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The hierarchical expansion of sorting and selection: sorting and selection cannot be equated

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Abstract.—In a nonhierarchical world, where selection on organisms regulated all nonrandom evolutionary change, the traditional equation of selection (a cause of sorting) with sorting itself (differential birth and death among varying organisms within a population) would rarely lead to error, even though the phenomena are logically distinct (for sorting is a simple description of differential "success," and selection a causal process). But in a hierarchical world, with entities acting as evolutionary individuals (genes, organisms, and species among them) at several levels of ascending inclusion, sorting among entities at one level has a great range of potential causes. Direct selection upon entities themselves is but one possibility among many. This paper discusses why hierarchy demands that sorting and selection be disentangled. It then presents and illustrates an expanded taxonomy of sorting for a hierarchical world. For each of three levels (genes, organisms, and species), we show how sorting can arise from selection at the focal level itself, and as a consequence either of downward causation from processes acting on individuals at higher levels or upward causation from lower levels. We then discuss how hierarchy might illuminate a range of evolutionary questions based on both the logical structure of hierarchy and the historical pathways of its construction—for hierarchy is a property of nature, not only a conceptual scheme for organization.

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I. Disentwining Sorting, and Selection

In Darwinian theory, evolutionary change is the product of sorting (differential birth and death among varying organisms within a population). Sorting is a simple description of differential representation; it contains, in itself, no statement about causes. As its core, Darwinism provides a theory for the causes of sorting—natural selection acting upon organisms in the "struggle for existence." However, other processes (genetic drift, for example) produce sorting as well; thus, the two notions—sorting and selection (a favored theory for the cause of sorting)—are quite distinct and should be carefully separated.

Yet our literature often conflates the two, particularly in citing as adequate evidence for selection only the simple observation of sorting or, even more confusing, by actually defining selection as nothing but sorting. Futuyma, for example, writes in his textbook (1979, p. 292) that "selection . . . *is* differential survival and reproduction—and no more."

In this paper, we advance three primary and related claims:

1. Biologists must clearly distinguish between sorting and selection.

2. Sorting has been equated with selection because the reductive argument of classical Darwinism—that selection predominates and that organisms are *the* target of selection—permits no important alternative nonrandom explanation for sorting. Thus, the two concepts, though logically distinct, are implicitly and easily conflated.

3. The hierarchical perspective identifies several kinds of evolutionary individuals (genomic constituents, organisms, species) at ascending levels of inclusion, and recognizes both selection at all levels and transfer of effects between levels. It thus provides a framework for an expanded taxonomy of the causes of sorting. We present such a taxonomy, explore its implications, and use it to delineate the role of traditional selection as one among many testable causes of sorting.

Our expanded theory of sorting arises from a nontraditional perspective about two fundamental aspects of life.

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1. Individuality.—If organisms were the only reproducing individuals, an exclusive focus on selection as the nonrandom cause of sorting would make sense (although the distinction between selection and reproductive success, as analyzed by Sober and Lewontin [1982] would still hold). But the broader concept of "individual" compels us to recognize that other entities, including genomic constituents and species, are also biotic individuals (Ghiselin 1974; Hull 1974, 1980). These other entities share with organisms all the crucial characteristics of reproducing evolutionary objects: they have births and deaths, and sufficient coherence and stability during their lifetimes; they interact with the environment; and they produce offspring by genealogical descent with both high inheritance and the possibility of change. (See also Hull [1980] on interactors and replicators.) Sorting may thus occur as surely among genomic bits of cells, and among species in monophyletic groups, as it does among organisms in populations.

2. Hierarchy.—These different kinds of individuals are nested within each other as ascending levels of a genealogical hierarchy (Arnold and Fristrup 1982; Gould 1982a,b; Vrba and Eldredge 1984). This hierarchy expands the potential causes of sorting in two important ways. First, sorting can occur actively among individuals at all levels, not just among organisms. Second, levels interact, and events at one level propagate as effects to others. Causation flows from lower levels to higher, and also in reverse (upward and downward causation [Campbell 1974]). Sorting at any focal level need not be caused by selection among individuals at that level.

