and studied in intact cells and in membranes from various tissues (*5*); binding can be altered or destroyed by treating cells with free or agarose-bound enzymes (*5*); the receptors are not detectable in intracellular membranes (*5*); adenylate cyclase activity in isolated membranes can be perturbed directly by physiological concentrations of insulin (*6*); and insulin receptors have now been extracted and purified from cell membranes (*7*). Although these studies are consistent with and further expand the observations made with insulin-agarose, it is very misleading to suggest (as Katzen and Vlahakes do in their first paragraph) that any such studies are based on a "basic premise" which is "justified" by the insulin-agarose studies. By analogy, the growing number of important studies concerning receptors for many peptide, cholinergic, and adrenergic hormones, and their localization to cytoplasmic membranes, are not based on studies with insoluble hormones.

A "unitary concept" of insulin action should in principle be viewed in the same way we evaluate the action of other hormones. In this respect the basic but still unanswered question is whether all of the metabolic effects of insulin can be explained on the basis of a single, unique, and fundamental biochemical event. This question is dependent not on interpretations of existing insulin-agarose studies, but on a better understanding of the detailed biochemical processes which follow the initial insulin-receptor interaction.

I take this opportunity to present additional evidence for the inherent activity of new and interesting insulin-agarose derivatives recently prepared in this laboratory. Insulin attached to agarose through very large macromolecular "arms," which consist of branched copolymers of poly-L-lysine-L-alanine can be demonstrated to be biologically active under conditions where no significant free insulin is released into the medium (Table 1). These derivatives may be especially useful because of the large distance which separates the insulin from the agarose backbone, and because of the great stability of the coupled insulin which results from the multipoint linkage of the copolymer to the agarose backbone.

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Generation Time and Genomic Evolution in Primates

It is widely accepted among those who study evolution at the molecular level that time is a major factor determining the degree of sequence difference among the homologous macromolecules of different species. Whether done on the globins (*1*), cytochromes c (*2*), fibrinopeptides (*3*), albumins (*4–7*), lysozymes (*8*), or DNA's (*9*, *10*), such studies have generally shown a strong correlation between the degree of sequence difference and the time that has elapsed since the two species being compared last shared a common ancestor. Substantial disagreement persists, though, on the question of whether the time factor should be measured in terms of astronomical time or generation length (*9–12*). These conflicting hypotheses have been subjected to direct test, and we here review evidence developed in such tests which strongly supports the astronomical time hypothesis.

It is important at the outset to make clear the distinction between measuring absolute and relative rates of macromolecular evolution. We believe that the failure to make that distinction has led others (*9–12*) unwittingly to favor the generation time hypothesis. To measure absolute rates of macromolecular evolution requires known times of divergence between living species and is thus completely dependent on a detailed and properly interpreted fossil record. The method is illustrated by the following example. If it is known that the albumins of two species A and B differ in sequence by x amino acid replacements and that the A and B lineages diverged t million years ago, the average rate of albumin evolution in this case is x/t amino acid replacements per million years of separation. Although t is sometimes known precisely, this will not be true for most absolute rate measurements. It is relatively easy

