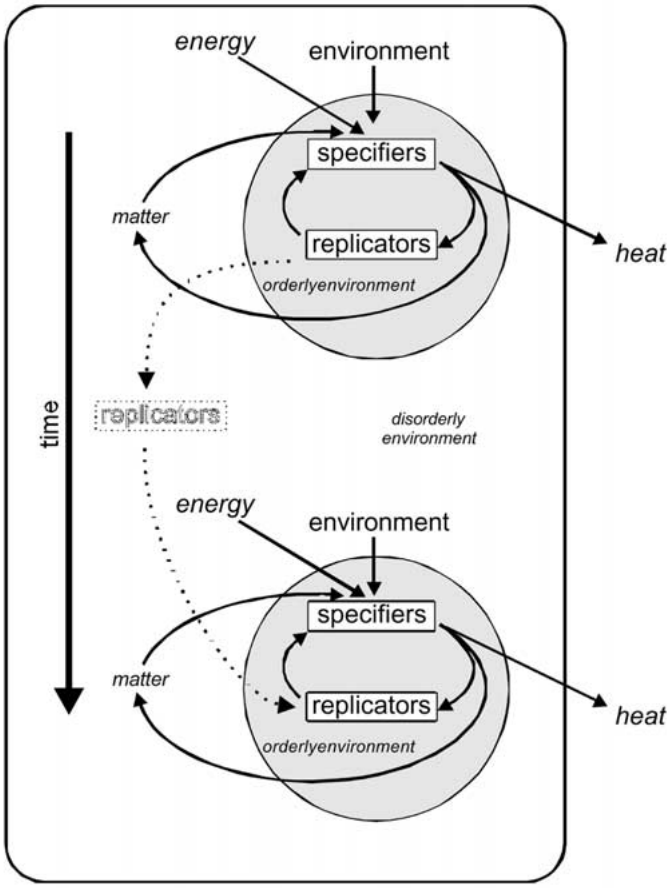

Figure 8. The conventional mode of replicator-based propagation. Replication of a particular living environment involves primarily the transmission of long-live replicators through time.

environments. Put simply, of all the multifarious influences that could be brought to bear on the specifiers of a living environment, the information encoded in genes simply outlasts any others (Figure 8). Specifiers and epigenetic effects on them come and go. Genes endure and evolve.

Not always, though. What if the tables were turned and the specifiers, or what influences them, outlasted the genes (Figure 9)? Now, natural selection would be more among alternate specified environments than among alternate genotypes. The inheritance of acquired modifications of ciliary patterns on ciliates provides a well-known example. These creatures have characteristic patterns of cilia, which can be altered by injury, difficulties in separation of conjugants, or experimentally. Ciliary patterns are determined by a sort of crystalline growth, in which pre-existing patterns determine future

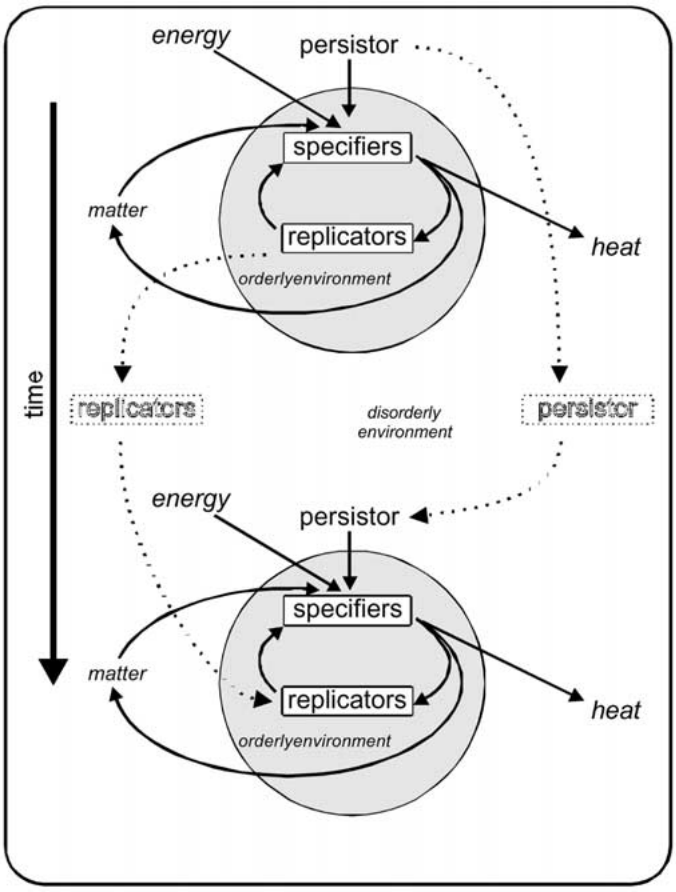

Figure 9. A scheme for persistor-based evolution. If epigenetic environmental conditions endure for long enough, function in future functional environments is determined more by these long-lived effects (designated *persistors*) than by the relatively short-lived replicators.

patterns. In such systems, defects are self-perpetuating. Thus, altered ciliary patterns, and the changes of swimming behavior – phenotype – that follow are transmitted to subsequent generations, even though the genes encoding the component proteins of the cilia are unchanged. Such transmissible structural "mutations" may even determine the longevity of genes (Grandchamp and Beisson 1981).