The traditional view that equates sorting among organisms with selection upon organisms arises as a consequence of neglecting hierarchy and its expanded concept of individuality. If we observe nonrandom sorting among organisms under the conventional view, then we must infer that it results from selection upon them, for the only alternative is forced correlation of other characters with traits actively selected. All recognized exceptions to the equation of nonrandom sorting with direct selection—pleiotropy, hitchhiking, and allometry, for example—arise as consequences of organismic selection upon other traits; death (or decrepitude) hitchhikes on life in the Williams-Medawar theory of senescence. However, with selection (and other causes) acting upon several kinds of individuals in a genealogical hierarchy, sorting among individuals at any level (organisms, for example) may only record causes at another level; no direct process need operate at all upon the individuals under consideration.

In studying evolution we have been strongly constrained by the limits of our scale. As organisms we readily see and accord significance to other organisms. We can directly comprehend much of the nature, spacing, distribution, interaction, and sorting of phenotypic subparts that make up emergent organismal characters. But analogous phenomena underlie genomic characters (which we are seeing more and more clearly through modern technology), and emergent characters of species (which most biologists are not even inclined to imagine). The problems of evolution extend well beyond organismal adaptive complexity. Characters and dynamics at higher and lower hierarchical levels are also crucial to life's history. Indeed, without reference to these other aspects of life, patterns at the organismal level can scarcely be comprehended.

II. Hierarchy and Expanded Possibilities for Sorting

Evolutionists have a good, almost intuitive understanding of how selection works upon organisms. But complexities arise when we expand the concept of selection hierarchically, and more precision is required in a statement of selection that applies equally to sorting among genomic constitutents and among species. The problem concerns the nature of characters (that is, those physical states arising from the nature and distribution of subparts in an individual). All reproductive individuals have *aggregate characters* (inherent properties of subparts) and also *emergent characters* (arising from organization among subparts). The identical genes in every cell form a set of aggregate characters of the organism, but in contrast, phenotypic characters (like proteins, coat colors, or behaviors) are emergent at the organismal level. Similarly, if all organisms in a moth species are black, then black is an aggregate character of the species. Only characters that arise from distribution and interaction *among* organisms are emergent at the species level.

(By characters, we refer only to physical patterns, and not to their dynamic or ecological consequences. Thus, possible emergent characters of species include population size, distribution, and composition. They do not include such phenomena as speciation rates, adaptive zones, and interspecific reproductive compatibility.) We suggest that individuals are subject to selection at any level for the characters they possess only if these characters are heritable and *emergent,* and if they interact with the environment to cause sorting (see also Vrba 1980a, 1984b; Gould 1982; Vrba and Eldredge 1984): *Selection encompasses those interactions between heritable, emergent character variation and the environment that cause differences in rates of birth or death among varying individuals.* Thus we would say that any other cause of sorting is not selection at the level under consideration. A hypothesis of selection is at the same time a claim about aptation. (We use "aptation" as an inclusive term for any character currently subject to positive selection regardless of how it evolved [Gould and Vrba 1982] in contrast to the narrower term adaptation for a character evolved by selection for its current function sensu Williams [1966].)

Sorting not based on selection among objects sorted arises primarily from interactions between levels. However, an interesting asymmetry distinguishes the propagation of effects in upward and downward causation. Selection at lower levels may, but need not, affect individuals at higher levels. Sorting among genomic elements need not change the phenotypes or fecundities of organisms (as in the "selfish DNA" hypothesis, where increase in copy number of middle repetitive DNA occurs, at least in initial stages, with no or minimal effect upon containing organisms).

However, any selection at higher levels must entail sorting among individuals at all lower levels. Downward causation always flows to lower levels; upward causation need not occur. Suppose, for example, that by species selection one monophyletic subclade of a larger clade increases the proportion of species that it contributes to the entire clade. If all species contain roughly the same number of organisms, then organisms bearing apomorphic characters distinguishing the selected subclade will increase in relative frequency within the clade. Similarly, genes held uniquely by phyletic heritage within the selected subclade will also be sorted positively. Thus, organisms and genes are sorted, but no active selection has occurred on emergent characters at either the organism or gene level. All sorting occurred as effects of downward causation from species selection for increase in numbers of species within a favored subclade. In such cases, sorting of organisms and genes still arises by some kind of selection, though at a different level and not upon the objects sorted (a crucial distinction). Our approach does not promote any negative statement about the natural frequency of selection processes; in most cases, nonselective sorting at a focal level may well be dictated by selection at another level above or below. But the hierarchical model does stress an important distinction: a causal process of selection at a focal level is ontologically different from a process of sorting among focal individuals driven by events at a higher or lower level.

