

Morphological complexity increase in metazoans

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Abstract.—The number of cell types required for the construction of a metazoan body plan can serve as an index of morphological (or anatomical) complexity; living metazoans range from four (placozoans) to over 200 (hominids) somatic cell types. A plot of the times of origin of body plans against their cell type numbers suggests that the upper bound of complexity has increased more or less steadily from the earliest metazoans until today, at an average rate of about one cell type per 3 m.y. (when nerve cell types are lumped). Computer models in which increase or decrease in cell type number was random were used to investigate the behavior of the upper bound of cell type number in evolving clades. The models are Markovian; variance in cell type number increases linearly through time. Scaled to the fossil record of the upper bound of cell type numbers, the models suggest that early rates of increase in maximum complexity were relatively high. The models and the data are mutually consistent and suggest that the Metazoa originated near 600 Ma, that the metazoan “explosion” near the Precambrian/Cambrian transition was not associated with any important increase in complexity of body plans, and that important decreases in the upper bound of complexity are unlikely to have occurred.

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Introduction

The complexity of an object, it has been suggested by Hinegardner and Engleberg (1983), may be defined as the size of its minimum description. The factors that influence the size of such a description have been reviewed by McShea (1991). The description must become larger as order within the object decreases (a jumbled pile of bricks is far harder to describe than a well-built brick wall) and as the redundancy decreases (a pile of twenty types of bricks is harder to describe than a pile of identical bricks). The jumbled pile is more complex than the ordered wall, and especially so if the items in the pile are heterogeneous. Thus, order and homogeneity are the opposites of complexity (Wicken 1979).

All organisms are quite complex, so they must not be well ordered. They are, however, highly organized (Wicken 1979; McShea 1991), meaning in part that they can exercise certain functions that could not be performed by an unorganized entity of similar complexity. Both the complexity and organization of organisms derive ultimately from their evolutionary histories, but proximally from the process of development. A human is more

complicated than a sponge, and one can imagine a scale of complexity by which all organisms might be measured. Here we use cell type number as an index of morphological complexity, and track the course of the upper bound of complexity throughout metazoan history by associating the times of origin of metazoan body plans with the cell type number estimated for each plan. We then use stochastic models to attempt to understand some of the evolutionary implications of the empirical estimates of complexity change.

Cell Type Number as an Index of Morphological Complexity

All metazoans possess multiple cell phenotypes; cell differentiation is a basic feature of the kingdom. Hall (1992) has emphasized that it is at the cellular level that the common link between evolution and development is forged. Kauffman (1993) has argued effectively that cell types are important units of evolution; in his view, cells represent constrained patterns of gene expression, and he hypothesizes that cell types are discrete. The morphological complexity of metazoans is organized hierarchically, with cells usually re-

garded as the basic building blocks. In more complex metazoans, several cell types are usually associated in a tissue, one or several tissues may be associated in an organ, and one or several organs in an organ system, the entire organism being composed of a number of organ systems. In less complex metazoans there may be no real organ systems, and in some cases organs consist of only a single tissue that is composed of only one cell type. Some authors have suggested using aspects of this compositional hierarchy—the number of levels, the number of units on a given level, or more commonly what is essentially a combination of interrelations among some units on various levels—to judge complexity (e.g., Stebbins 1969). The number of cell types, which is multiplied as the number of different tissues, organs, or organ systems is increased, is clearly very sensitive to differences in morphological complexity, and therefore has been suggested by several workers as a particularly useful metric of an organism's complexity (see especially Bonner 1965, 1988; also Sneath 1964; Raff and Kaufman 1983; Valentine 1991, *in press*). While cell type numbers are probably not an accurate interval measure of complexity, as they do not take into account the configuration of the various hierarchical levels, they should nevertheless provide a useful ordinal index. Using cell type numbers has the further advantage that it is possible to estimate the approximate geologic time when a given level of complexity was reached in a number of cases. Thus, for both theoretical and practical reasons the cellular level would appear to be the best choice available to indicate morphological complexity.

Some workers have believed that the measure of complexity should be reduced to an expression associated with the information content of the genome or egg (Hinegardner and Engleberg 1983), such as indicated by the amount of DNA (Sneath 1964) or the number of genes. By such a standard there would be no difference between the complexity of a fertilized egg and of the adult organism that it produces; both contain the same genetic information. Hinegardner and Engleberg (1983) explicitly defend such a stance, admitting only a slight complexity increase dur-

ing development. One of their arguments is that development is merely an ordering process, which does nothing to increase complexity.