Because function plays out at a variety of scales and at a variety of rates, the temporal interplay between functional environments and genes can occur at multiple levels of organization and at multiple scales. For example, genes within gametes outlast the bodies that produce them. Yet, the bodies themselves are long-lived functional environments compared to most of the

Figure 10. Heuweltjies (light-colored patches) distributed across a valley floor. Tierberg, near Prince Albert, South Africa.

cells they comprise. What will drive the evolution of lineages of cells within bodies, and will these evolve in similar ways to lineages of the bodies themselves? Looking outward, the extended physiology that attends an extended phenotype may either be very short-lived with respect to the organism that produces it or it may be long-lived. A burrow dug by an animal, for example, has an extended physiology only as long as there is an animal in it: once the burrow is vacated, it becomes simply a hole in the ground. In this case, the extended phenotype is short-lived with respect both to the body that created it, and more to the point, with respect to the replicators that will ensure burrow-digging animals in the future. If, on the other hand, the same burrow was inhabited by many generations of burrow-diggers, the burrow is now a persistent functional environment – a persistor, if you will – that outlasts both the vehicle that dug it and the replicators that produced the burrow-digger.

An interesting candidate for a persistor-driven evolution at work is provided by the southern African harvester termite *Microhodotermes viator*. These insects are widespread through the various Karoo biomes of South Africa, where they produce curious landforms known as *heuweltjies* (pronounced hue'-vull-keys, Afrikaans for "little hills"). As the name suggests, heuweltjies are low mounds, rising typically one to two meters above ground level, and typically twenty to thirty meters in diameter (Lovegrove and Siegfried 1989; Lovegrove 1991). They are distributed across landscapes in a distribution that suggests maximum repulsion between heuweltjies, undoubtedly the result of competition between the termite colonies that produce them (Figure 10). Heuweltjies are visually conspicuous because they support plant communities that differ markedly from those

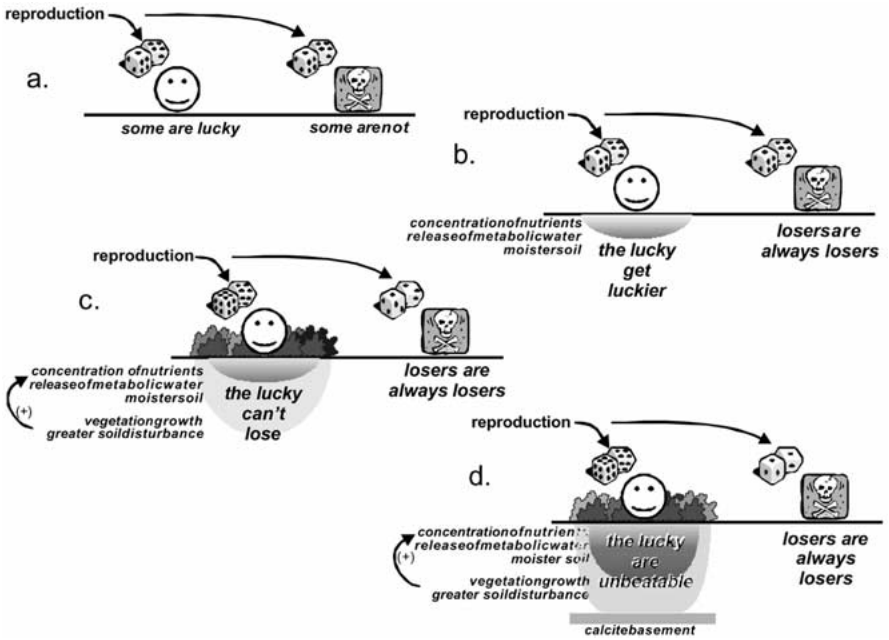

Figure 11. A scheme for evolution of a heuweltjie. Details in text.

on surrounding soils (Knight and Rebelo et al. 1989; Midgley and Musil 1990; Esler and Cowling 1995). Although the plant species composing the heuweltjie communities vary from region to region, the involvement of *Microhodotermes* is common throughout.