Sorting, in other words, can occur as an effect of causes at other levels. All causes of sorting are not selection, and we need an expanded taxonomy of sorting that includes selection at a focal level—the only category usually recognized (and generally allowed only for organisms)—as only one possible cause of sorting.

III. An Expanded Taxonomy of Sorting

Our chart (Table 1) includes only heritable, nonrandom causes of sorting and the flow of causality between levels. But biologists also recognize two other modes, which we will record below but not discuss extensively: (1) nonheritable nonrandom sorting based on ecophenotypic differences (organisms) or varying histories of climate and geology (species), and (2) heritable but random sorting (from genetic drift at low levels to macroevolutionary trends at high levels, as in Raup and Gould [1974]). We will then present our taxonomy of the mode that has been of central concern to evolutionary biologists for more than a century: heritable, nonrandom sorting, including the results of selection. For each of three categories—character environment interaction at the focal level, downward causation, and upward causation—we will discuss examples at three ascending levels: genes, organisms, and species.

1. The distinction between sorting correlated

TABLE 1. A classification of nonrandom, heritable causes of sorting. Examples are discussed in the text. The arrows indicate general directions of causality between levels (not necessarily between the particular examples). Note that the middle category, phenotypic selection on organisms acting at the focal level, has been usually interpreted as *the* cause of *all* sorting involving organisms. The reality is far more complex and interesting.

Focal level	Cause of sorting: Downward	At focal level	Upward
Genes	E.g., Protan mutant hitch-hiking on selection for malarial resistance (Templeton 1982)	Selection of "selfish DNA" E.g., *Alu* family (Doolittle, 1982)	
Organisms	E.g., rodent phenotypes hitch-hiking on species selection in *Spalacopus* and *Ctenomys* (Gilinsky 1987)	Conventional phenotypic selection, e.g., industrial melanism in moths (Kettlewell 1958)	Effect sorting of organismal phenotypes
Species		Species selection, e.g., rodent species in *Spalacopus* and *Ctenomys* (Gilinsky 1987)	Effect macroevolution, e.g., antelope species in Alcelaphini-Aepycerotini (Vrba 1984a)

with heritable characters and sorting that involves only ecophenotypic (nongenetic) variation is not controversial. Variation among conspecific organisms in birth and death rates may depend entirely on local or temporary environmental differences (see Stearns 1982). Similarly, at the species level, phylogenies may differ in rates of species diversification, not because they differ in heritable characters, but purely as a result of extrinsic factors, including varying climatic and geological histories (Cracraft 1982).

2. The term "random" can be used in more than one sense to describe evolutionary phenomena. The most restricted biological sense implies that no consistent relationship exists between random events at a focal level (such as organismal sorting) and *any* character variation (whether genomic, organismal, or among species). Theoretically, sorting that is random in this sense may occur among noncoding DNA sequences (Dover 1980), at the levels of genotypes and phenotypes (the familiar random drift) and among species (Raup et al., 1973).

However, in a second and technically incorrect but common usage, events at a given level are viewed as *random with respect to natural selection at that level.* This concept is most familiar in the conventional statement that gene mutations are random in relation to selection among organismal phenotypes. In such cases, everyone understands that proximal causes govern mutation, and that observed mutations are part of a limited possible set and may strongly biased. Mutations are "random" only with respect to adaptive requirements of phenotypes at the next higher level. In *Drosophila melanogaster,* for example, the transposable elements called *P* factors have a special tendency to induce mutations at the *singed* locus, which may engender selective consequences at the phenotype level (Lindsley and Grell 1944; Golubovsky 1978; Shapiro 1983). In labeling such *singed* mutants "spontaneous" or "random," explanatory focus is placed on selection in populations, and not on the directive, genomic process. At a higher level, selection among organisms can result indirectly in characteristic, genetically based group size. Theoretically such variation among species may lead to species selection. The de novo introduction of the species character is determined by upward causation from lower levels and yet, in the conventional sense, it is random with respect to any selection pressure among species.

In the same usage, "randomness" applies not only to up- and downward causes of introduction, but also to *sorting* in variation, with respect to any natural selection at the focal level. For instance, differential rates in reproduction of chromosomes, as in segregation distortion, would cause a change in variation of any "selfish

DNA." But such a cause would be random with respect to selection at the noncoding DNA level. Segregation distortion may also alter the differential frequency of organismal phenotypes by upward causation, which would then be random with respect to a (possibly opposing) force of selection at the organism level (see Lewontin 1970). Reference to a "random" process in this second meaning usually implies that "we either cannot know or do not choose to know the deterministic basis of the process we are considering." In traditional biology, we almost always place our explanatory focus on a particular causal series at one level (usually selection in populations), thus relegating deterministic bases from other levels to the "black box" of randomness. In contrast, the hierarchical approach restricts the term "random" to its first meaning, and seeks to expand the analysis of such deterministic bases by addressing a variety of causes that transfer between levels.