We take quite a different view, i.e., that development should be regarded as an organizational process that precisely permits an increasing morphological complexity. Although organisms are not well ordered, they are highly organized (see McShea 1991). Indeed, organisms must necessarily possess a high level of organization (Saunders and Ho 1976; Riedl 1977), which most likely arose originally along fitness pathways defined by natural selection. The level of complexity of an organism may be partly a result of the information content of its genes, but that does not endow the genes with the complexity of the organism. As development proceeds cell types become differentiated, distinctive tissues and organs appear, and thus, the heterogeneity of the units increases. The complicated geometry assumed by the developing cell types, tissues, and organs is far from well ordered, though it is certainly organized. Thus, development produces complexity in the structures that are realized. The configurational information resides with or depends upon epigenetic processes as well as it does upon information coded in the genes. While the epigenetic events may be imminent in the development of the egg they are not part of the instructions; genes do not contain a complete physical description of an organism (Apter and Wolpert 1965). Perhaps the chief differences in complexity among metazoan body plans arise through epigenetic components of development.

As development proceeds in a metazoan, then, cells differentiate in a pattern that is controlled through an interplay of genetic and epigenetic processes. Different genes are expressed (or are expressed at different times or in different doses) in different cells to give rise to a number of distinctive cell phenotypes—the cell types with which we are concerned here. Many cells that have very similar phenotypes, and would be classified as the same cell type, have nevertheless been subjected to different control signals and have had distinctive transcriptional histories, so

that precisely the same genes are not necessarily expressed in all members of a given cell type population (see Davidson 1990). Thus, in using the level of the cell type to infer complexity we are indisputably lumping cells that have biochemical and no doubt functional differences. As we are not in fact attempting to measure complexity itself, but are using cell type numbers simply as an index of complexity, this may not present a major problem so long as there is a standard degree of lumping and splitting of cell types to permit a practical ordering of complexity among organisms with different body plans.

Cell Type Numbers in Metazoan Clades

Histologists have made few estimates of the total cell type numbers of organisms, but they have established a tradition of describing cell phenotypes that has permitted comparative histological studies that have added much to our understanding of comparative anatomy and physiology. Systematists and developmental biologists, interested in the evolution of differentiation and complexity, have been able to draw upon this histological literature to estimate cell type numbers for a variety of organisms across a number of phyla. Sneath (1964) plotted estimates of cell type number against measures of DNA content of the haploid genomes of a range of organisms including metazoan phyla, and the two appeared to be roughly proportional; Kauffman (1971, 1991) has cartooned a similar relationship. However, Raff and Kaufman (1983) made a similar comparison and emphasized that there is a great range of DNA content in the haploid genomes of higher animal taxa, and that (although there is a range of cell type numbers in higher taxa as well) there is little or no correlation between the amount of DNA and the cell type numbers of metazoan phyla. Bonner (1965) plotted estimates of cell-type number against maximum body lengths of some animal and plant groups; he showed that the rate of increase in cell type number outpaced the rate of increase in maximum body size, and that animals have more cell types than plants, size for size.

Here we present a plot of the estimated somatic cell type numbers found in selected

metazoan taxa against the estimated time of appearance of those taxa in geologic time (fig. 1), using the phenotypic criteria commonly employed by histologists to differentiate among cell types (see for example the cell types recognized by authors in Harrison and Bogitsh 1991; Harrison and Westfall 1991; Harrison and Ruppert 1991). As the presence of larval cell types is variable even among congeneric species, e.g., depending upon whether or not development is direct, we restrict our cell counts to types in adults. The absolute dates of fossiliferous horizons are not often known with much exactitude, particularly for the Paleozoic and Precambrian; we have used the latest estimates available. The times of origin of the various taxa are also not exactly known, even in terms of the geological column. However, these times are usually constrained in some way—certainly by the earliest appearance of the taxon in the fossil record, and also by the lack of appropriate ancestors, or the lack of the kinds of trace fossils appropriate to a taxon, or by the duration of gaps expected in the record of taxa with a given probability of fossilization, and so on—so that our assigned dates are probably reasonably close considering the scale of the plots.