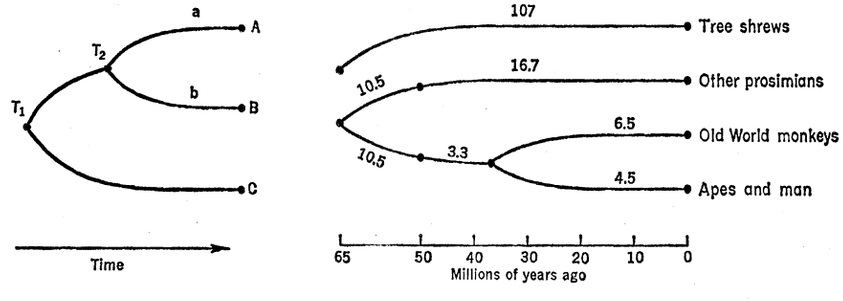

Fig. 1 (left). Phylogenetic relationships among three hypothetical living species, A, B, and C. The most recent common ancestor of A and B lived at time T_2, and the most recent common ancestor of all three species lived at time T_1; a and b are the amounts of sequence change (measured in amino acid replacements, immunological distance units, or nucleotide replacements) along the A and B lineages from T_2 until the present. The experimental A-B difference is assumed to be $a + b$; a and b can then be calculated if the A-C and B-C distances are known (*5*). Fig. 2 (right). Number of generations ($\times 10^6$) along various primate lineages according to the model of Lovejoy *et al.* (*12*). The divergence times are those used in (*12*); thus, we do not denote a specific divergence time for the tree shrews. For our calculations we have used the relationship between generations per year [$X(t)$] and time in millions of years (t) given in (*12*). However, one of their equations, $X_2(t) = 0.216t^{-0.181}$, is incorrect and should read $X_2(t) = 0.358t^{-0.181}$; we have used the latter in our calculations.

to date fossils, but far more difficult to date divergence events.

To measure relative rates of molecular evolution, on the other hand, requires only a knowledge of the cladistic (branching order) relationships among the lineages in question. Even when the fossil record has many gaps and uncertainties, cladistic relationships can usually be determined unambiguously.

The basic strategy of the relative rate method is outlined in Fig. 1 in terms of three living species (A, B, C) whose relationships are known from nonmolecular evidence. If the time of divergence of A and B is more recent than that of A and C or B and C, the relative amounts of change along the A and B lineages (*a, b*), may then be estimated by comparing the A-C and B-C molecular differences. Thus, if A is designated as a primate species whose time between generations is long (for example, man) compared to that of another primate species B (for example, tree shrew or other prosimian), the generation time hypothesis predicts that more change should have occurred along the B lineage than along the A lineage. Thus, the critical issue can be tested without any attempts at absolute rate measurements, and therefore without any dependence on the primate fossil record. Assuming, then, a monophyly of the modern primates relative to any nonprimate mammal (*13*), we may let C represent a modern carnivore species. The generation time hypothesis predicts that we should observe a greater molecular difference between prosimian and carnivore than between carnivore and human. This prediction has been tested (*7*) by using a series of antiserums to the albumins of several of the Carnivora to measure the immunological distances to various primate albumins. We found (Table 1) that the prediction was not fulfilled. Indeed, the immunological distance to human albumin (162 units) was greater than that to any prosimian albumin: slow loris, 125; lemur, 135; tarsier, 137; tree shrew, 156. Thus, there is no correlation between the amount of primate albumin evolution and generation length, the two species with the strongest contrast in generation length (man and tree shrew) having albumins very nearly equidistant from our carnivore reference points.

The logical inference that must be drawn from these data in the context of the particular primate-carnivore relationship is that human albumin, along with those of the Old World monkeys (Table 1A), has changed somewhat more from the ancestral primate albumin sequence than have those of the prosimians, in spite of the undoubted increase in generation length among the higher primates. A more recent and hitherto unpublished study involving the quantitative precipitin technique and a larger variety of antiserums leads to a similar conclusion (Table 1B). And finally, antiserums to a series of New World monkey albumins indicate that the albumin of man or gibbon is no less changed from the ancestral catarrhine sequence than those of the Old World monkeys (Table 1C) (*14*).

Now neither the above arguments nor much of the supporting data are new, and this puts into a curious light the statement of Lovejoy *et al.* (*12*):

Table 1. Three tests for the occurrence of an evolutionary slowdown in the hominoid lineage. Abbreviations: ID, immunological distance; MC'F, micro-complement fixation.