We may now present our taxonomy of nonrandom, heritable causes of sorting (Table 1), considering sorting at three levels: genomic, organismal, and species. The cause of sorting always flows from interaction of characters with the environment, but we ask whether characters that cause sorting at a focal level are emergent at that level, at the next higher level, or at the level below. To rephrase the question: does sorting at a focal level result from selection at that level, or from downward or upward causation?

A. Character-environment interaction at the focal level: the conventional use of selection.— Selection at the level of noncoding genomic elements, "selfish DNA," was proposed by Doolittle and Sapienza (1980) and Orgel and Crick (1980). In a more recent review, Doolittle writes (1982, p. 3): "Pieces of DNA which code for nothing useful run the risk of deletion. If there are sequence-specific strategies by which deletion can be avoided, natural selection operating within genomes, independently of organismal phenotype or population adaptability, will give rise to and maintain DNAs which adopt such sequence-specific strategies." He cautiously concludes that the observed patterns, both intragenomic and within and among species, do indeed support the notion. He cites as an example the middle-repetitive DNA *Alu* family, repeated some 300,000 times in the human genome, and with identifiable relatives in other mammals.

A particular selection process need not be confined to individuals within a single higher individual—as in selection of homologous DNA sequences. If particular *Alu* sequences possess a transposition mechanism that boosts their selective success (Elder et al., 1981), then that mechanism will operate wherever it occurs, across separate organisms and even species.

The same principle applies to selection at the organism level. A particular selection process among genotypes and phenotypes need not respect species boundaries. Consider selection for industrial melanism in moths, demonstrated in lineages belonging to several different lepidopteran families in Britain and elsewhere. If the genetic basis is homologous in different species (as seems likely [Kettlewell 1958; Ford 1971]), then the same selection regime may have operated across boundaries of species and even higher taxa.

The idea of species selection has long been in our literature in the form of isolated reflections (e.g., Wright 1956; Fisher 1958), but the model of punctuated equilibria (Eldredge and Gould 1972) suggested that rules of sorting among species are central to explanations of long-term evolution. This led in turn to renewed explorations of species selection (Eldredge and Gould 1972; Stanley 1975, 1979; Gould and Eldredge 1977; Eldredge and Cracraft 1980), but few studies have yet tested for species selection with the proper criterion of heritable, *emergent* species variation. Gould (1982b) found the hypothesis of species selection supported by the fossil record of volutid neogastropods, as studied by Hansen (1978). Vrba (1984a) examined phylogenetic trends in African Miocene-Recent antelopes and other mammals and did not find evidence for species selection. An interesting example in South American rodents has recently been discussed by Gilinsky (1987). Tuco-tucos (genus *Ctenomys*) and coruros (*Spalacopus*) have evolved remarkable fossorial features—an "adaptive complexity" very probably produced by organismal selection for fossorial existence. But these phenotypes form only a part of the biological pattern that needs explanation. The younger genus *Ctenomys* contains about 60 living species, while *Spalacopus* is monospecific. *Ctenomys* has a wider geographical distribution, but, even where they are sympatric, *Ctenomys* outnumbers *Spalacopus* by 16 species to 1. Gilinsky points

to strong evidence for low gene flow between populations within species of *Ctenomys,* while populations of *Spalacopus* are rarely isolated from each other. He interprets this difference in distribution and interaction of populations as an emergent species-level character and concludes that the data indicate past occurrence of species selection.

B. Character-environment interaction at higher levels: downward causation to sorting at the focal level.—Two distinct processes can be identified in this category (for extended discussion, see Vrba and Eldredge 1984). (1) First, any differential birth and death processes *among* individuals at a higher level will inevitably also sort entities at lower levels. These entities just "come along for whatever ride" is dictated by sorting among the higher individuals in which they reside. (2) Second, the structural, emergent properties of a higher individual may "downward cause" sorting of variation *within* the body of that single individual.