Finally, we are interested in tracing the history of the upper bounds of morphological complexity as measured by cell type number. For this purpose, the first appearance of a clade, the cell type numbers of which can be inferred from data on living forms, provides a unique opportunity to associate the first indication of a given level of complexity with a geologic date. To evaluate and supplement the available data on cell type numbers within clades, of which Sneath's (1964) has proven to be the most consistent with modern ultrastructural studies, somatic cell type numbers were tallied from the literature for those organisms for which reasonably complete histological descriptions exist (see Appendix). Nerve cell varieties were not counted, following Alberts et al. (1989). Within some groups there is a significant range of cell type numbers. Ideally for our purposes, each group should be represented by its primitive cell type number, which can then be plotted at

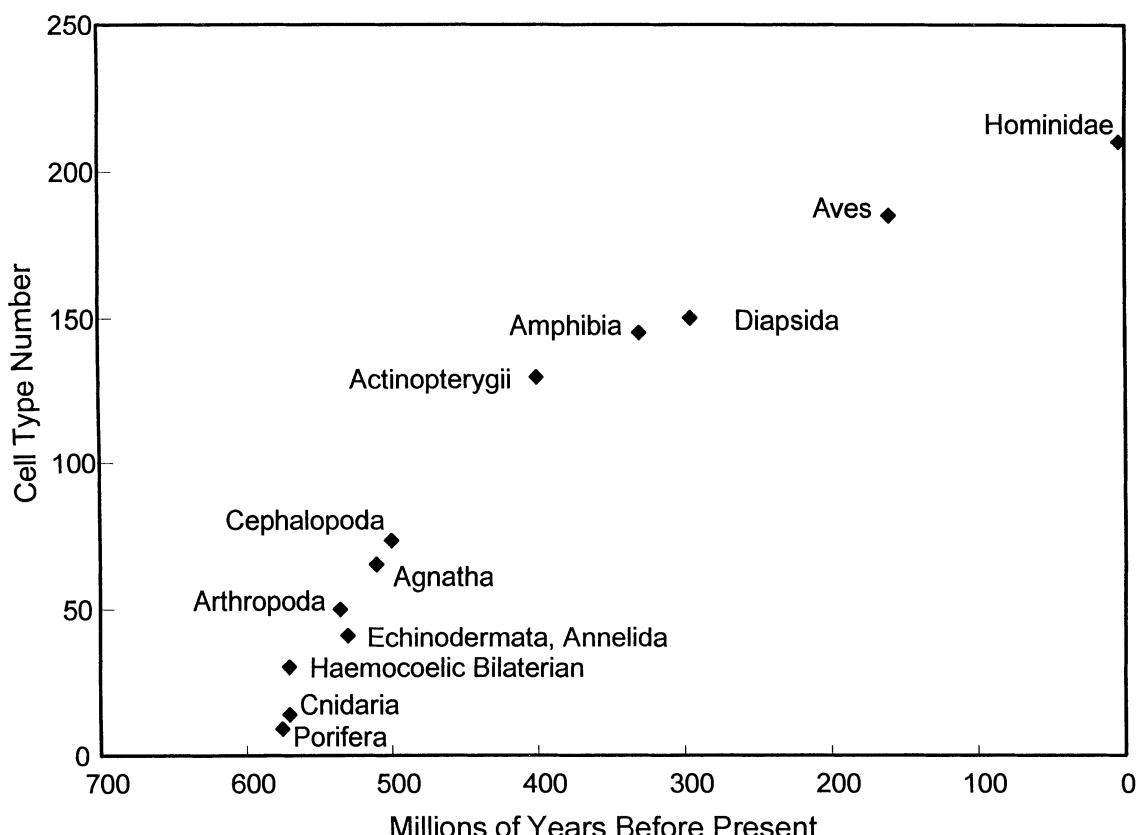


FIGURE 1. Estimated cell-type numbers of primitive members of selected metazoan taxa, inferred from counts of cells in living individuals, plotted against the estimated time of origin of the body plan of each taxon. Only taxa that are believed to have been rather near the upper bound of cell-type numbers when originating are included. For sources on which cell-type number estimates are based, and sources for ages, see Appendix.

the estimated age of origin of the body plan; we judge this primitive number from descriptions of the more primitive living forms within each group. Thus, although we have labeled the points in figure 1 with the familiar names of higher taxa, the estimates of cell type numbers can be thought of as representing the most primitive families in each taxon, those that founded the clades that the taxa represent. There is in effect an entire tree of life between the upper bound and the floor, and the plot of cell type number of families that founded many important clades lie within this region rather than at the upper bound. However, estimates of cell type numbers for taxa that are significantly below the upper bound of cell type numbers are not plotted here.