Primate albumin tested	Mean ID
A. MC'F test with antiserums to the albumins of four carnivore species*†	
Homo sapiens	162
Macaca mulatta (rhesus monkey)	166
Ateles geoffroyi (spider monkey)	149
Nycticebus coucang (slow loris)	125
Lemur fulvus (brown lemur)	135
Tarsius spectrum (tarsier)	137
Tupaia glis (tree shrew)	156
B. Precipitin test with antiserums to the albumins of seven nonprimate species‡	
Homo sapiens	169
Hylobates lar (gibbon)	169
Macaca mulatta	169
Aotus trivirgatus (owl monkey)	150
Cebus capucinus (capucin monkey)	169
Nycticebus coucang	143
Lemur fulvus	150
Tupaia glis	163
C. MC'F test with antiserums to the albumins of five New World monkey species†§	
Homo sapiens	55
Hylobates lar	53
Macaca mulatta	53
Presbytis entellus (langur)	52

* The four carnivores were *Hyaena, Genetta, Ursus,* and *Arctogalida.* † Each purified albumin from a carnivore, bat, or New World monkey was injected into a different group of four or five rabbits, and the antiserums were obtained after a 3-month immunization period (*5*). Antiserums were mixed in reciprocal proportion to their micro-complement fixation titers and used as described elsewhere (*11*). The term immunological distance is used in the sense described by Sarich (*5*). For albumins, ID units are now known to be approximately equivalent to the number of amino acid differences. ‡ The seven nonprimates were *Hyaena, Genetta, Ursus, Pteropus, Tadarida, Antrozous,* and *Hipposideros.* Percentage cross-reaction in the precipitin test was converted to ID by means of a calibration curve established in (*11*) and by Sarich (unpublished results obtained with albumin immune systems). § The five New World monkeys were *Cebus, Ateles, Aotus, Saguinus,* and *Pithecia.* Results consistent with this, but obtained with other antiserums, were published in (*4*).

"Recalculation of the time of divergence of the Pongidae and Hominidae after correction of immunological distance by inclusion of generation length yields minimum dates of approximately 14 million years ago." Its relevance depends on a demonstration that generation length does indeed affect rates of protein or DNA evolution. Lovejoy *et al.* did not even attempt such a demonstration and, further, ignored extensive data (such as those in Table 1, A and C) which we previously presented (*4–7*) and which seem to show no generation length effect. However, a more quantitative assessment of the model of Lovejoy *et al.* suggests that the matter is not all that simple.

The equations of Lovejoy *et al.* can be used to calculate the number of generations that have accrued along various primate lineages (Fig. 2). Such an analysis demonstrates that the particular assumptions about generation length made by Lovejoy *et al.* result in a large proportion of the total number of generations along any primate lineage (other than that of the tree shrews) being seen as having accumulated in its early history. A hominoid lineage, for example, would have gone through 10.5×10^6 generations in its first 15 million years and only 7.8×10^6 generations in the subsequent 50 million years. Yet any influence of generation length on the amounts of amino acid or nucleotide substitution among the nontupaiid primate lineages would be limited to this latter period. The effect of these strictures on our rate tests can be illustrated by considering the average immunological distance of 122 units (*7*) from prosimian albumin to higher primate albumin; according to the figures of Lovejoy *et al.*, this should be distributed with 71 units along the prosimian lineage and 51 along the higher primate lineage (*15*). This distribution is within 1 standard deviation of the 61 units along each lineage predicted by the astronomical time model; thus, even for the most extreme generation length contrast among the nontupaiid primates, the predictions of the two hypotheses are virtually indistinguishable statistically.

However, the same assumptions about generation length that produce this seeming impasse also require the tree shrew lineage to have gone through five times as many generations in the last 65 million years as has the average higher primate lineage [107×10^6 compared to 19.3×10^6 (see Fig. 2)] and, therefore,

to have accumulated at least five times as many albumin amino acid substitutions. Now it has already been shown that since lemurs and higher primates last shared a common ancestor about 75 immunological distance units of change have occurred along an average higher primate albumin lineage (*7, 16*). In that same time span, though, the tree shrew albumin lineage should then have accumulated more than 350 units of change. Yet the carnivore and bat data of Table 1, along with the mean immunological distance of 128 units (*7*), from tree shrew albumin to higher primate albumin allow no more than 60 units of change along the tree shrew albumin lineage (*17*).

In addition, a similar study has shown the albumins of species with short generation lengths, such as rats and mice, to be no more distinct from those of a series of carnivore albumins than are those of man and chimpanzee (*18*).