Many examples of the first case have been investigated at the genome-organism interface. For instance, intense selection on a major locus may result in hitchhiking effects (Maynard Smith 1978). As an example, Templeton (1982) cites the work of Filippi et al. (1977) on human resistance to malaria on the island of Sardinia. Selection favoring the X-linked allele that causes glucose-6-phosphate dehydrogenase deficiency (a malarial adaptation) might have entailed a hitchhiking effect at the closely linked locus causing Protan color blindness.

The same principle applies at the organism-population and organism-species interfaces. Thus, if Gilinsky's (1987) argument is correct, then species selection determined that the phenotypes characteristic of *Ctenomys* became vastly more diverse and multiplied than those of *Spalacopus.* We note here a component of sorting at the level of tuco-tuco and coruro organisms that is dictated not by selection at their own level but via the rules governing rates of speciation and extinction.

Examples of case 2 at the higher levels—that is, downward causation from emergent species characters to sorting among genotypes and phenotypes within a population or species—are also well known. Any case in which the size and separation of populations leads to fixation of mutants against the force of selection within populations belongs here. Consider *Clarkia lingulata,* an annual flowering plant, and its sister species, *C. biloba,* two species differing in chromosomal translocations. Hybrids are sterile because chromosomal heterozygotes experience mechanical problems during meiosis (Lewis 1961; Paterson 1982). Thus the translocations must have been fixed originally not by selection but randomly, in spite of selection against the heterokaryotypes. Population genetic theory indicates that extremely small effective population sizes are necessary for random fixation of such negative heterotic karyotypes (Wright 1941; Lande 1979). In this example we see a probable flow of causation between levels: Initially, dynamics at the level of organisms produced a population structure of small effective size—the emergent population character. In turn, downward causation from population structure was necessary to yield positive sorting of mutant translocation phenotypes, in opposition to selection against the heterokaryotypes.

Of course, these population structures in *Clarkia* need not be heritable. Such emergent features of populations and species may arise as transient phenomena, induced by extrinsic environmental conditions, and still exert downward causation with long-term evolutionary significance. Only if characters are genetically based, and vary among lineages in a phylogeny, can they be implicated in species selection. But the notion that heritable, emergent variation exists at the species level, and may be ubiquitous in some groups, is entirely reasonable. An example might be characteristic population structure resulting from social behavior, as in mice (Lewontin 1970), in antelopes (Vrba 1984a); and in other mammals (Wilson et al. 1975).

C. Character-environment interaction at lower levels: upward causation to sorting at the focal level.—Selection at a lower level may churn without attendant birth or death (or any change in emergent variation) among more inclusive individuals. Often, however, causes do propagate upward.

We suggest that such propagation may frequently occur at the genome-organism interface, a controversial argument (see also Vrba 1984b) because all heritable, nonrandom sorting among organisms has traditionally been subsumed un-

der the catch-all concept of selection. But we maintain that unless emergent organismal characters (that is, phenotypes sensu lato) interact with the environment, not merely during the reproductive process per se but *as the cause of differential reproduction,* then the process should not be termed selection. Consider the following hypothetical case. A mutant, noncoding DNA sequence is "selfishly" selected at its own level. Suppose that the presence of this sequence at higher frequencies directly produces an increase in birthrate among the organisms containing it. If, by such a process, no other phenotypic variation whatsoever has a deterministic relation to differential birthrate, would we call such a phenomenon natural selection at the phenotype level and the mutant noncoding sequence an organismal adaptation? We suggest that we would not, but rather that we should distinguish such a process from conventional natural selection not only theoretically but also by name. We can cite no documented example, but suggest that this absence may say more about the state of our knowledge and expectations than about the natural frequency of such processes. For example, several reports of *r* versus *K* life-history variation within and between species, and the little we know of their genetic bases, might merit closer examination in this light.

Biologists have, however, documented upward causation to the biased *spread* of variation among organisms without organismal selection. The dynamics of *t*-alleles in house-mice provide a classic example. The effective sperm pool of heterozygous males in some populations contains about 85% *t*-bearing sperm, probably as a result of powerful organelle selection at the level of chromosomes (Lewontin 1970). The frequency of *t*-bearing individuals is thus biased by upward causation, but selection at the level of organisms reduces the frequency of *t,* because males homozygous for *t* are sterile. Another kind of process involving the spread of repeated DNA sequences has been discussed by Dover et al. (1982) as "molecular drive." Multiplication and transfer of certain sequence variants among chromosomes in parental genomes occur in such a way that the variants gain a biased representation in descendants—a process that operates outside the Mendelian system and, unless relevant phenotypic effects occur, without selection among organisms. In both cases, the spread of variants does not involve upward causation to *sorting.* That is, the *t*-alleles and DNA sequences do not gain greater representation by causing a higher birthrate among organisms in which they reside. They will spread even if birthrates are the same among organisms containing and lacking them. In other words, the dynamics causing the spread are resolved at lower genomic levels and not by organismal sorting. But we predict that upward causation to nonselective differential birth among organisms may also be quite common.