Among the earliest metazoan fossils, ap-

pearing probably near 570 Ma are trails that are generally interpreted as having been formed by bilaterians. Many Precambrian trails were formed by organisms measured in centimeters and capable of furrowing sediment and leaving corrugated trails; the trails sometimes have bordering dikes, and sometimes contain pellets, presumably of fecal origin (for illustrations of many trace types see Crimes 1992). Such trails indicate organisms more complex than flatworms (see Valentine 1991; Fedonkin and Runnegar 1992), with through guts and hydrostatic skeletons, suggesting a blood vascular system with a hemocoel. A bilaterian of that grade is estimated to require a minimum of about 30 cell types (Valentine 1993), thus anchoring one end of the plot. At the other end are human beings, with about 210 cell types (Alberts et al. 1989),

which we plot as the family Hominidae. This taxon has not founded a major clade morphologically, but it has the highest number of cell types recorded and we know its approximate time of origin, so it meets our criterion of representing the upper bound of cell type numbers. More estimates of cell type numbers among complex taxa, such as both primitive and advanced mammals and arthropods, are much needed, and should lead to improved estimates of the character of the upper bound. When such estimates are forthcoming they will form tests of the history of the upper bound as hypothesized here.

Considering the uncertainties attached to all of the estimates plotted in figure 1, restraint in interpretation is clearly indicated. It does seem that maximum cell-type number as tallied here (and thus morphological complexity by this index) has increased, at an average rate of about 0.32 cell types/m.y., let us say 1 cell type every 3 m.y., from the early traces of metazoans. This is in direct contrast to the speculations of Hinegardner and Engleberg (1983) and Gould (1985) that metazoan complexity has not much increased since the Cambrian. From the plot, the rate of increase could have been relatively linear. Other theoretical possibilities are that the rate of increase could have been accelerating, or it could have been decelerating, perhaps to some asymptote, or it could have been quite irregular with frequent plateaus or even with negative intervals. Fisher (1986), speculating on the possible causes of the perceived complexity increase in metazoan morphology, pointed out that even if complexity followed a random walk in each lineage, the mean complexity of all lineages should increase through time because there is a floor (or "reflecting barrier," McKinney 1990) below which metazoan complexity may not fall—a floor of two cell types in the present case. McShea (personal communication 1993) has noted that the change in cell type numbers in lineages can be modeled as a Markov process (see Raup 1977; Berg 1983), and that as such a process unfolds the variance increases linearly, and he further suggested that the upper bound of cell type numbers might increase proportionately to the square root of

time without the involvement of any special forcing mechanism. The applicability of such a process to interpreting the cell type number plot can be investigated by computer modeling.

Modeling Cell Type Increases as a Stochastic Process

The process of evolution entails the characteristic that each change is constrained by the prior states of the system; the character of a lineage is strongly contingent upon its history. Sequences of states of this sort are referred to as Markov chains. Raup (1977) has championed the use of probabilistic computer models to mimic Markov processes in attempting to recreate patterns seen in the fossil record. Here we use computer models to explore the conditions that would lead the upper bound of complexity to increase, assuming the underlying process to be Markovian. The model generates a series of steps or states in a number of evolving lineages, treated as a time series. At each step, each lineage may gain one cell type, lose one cell type, or remain unchanged, at random, except that there is a floor of two cell types, viewed in the model as the minimum complexity for metazoans, beneath which lineages are not permitted to fall. Lineages that attempt to decrease below two cell types are treated as if they were unchanged. We report on four models run under these rules. The models do not represent explicit attempts to simulate evolutionary history; the focus in the models is on understanding the behavior of the upper bound of cell type number under a variety of conditions.