Heuweltjies are not simply the sites of an active termite colony, as are the mounds of *Macrotermes*. They are, rather, sites of persistent recolonization by many generations of termites (Lovegrove and Siegfried 1989; Milton and Dean 1990; Lovegrove 1991). Like other termites, *Microhodotermes* colonies reproduce by broadcasting an annual crop of alates that are distributed mostly by winds. There seems not to be any particular tendency for alates to seek out and establish incipient colonies on heuweltjie soils. Rather, recolonization of the heuweltjie is the result of enhanced survivability of any alates that happen to land on a pre-existing structure.

Heuweltjies are favorable environments for termite alates because they mediate an extended physiological function that is crucial to the alates' survival – water balance. Adaptation of termites to the semi-arid environment of the Karoo involves more the evolution of heuweltjies rather than of the termites themselves. The course of a heuweltjie's evolution is instructive (Figure 11). Imagine a pristine uniform environment into which a cohort of *Microhodotermes* propagules are broadcast. Some will found a colony, but

as is typical among termites, most will fail. A surviving natal pair, and the myriad workers it produces, will then set excavating the tunnels and chambers in the nest. This, in turn, increases local soil porosity so that scarce rains that fall there are more likely to infiltrate rather than run off. Even when the first colony dies, the altered soil environment persists, so that any subsequent propagules that land there will find more equable conditions – soils that are more porous, more easily worked, and damper – and so enjoy enhanced survivability than the propagules that preceded them. These will, in turn, modify the soil further, bequeathing a still more equable environment to propagules that follow them. This continuous and building recolonization bias eventually produces soils that are locally enriched by the many generations of termite colonies gathering nutrients from the surroundings and transporting them to the central site of the colony. This focal enrichment leads to the development of the heuweltjie's distinctive plant community. If recolonization continues for a long enough time, an impermeable calcite basement eventually forms about two meters below the surface, which enables perched water tables to accumulate below the heuweltjie. Once the calcite basement is formed, the heuweltjie is essentially a permanent feature of the landscape.

As in the *Macrotermes-Termitomyces* symbiosis, heuweltjies embody a competition not so much between genomes but between two large-scale pathways for mass and energy flow: heuweltjie soils that hold rainfalls well, and non-heuweltjie soils that divert rainfalls to runoff (Figure 12). Furthermore, the environment of the heuweltjie determines function much farther into the future than do the termites that build it. The ages of some heuweltjies have been dated to around four thousand years (Moore and Picker 1991), while the lifespan of a typical *Microhodotermes* colony is three to four years. Thus, it is not so much the termites that evolve to the prevailing arid environment, it is the environment that evolves to suit the physiology of the termites. The heritable memory that enables these soil environments to evolve resides not in the enduring genes, but in the enduring legacy of the modified environment of the soil.

Sitting up at the organism's wake

Nothing in what I have outlined here negates or undercuts the essential roles that genes play in determining function, fitness, and the evolution that follows. However, genes really have no meaningful existence outside the functional environments that carry them into the future. Any theory of evolution that fails to account for the behavior of these functional environments will be incomplete, at best.

It has been said that the essence of the Darwinian revolution was the banishment of typological thinking from biology, and its replacement by

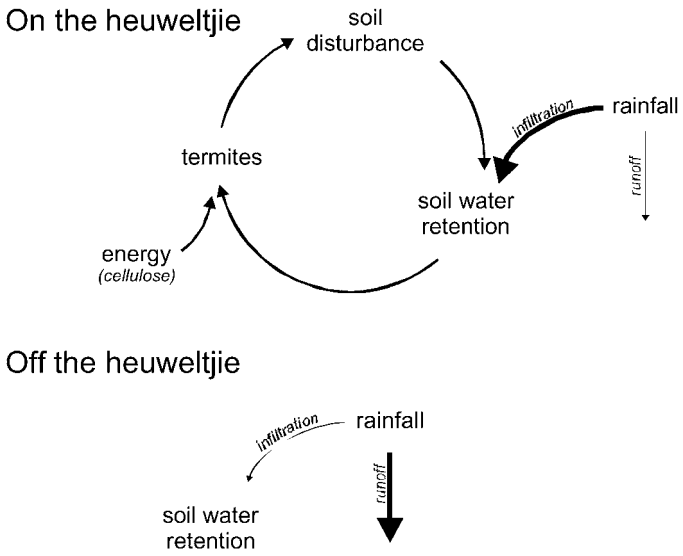

Figure 12. Extended organismal function on a heuweltjie (*a*.) and off (*b*.). On the heuweltjie, soil water balance is dominated by infiltration, which establishes conditions for the recolonization by termites. Off the heuweltjie, soil water balance is dominated by runoff, which leaves a soil environment inhospitable to termites.

populations as the only proper venues in which to think about evolution and its subsidiary questions – what species are, whence they come, how they evolve. This essentially Neoatomist approach to evolution, indeed to biology in general, has been extraordinarily successful, evidenced by biology's extraordinary transformation through the twentieth century. Left largely unanswered, though, has been an important question: populations of what? Organisms? Cells? Ecosystems? Molecules? Genes? In 1978, the answer seemed reasonably clear – it was the "atoms of heredity" – genes – that were at the heart of it all. For a host of reasons, some alluded to briefly above, that answer is no longer so clear.