The principle of upward causation to "sorting that is not selection" was first discussed at the organism-species interface and called the "effect hypothesis of macroevolution" (Vrba 1980a, 1983). The term "effect" follows Williams (1966), who suggested that only those characters shaped by natural selection to perform particular functions should be called adaptation; and that adaptations may have incidental effects not arising as direct consequences of selection. Paterson (pers. comm. 1978) articulated the idea that speciations are always effects sensu Williams (1966). Vrba (1980a) suggested that effects may extend upwards across hierarchical interfaces—especially that regimes of organismal selection (differing among the lineages of a monophyletic group) may yield disparate patterns of species diversification as well as long-term trends in divergence. Biologists have long recognized that species become extinct because organismal selection removes their organisms one by one until none are left—species sorting as an effect of organismal selection in the Darwinian sense. The crucial aspect of the effect hypothesis (and its novel component not addressed by previous theory) lies elsewhere: selection for proximal fitness of organisms may also, and incidentally, drive *speciation rates.* To the extent that punctuated equilibrium pertains, differential net increase in species must also force an overall trend in divergence of organismal characters. These organismal characters get to the pinnacle of a trend not because they are better than others—the adaptations at the nonspeciose end of the trend may be doing an equally good or better job of promoting organismal and genic fitness. They prevail in trends because they incidentally cause the multiplication of higher individuals, the species, that bear them—"incidentally" because

their selection clearly did not occur for the function of promoting speciation.

Apparent examples can be found among Miocene-Recent phylogenies of African mammals. Vrba (1984a) used monophyletic groups of extinct plus extant species to test a series of rival hypotheses:

1. The cause of differential net increase in species is random.

2. The cause resides in the interaction of organismal characters with the environment (the effect hypothesis). For example, phenotypic characters conferring breadth of resource utilization (measured, for example, by recording the range of food items eaten by organisms in a species) may regulate speciation rate in the following way: the probability that organisms will perceive environmental changes as deterioration of habitat is greater in the case of certain specialists than in generalists. Thus, such stenotopes should be subject more frequently to directional selection at the level of genotypes and phenotypes, and consequently to fragmentation and divergence of populations, speciation, and extinction.

3. The cause arises from the interaction of emergent species characters with the environment (species selection). For instance, within these mammal species, phenotypes related to mobility and social behavior might result in characteristic population structure and separation—emergent and heritable characters of the species. Lineages of isolated populations with low gene flow may be expected to exhibit high speciation rates.

In the cases examined, results falsified the random and species selection hypotheses and upheld predictions of the effect hypothesis. The Alcelaphini-Aepycerotini sister group has been analyzed in detail elsewhere (Vrba 1984a). Briefly, the alcelaphine (blesbuck-hartebeest-wildebeest) fossil record and extant biology indicate: (1) high speciation and extinction rates (33 extinct and 7 extant species since the terminal Miocene, and at least 18 events of splitting and about a dozen terminal extinctions); (2) high divergence at the level of organismal phenotypes expressed in a massive trend toward such features as increasingly complex horn shapes, long faces, and masticatory specializations for tough forage; (3) extant species are all specialist grazers; and (4) extant species are migratory, with apparently, low degree of isolation but high gene flow among populations. In contrast, the aepycerotine (impalas) sister lineage indicates: (1) no lineage splitting or terminal extinction since common Miocene ancestry with the alcelaphine clade; (2) low divergence (virtual stasis) in organismal phenotypes (skulls and skeletons of modern impalas look much like fossils several million years old); (3) each extant organism both grazes and browses on a broad variety of kinds and parts of plants; (4) modern impala populations are markedly nonmobile, with static clumped distributions and low gene flow among isolated populations. In sum, the data suggest that local pressures of selection on specialist grazing organisms, and not factors of gene flow arising from species-level characters, caused this highly disparate pattern of diversity and trend between the two sister groups.