If no effect of generation length on rates of genomic evolution can be seen in cases where it should have been most evident, then it would appear futile to further pursue the generation length hypothesis. We conclude that this hypothesis, along with the placentation hypothesis of Goodman (*19*), has been put forward to explain a phenomenon not yet shown to have an existence independent of certain assumptions concerning the primate fossil record. The basic problem would appear to be an unnecessary reliance on the absolute rate approach, for it is only if we accept current interpretations of the primate fossil record, which suggest a divergence time of at least 15 to 20 million years between man and the African apes (*14, 20*), that the conclusion of a marked slowdown in rates of albumin, hemoglobin, and DNA evolution in the evolution of the ape and human lineages since that divergence inevitably follows. Any number of selective variables are then available to explain this "slowdown," for example, generation length (*10, 12*) or increased placentation in the higher primates (*19*). Whether or not the slowdown actually occurred, however, is a matter subject to relative rate tests—and these have consistently failed to show it.

The obvious corollary is that the starting assumption of a time of divergence of at least 15 to 20 million years between man and the African apes is in error, as has been pointed out on numerous occasions (*6, 7*), and it remains difficult if not impossible to reconcile the growing body of protein and nucleic acid data with a divergence time of more than about 5 million years ago between man and African apes.

Why generation length does not seem to affect rates of protein and DNA evolution is a question that currently admits of no unequivocal answer. We do, however, call into question the assumption that mutations are, in the main, errors of DNA replication, and propose the following hypothesis: most protein and nucleic acid evolution is due to the random fixation of nucleotide substitutions which occur as a result of processes other than DNA replication, that is, presumably continuous ones such as DNA repair (*21*). We further suggest that selective control of any such processes must be very finely tuned indeed, for it provides a positive regulation of the input of variability and, therefore, of the scope of populational response to changing environmental conditions.

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13. It might be argued here that in assuming primate monophyly and not specific divergence times within the primates we are rejecting one paleontologically based conclusion (the times involved) in favor of another (the cladistic relationships of the taxa involved) with no substantive reason for making such a choice. The answer lies in the knowledge that cladistic conclusions are generally based on a summation of difference-similarity judgments made on a variety of anatomical characters which can, to a large extent, be interpreted evolutionarily without reference to the fossil record.
14. T. Uzzell and D. Pilbeam [*Evolution* **25**, 615 (1971)] have criticized this type of rate test as inherently insensitive. While the point they make—that phenetic underestimation of phyletic distance would reduce the sensitivity of rate tests—is certainly valid, this does not get at the question of the degree of insensitivity introduced. We have dealt with this matter and point to the actual data which do indicate some albumins as more changed, and some as less changed, than others and, more important, show such differences in a quantitatively consistent fashion. For example, *Aotus* albumin is less changed than that of *Cebus*, and by about the same amount, whether the reference albumins be those of the catarrhines, prosimians, carnivores, or bats [(*7*) and this report]. Similarly, *Ursus* albumin is less changed than those of other canoid and pinniped carnivores, and again by about the same amount, whether the reference albumin is canoid, pinniped, feloid, or primate (*5*).
15. In these comparisons we have assumed that the number of amino acid substitutions along a lineage is proportional to the number of generations along it. According to (*12*), the number of generations along a nontupaiid prosimian lineage in the last 65 million years is 27.2×10^6 and the corresponding figure for an average higher primate (catarrhine) lineage is 19.3×10^6 (Fig. 2). The "expected" distribution is then $(27.2/45.5) \times 122 = 71$ for the prosimian lineage and $122 - 71 = 51$ for the catarrhine lineage.
16. If A represents the set of prosimian species (*Nycticebus, Lemur*, and *Tarsius*) in Table 1, B the set of higher primate species, and C the set of carnivore and bat species in the table, then with the logic outlined in Fig. 1 one can calculate the amount of albumin evolution that has taken place along the average higher primate lineage, as follows. The mean A-B albumin distance is 122 units, the A-C distance is 138 units, and the B-C distance is 166 units. Thus $a + b = 122$ and $a - b = -28$; therefore $a = 47$ and $b = 75$. It is, of course, possible that this evolutionary conservatism of lemuriform, lorisiform, and tarsier albumins has a selective rather than a statistical basis. One could, for example, note that nocturnal primates (*Nycticebus, Loris, Galago*, some *Lemur* spp., *Tarsius, Aotus*) tend to have "conservative" albumins, and look for a selective factor associated with such an existence. Or one could argue that, as "higher" primates tend to have somewhat more changed albumins, the prosimian-anthropoid transition briefly selected for more rapid albumin evolution. What one cannot show is any slowdown in anthropoid albumin evolution.
17. In this case A can be the tree shrew, and B and C as in (*16*); $a + b = 128$ and $a - b = -7$ (from Table 1). Thus $a = 60$ and $b = 68$.
18. The generation time hypothesis was originally prompted by the very large *Rattus-Mus* difference shown in DNA hybridization studies, coupled with a presumed recent divergence between the two genera (*9, 10*). The corresponding *Homo-Pan* difference is some tenfold smaller, and the albumin differences in the two pairs are approximately in the same proportion. Nevertheless, if an experimental approach identical to that presented here is used, rat and mouse albumins cannot be seen as more changed than those of man and chimpanzee [V. M. Sarich, *Biochem. Genet.* **7**, 205 (1972)].
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22. We thank S. L. Washburn, B. N. Ames, and C. O. Lovejoy for helpful discussions. Supported in part by NSF grants GB-20750 and GB-13119 and by a fellowship award to A.C.W. from the J. S. Guggenheim Memorial Foundation. *Pteropus* and *Hipposideros* were collected on the *Alpha Helix* expedition to New Guinea in 1969, and *Antrozous* and *Tadarida* were collected locally by P. Leitner.
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Fragile Ecosystems