Perhaps the time is now ripe for another rethink of biology's philosophical underpinnings, liberating the gene from being simply an atom of heredity, and putting it in its proper place as one of several arbiters of the suites of physiological transactions that organisms comprise (Laland and Odling-Smee et al. 1996; Odling-Smee and Laland et al. 1996; Laland and Odling-Smee et al. 1999). Integrating evolution and physiology in this way would involve reintroducing a sense of purposefulness to our thinking about evolution. Purpose has been, of course, forbidden intellectual territory for some time, presumably because it steers us dangerously close to the Platonic teleology that Darwinism has rightly put aside. Nevertheless, organisms are, if nothing

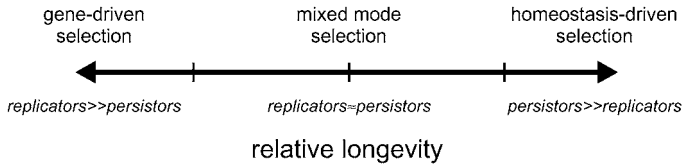

Figure 13. Course of evolution can be influenced by relative longevity of replicators and persistors.

else, purposeful creatures, and failure to acknowledge this frankly steers us in other dangerous directions. The important questions are: how are organisms purposeful, and how does the purposefulness work? To the physiologist, the purposefulness of organisms is embodied in the phenomenon of homeostasis, which is tantamount to the forward reach in time of physiological function. Natural selection is simply the emergence of living systems that extend their purposeful reach farther into the future than others. Inevitably, they reach toward states of homeostasis, propelled there by agents of homeostasis that can operate over many scales of both time and space. A gene, then, is not simply a device for encoding a function, or an atom of heredity, but a means of imposing a degree of predictability on future flows of mass and energy through living systems. The key to an encompassing theory of evolution that unites Darwinian selection, Mendelian inheritance and physiological homeostasis requires the frank embrace of the future, and the goals that lie there.

Evolution then becomes less the province of one class of arbiters of future function – genes – and more the result of a nuanced interplay between the multifarious specifiers of future function (Figure 13). In some systems, and over some time scales, genes will indeed have the farthest forward reach, as they do in multicellular organisms that reproduce by gametes and then die. For other systems, persistors – enduring or resilient modifications of organisms' environments – will have more of a forward reach than will the replicators. The dynamics of evolution at any scale may be determined not by a single model of competition and selection among replicators, but by a spectrum of models determined by the relative longevities of replicators and persistors. At one end of the spectrum, where replicators are the most enduring, will reside our conventional models of gene-centered Darwinism. At the other end, where functional environments far outlast replicators, persistor-driven evolution will be the norm, with competition among, and evolution of, environments, rather than genes, determining the future fates of organisms. In between will be a host of evolutionary dynamics influenced to varying degrees by both replicators and persistors.

Richard Dawkins' intention in writing *The Extended Phenotype* was, to paraphrase him, "not to praise the organism, but to bury it". It is worthwhile asking, twenty-five years later, did he succeed? My answer would be: yes and no. On the yes side, *The Extended Phenotype* was a splash of cold water in the face of an organism-centered view of evolution that seemed unable to sustain the full implications of Darwinist thinking. I think it fair to say that the organism never quite recovered from the blow. But a funny thing happened on the way to the graveyard. As we mourners gathered at the wake to reminisce and recount the life of the organism concept that has now passed away, we have come to see the organism in an entirely new light, revealing dimensions that were hidden as long as the organism was confined to a body – the extended physiology, the importance of homeostasis, the overriding importance of scale and dimension. And as we mourned, as in those resurrection dramas that were a traditional part of Celtic wakes, the corpse sat up, the concept of the organism taking on new life and inviting us to appreciate it in ways we could never have conceived before its passing. In my opinion, it was this, the resurrection of the organism, and not its burying, that is Richard Dawkins' greatest legacy.

Notes

[1] Hillaire Belloc. *The Microbe* (1897).

[2] *Diner:* Waiter, there's a fly in my soup!
Waiter: Don't worry sir, he won't eat much.

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