One model (Model I) was started at time zero with an initial condition of 2000 lineages, each with two cell types, and was run for 4000 iterations. At each step the cell type number increased, decreased, or remained constant in each lineage; the probabilities of each of these events were the same at each step in all lineages, except that the lineages at two cell types could not decrease to one. The behavior of the upper bound of cell type number, achieved by the most complex lineage (by definition) present at each time step, is shown in figure 2A. The variance of the set

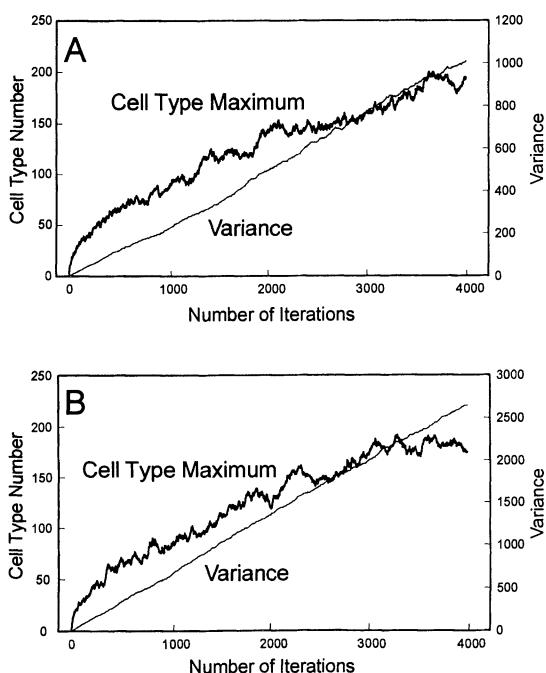


FIGURE 2. Computer models of the upper bound of cell-type numbers. A, Model I, 2000 lineages of 2 cell types each begin at time zero and proceed for 4000 steps. At each step each lineage may increase or decrease by one cell type, or remain the same, except that no lineage may fall below two cell types. Chances of increase, decrease and stasis each set at one-third per step. B, Model II, 2000 lineages, same rules as A except that lineages are permitted to fall below two cell types (and may have negative cell-type numbers).

of lineages increased linearly; this was true in all four models. However, the upper bound did not increase linearly; instead it approximated a square root function, with the rate of increase in maximum cell type number highest in the earliest steps and declining thereafter. The explanation for this pattern is straightforward. The maximum rate of increase for the upper bound is one cell type at each step. During the earliest steps, more lineages have the opportunity to achieve the maximum cell type number; the lineages are, so to speak, crowded between the bound and the floor. The first few steps are inevitably characterized by increases of one cell type each per step. As the steps continue, the number of lineages at or near the upper bound become reduced as the variance increases, and the probability that at the next step no lineages at the bound will increase, or that all lineages at the bound will happen to decrease

in cell types, becomes steadily larger. The rate of increase of the upper bound therefore begins to fall behind the one-type-per-step rate, and continues to decline while displaying the stochastic variation inherent in the model.

Model II was run under the same conditions as Model I but without the floor, so that one option for the initial step (and subsequent steps) was downward, i.e., negative cell type numbers were permitted. Again, during the earliest steps the upper bound increased at the fastest rate, but the rate decreased more rapidly because fewer lineages remained near the bound (fig. 2B). It is interesting that the absence of a floor, which dilutes the density of lineages by one half, leads only to a minor lowering of the early rate of increase of the upper bound.

Models III and IV were run to examine the effect of beginning the metazoans with a single lineage at two cell types, and then permitting a diversification from this founder to a level of 2000 lineages, while the same rules for cell type number change applied to each daughter lineage as it appeared (fig. 3). These models involved a floor but no initial crowding. In Model III the rate of diversification of the lineages was regulated by a logistic ($r = 0.014$, $K = 2000$), and thus, there was a number of early steps at very low lineage diversities (when variance in cell type number remained very low), followed by higher rates of increase in lineage numbers (when rising variance became stochastically constant), the rate of increase declining to zero at a lineage number of 2000. In Model IV, lineage diversification was forced to be linear until 2000 lineages, after which the lineage number was constant, in order to test whether the steeper part of the logistic curve of lineage richness affected the behavior of the upper bound of cell type number. In both of these models the cell type number rose more rapidly at first despite the paucity of lineages in early steps, and then the rate of increase declined as in Models I and II.

Implications of the Models for Metazoan Complexity Increases

There are several features revealed by the models that might be applied to an interpre-

tation of the increase in cell type numbers indicated in figure 1. The variance of cell type numbers within all lineages tended to increase linearly over time except when low numbers of lineages were maintained for a number of steps, as predicted by the mathematics of such a Markov process. Furthermore, as Fisher (1986) and others have suggested, the maximum cell type number trended upward even when the chances of increase or of decrease were symmetrical, and the upwards trend appears to be parabolic as follows from McShea's hypothesis (personal communication 1993, see above).