In their plea for the preservation of large areas of tropical rain forest, Gómez-Pompa *et al.* (*1*) emphasize that "if we wait for a generation to provide abundant evidence [of the vulnerability of the rain-forest ecosystem], there probably will not be rain forests left to prove it." Having been interested for several years in the problem of mass extinctions (*2*), we should like to verify the soundness of their view from the paleontologist's perspective. In a sense, the documentation which they seek in order to elicit massive international action toward conserving the tropical biota has been provided by the fossil record. We have noted previously, following Sanders (*3*), that in its high diversity the tropical ecosystem is analogous to offshore marine assemblages which underwent radical reorganizations in community structure, especially at the end of Devonian and Permian time. Nearshore assemblages of lower diversity—perhaps analogous to the temperate-zone forests which have been resilient to intensive and extensive agricultural exploitation —have been much more stable throughout geologic time.

By contrast, as Gómez-Pompa *et al.* indicate in their first paragraph, neontologists have tended to believe that the more diverse a community is, the more stable it should be—at least if stability is defined in terms of resistance to the effects of fluctuations in the number of individuals of a constituent species (such fluctuations comprehending also the total disappearance of a species) (*4*). We have postulated (*2*) that mass extinctions primarily affecting species belonging to assemblages of high diversity came about because these taxa, having lived for many millions of years in a predictable environment where there was little selection for genetic or physiological flexibility, were vulnerable to changes in the physical environment, such as the shrinking of the seas during periods of mountain building (*5*). Our postulate of loss of genetic polymorphism has recently been challenged by workers who have found that species of several different phyla from the deep sea, one of the most diverse ecosystems and apparently one of the most predictable environments on the globe, show as high a level of genetic variability as do species from less predictable environments (*6*). [Other studies, however, indicate that genetic variability among species of the Class Bivalvia decreases along a gradient from less predictable to more predictable environments (*7*).] Among tropical plants, vulnerability to physical stress appears to result primarily from the loss of adaptations for dispersal and dormancy. Identification of the exact cause of mass extinctions is, however, comparatively unimportant in the present context; our primary purpose is to reiterate that, although the activities of man which destroy tropical rain forests are different from the natural forces that destroyed diverse marine biotas of the geologic past, the effects are likely to be the same. We believe that the lesson of earth history is that highly diverse ecosystems in physically predictable environmental regimes are in fact very fragile and require respect and care if they are to be preserved.

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