Consider also the modern abundance of organisms in these two sister groups: census data (Vrba 1980b; Greenacre and Vrba 1984) suggest that the single impala species may be leaving as many genes to future generations as all the diverse alcelaphine species put together. This result strikes a jarring note under the traditional view that organismal success extrapolates smoothly to success of species. But the observed pattern is quite compatible with the hierarchical perspective. If one insists on equating "more individuals" with "success," then, in this example, organismal and species success are, to a large extent, independent. We would not link the alcelaphine trend to this traditional notion of "success." In the teleological sense of selected progress, alcelaphine evolution is going nowhere. The trend represents pure upward causation and effect.

Nonetheless, it would be equally false or misleading to argue that the case involves no trend at all because each subclade maintains about the same number of organisms, while one (the alcelaphines) only parcels them into more species. Simply by generating more species, the Alcelaphini develop a potential for phenotypic diversity denied to the Aepycerotini, which maintain the same number of organisms at a single modal phenotype. Since, empirically, a pattern of punctuated equilibrium seems to predominate in this phylad (Vrba 1980a, 1984a), the impala subclade, by failing to speciate, can develop neither

increase in diversity nor net change in form—while the alcelaphines, with potential for change in each event of speciation, become evolutionarily labile as an incidental effect of high speciation rates caused at other levels.

IV. Some Implications of Hierarchy

Our taxonomy of sorting (Table 1) may strike some readers as a complex multiplication and overrefinement of distinctions. We regard it as an effort in simplication and unification. A general theory of biology is a theory of hierarchial levels—of how they arise and interact. Entities that play the same role in the evolutionary process must be classed together. This point has been made for individuals (Ghiselin 1974; Hull 1974, 1980), replicators (Dawkins 1978), units of selection (Hull 1980), adaptations (Williams 1966; Gould and Vrba 1982), and aggregate versus emergent characters of individuals (Vrba and Eldredge 1984). The same principle also applies to evolutionary processes themselves. The same general causes are likely to operate at each level, both in its initial evolution, and subsequently in the de novo introduction and sorting of variation. We must consider the evolutionary process itself as basic, and explore its common modes of action up and down the hierarchy—particularly interactions between levels (see also Pattee 1970; Hull, 1980).

Such an approach provides a proper framework for understanding evolution as a historically contingent process, and for grasping the primary trends of that history itself. For example, hierarchy permits us to reformulate the basic statement that mutations are "random" as raw material for evolutionary change (see Sec. III, para. 2). This claim, though canonical to the point of catechism, has always bothered evolutionists because we know that, considered at the physicochemical level of gene structure and action, mutations are not random events. They represent causal series independent from selection upon organisms, and they maintain their own lawlike properties of origin, sorting, insertion, and expression in phenotypes.

Of course, evolutionists have really meant something else in stating that mutations are "random": namely, that they occur without reference to adaptive requirements of phenotypes. In other words, "random" has been technically misused to express a relationship—a claim that the frequency and direction of mutation is unrelated to natural selection upon phenotypes. (We also recognize that the statement is not always true, as in the derepression of mutator loci under regimes of intense selection.) "Mutations are random" is a misstatement that subtly embodies a restrictive, nonhierarchical view of life—selection in populations as *the* process of evolution, with others evaluated in relation to it.

The hierarchical perspective resolves any paradox or difficulty. Since mutations occur at a level below organisms, their independence should lead us to anticipate no direct causal relationship with natural selection at the conventional level of phenotypes. We coined the term "exaptation" for features arising for one reason, and then fortuitously available and co-opted by selection for another reason (Gould and Vrba 1982). Form-function correlations may be ubiquitous in nature, and may express the good design of organisms and other entities, but they need not arise directly by "adaptation" (sensu Williams 1966)—that is, by direct selection for current utility. We originally restricted our discussion of exaptation to features arising at one level and later co-opted for different function at the same level—feathers evolving for thermoregulation and later co-opted for flight, for example. But the scope of exaptation becomes vastly expanded under the hierarchical perspective—because *all upward or downward causation to new characters may lead to exaptation.* Mutations arise for their own reasons at their own level. If they affect phenotypes in a way that selection upon organisms favors, then these mutant phenotypes are exaptations at the level of organisms. We grasp this clearly for upward causation, since all heritable, emergent novelty that becomes subject to selection can be described as exaptation. But the principle also works for downward causation. Mass extinction, for example, may generate a pervasive realignment of life's diversity. The new proportions and types of creatures available for adaptive molding to renewed ordinary environments after such an extraordinary event form a largely fortuitous pool of exaptive potential.