Owing to the stochastic nature of cell type generation, the rise in numbers is subjected to numerous local accelerations and even some local declines, giving the upper bound a saw-tooth appearance as it generally rises. Thus, to the extent that this process reflects evolutionary history, complexity increases would have been variable in rate and occasionally would have been interrupted by decreases, within a generally rising trend. However, when lineage density between the floor and the upper bound is greatest, stochastic variation is least, and during biotic history the upper bound has increased only from two (or so) to 210 by our standard of cell type numbers, while the number of lineages has increased from some very low number to millions. The density of lineages between the floor and the upper bound has thus greatly increased, and accordingly the rise of the upper bound of cell type number would be expected to have been significantly smoother than in our models, to the extent that the behavior of the models indicates the behavior of that process.

The features of the model curves can be used to suggest whether the actual history of the upper bound of cell type increase, so far as it can be inferred, is simulated by the models. In figure 4 the inferred historical points of the upper bound of cell type numbers from figure 1 are overlain by a curve of cell type number increase generated by Model III. The average of five model runs was scaled to the history of maximum cell type numbers by being pinned at 30 cell types at 570 Ma and 210 cell types at 5 Ma, an average rate of in-

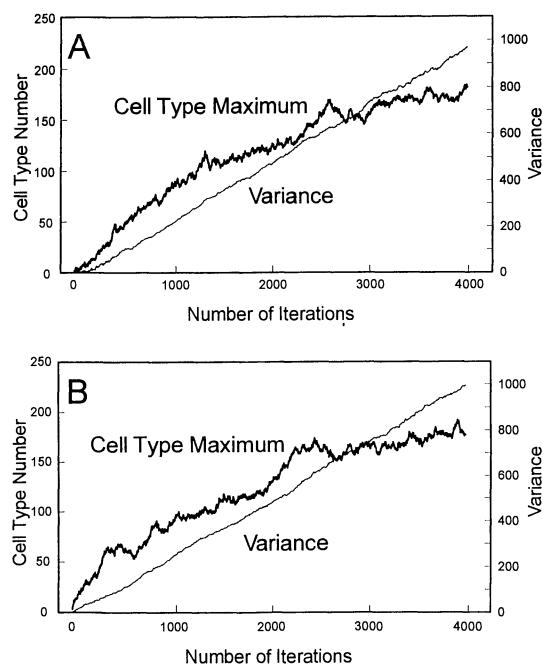


FIGURE 3. Computer models of the upper bound of cell-type numbers in diversifying clades. A, Model III, one lineage at time zero diversifies logically ($r = 0.014$, $K = 2000$) with rules for increasing, decreasing, or static cell-type numbers the same as in figure 2A and applying to each lineage as it is founded. B, Model IV, one lineage at time zero diversifies linearly to 2000 lineages, with rules for increasing, decreasing, or static cell-type numbers the same as in figure 2A.

crease of 3.14 cell types/m.y. from the origin of metazoans. As the model has 4000 steps, there was a rate of increase, from two cell types, of 0.053 cell types/step. Although the history of life had but one run, the model is based on a maximum of only 2000 lineages, and accordingly any single run displays more stochastic variation of the upper bound than would have occurred during the history of tens of thousands to millions of model lineages. Still, there have not been 2000 major metazoan clades, and it may be partly at the level of major clades, rather than at the level of individual lineages, that complexity, and therefore cell-type numbers, can best be summarized. The average that we use somewhat smooths the model curve but, as can be seen from inspection of single runs in other figures, does not otherwise misrepresent the model behavior, and it preserves some representation of a stochastic process. If we av-