This expansion of the role of exaptation vs. adaptation contains an important lesson for our basic attitude toward history. Hypotheses of

adaptive optimality represent the extent to which we can ignore history and treat form and ecology as a problem of unconstrained equilibrium in a timeless world. Exaptation embodies all the quirkiness of historical contingency—you only get to an advantageous place if some other process for another reason gave you the goods. Hierarchy and the exaptive status of cross-level causation teach us that we inhabit a world of enormous flexibility and contingency—a world built by irrevocable history.

Hierarchy and cross-level causation are claims about the empirical status of things, not mere conceptual models. If nature's hierarchy is factual, then it was built historically—and its present structure might provide clues to its origin in time.

If we assume that hierarchies generally are built from the bottom up (chemical constituents must have preceded monophyletic groups), then we can recognize two important features of the genealogical hierarchy as historically progressive: loss of autonomy toward lower levels, and increase of autonomy toward higher levels (Vrba and Eldredge 1984). We may express this increased spatiotemporal bonding among lower-level individuals in two ways: (1) as a consequence of the laws of hierarchy (causality is asymmetrical, with downward flow ineluctable and upward flow possible; lower levels are more strongly impacted and constrained, higher levels more independent); (2) as a result of history (as hierarchies build and elements become incorporated as functioning parts of a complex system, their lineages evolve from selfish and competitive to mutually interacting entities).

Incorporation and tighter bonding are recurrent themes of evolution (from independent exons to gene clusters, prokaryotes to eukaryotes, asexual to sexual reproduction, unicellular to multicellular organization, parasites to symbionts). Individuals at lower levels aggregate and lose their independence, but higher-level individuals are not so strongly constrained and their parts retain more autonomy. Species as high-level individuals are affected by few levels above, and subject to relatively little context dependence (arising from their monophyletic groups). They are largely circumscribed in space and time by the externalities of environments—habitat preferences of component organisms in space, and vagaries of shifting environments in time. In contrast, spatiotemporal bounds upon organisms arise from internal epigenetic programs that, together with environmental causes, determine their forms and life spans. (Sexually reproducing organisms are additionally constrained by population structure from above because they must recognize and mate with another conspecific individual to ensure their genetic survival.) While sorting of organisms is, to a large extent, conditioned by their own interaction with the environment, the sorting of still lower-level individuals, genes and cells, is almost entirely constrained by whole-organism imperatives (or produces disastrous results at several levels if not so regulated, as in cancer).

The concept of progress has been particularly vexatious throughout the history of evolutionary biology. Darwin denied it explicitly as a logical consequence of natural selection (which only produces local adaptation, not general advance), but smuggled it back obliquely (Gould 1985) because he could deny neither his culture's obsession with the idea nor the broad sweep of life's history from early prokaryotes to recent intelligence. Hierarchy may resolve the issue by explaining life's weak and impersistent vector of progress as the result of deeper structural principles more inclusive than natural selection. If a historical system begins with simple components (as ours presumably did), and if complexity requires hierarchy and the bonding of lower-level individuals into higher entities (with a partial suppression of their independence and an altered status as parts of a larger whole), then a structural ratchet will ordain increasing complexity—"progress" if you will—as hierarchy builds historically.

Such a perspective raises interesting questions about life on earth. Could the earth have lost several early "experiments" in life because they didn't aggregrate to hierarchy? Can bonded parts that were once autonomous, lower-level individuals regain their independence? May we view some viruses as escaped units that have regained autonomy? In any case, if historically built hierarchy is a prerequisite for complexity, we may include intelligence among its improbable results and acknowledge hierarchy as the precondition of our ability to analyze it. We must also acknowledge that Darwin's theory does not have

the range to encompass the new kinds of data increasingly coming to light. In so stating, we are not merely pointing to the hitherto unsuspected *presence* of particular genomic parts or additional fossils. Rather, the *patterns* they form speak clearly of evolutionary laws beyond neo-Darwinism. Although we can as yet only see "through a glass darkly" into the workings of genes, the ontogenies of organisms, and the lives of species in macroevolution, we already glimpse enough to know that the expanded hierarchy is a reality. Darwin, who brilliantly inferred so much with substantially less information, was far more pluralistic in his conceptual explorations than most of his followers. We must recapture this spirit of discovery.

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