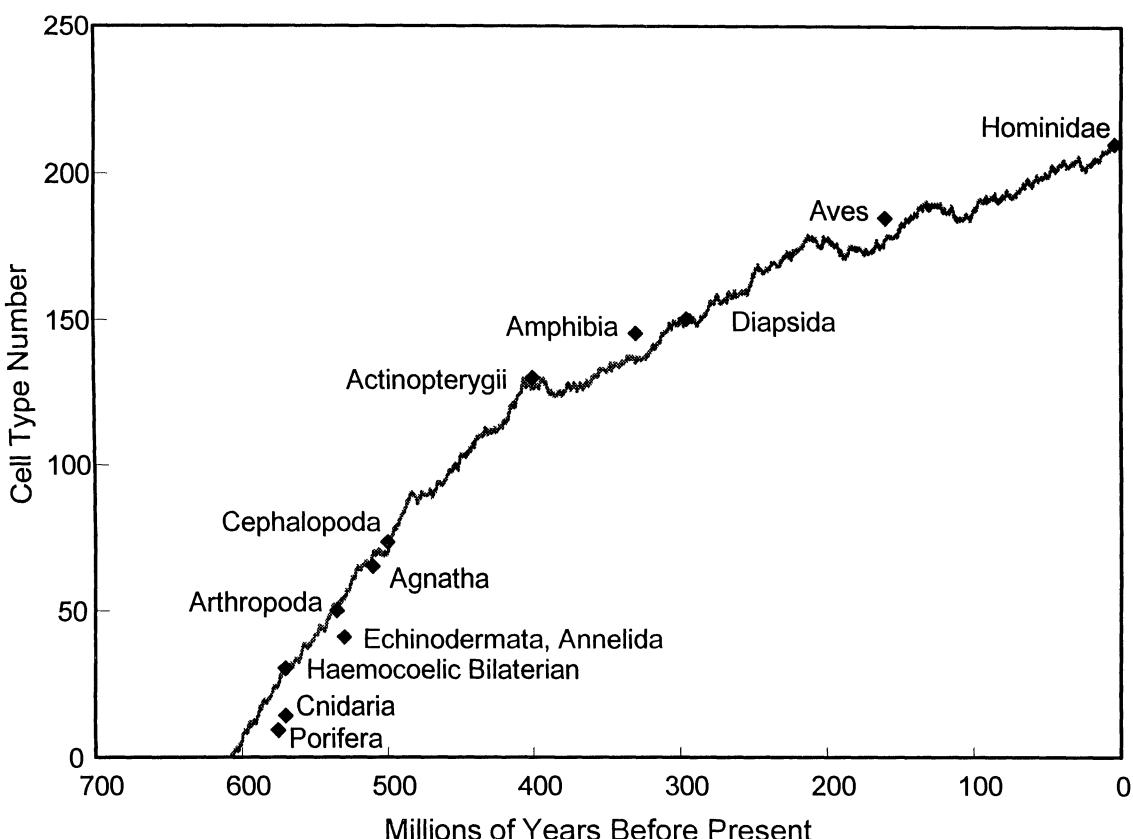


FIGURE 4. Estimated time of origin of organisms with cell-type numbers near upper bound (from fig. 1) and curve of the average of five computer runs of Model III (fig. 3A), pinned to points at 30 and at 210 cell types. Note that the early portion of the curve suggests a late origin for the metazoans, that the Precambrian/Cambrian transition is not marked by an important increase in cell-type numbers, and that there are no obvious important downward trends.

eraged a large number of runs, on the other hand, the model curve would become perfectly smooth.

According to the models, the origin of metazoans should have been followed by a relatively rapid rate of increase in cell type number, a rate that would have been higher the more rapid was the rate of initial increase in metazoan taxonomic diversity (richness). During the Phanerozoic, when there is empirical evidence of rates of diversification to be found in the marine invertebrate fossil record, there is a tendency for the more rapidly diversifying lineages to become dominant when a fauna is faced with numerous adaptive opportunities, such as during the initial diversification of higher metazoans early in the Paleozoic and, probably, in the early Mesozoic following the massive Perm-

ian/Triassic extinction (for examples see Valentine et al. 1991). The rationale is simply that, when faced with empty "adaptive space," the more rapidly speciating lineages are most likely to occupy a lot of it, while more slowly evolving lineages will not be able to exploit the opportunities as quickly. It would seem to be a plausible speculation that the origin of the Metazoa, a major new organizational grade, would have provided an opportunity for those lineages that happened to be the more rapid diversifiers to become common and thus to provide for an especially rapid early increase in metazoan richness.

There may have been another reason for a rapid initial increase in complexity among early metazoans, based on the large percentage change that the addition of a single cell type makes when cell type number is low.

While an increase from 39 to 40 or 41 cell types would not appear to be a change that might open new regions of adaptive space to an evolving lineage, a change from 2 to 3 or 4 might well have. Therefore, if selection is to be invoked as an important engine of complexity change, it would seem likely to have operated more strongly initially than later.

Whether or not selection promoted complexity, the models suggest that the early rate of increase in cell type number (for which we do not yet have fossil evidence) was likely to have been higher than the subsequent rate of increase, and thus, that the origin of the Metazoa occurred later than would be estimated by projecting backwards a rate of increase based on the Phanerozoic record. Using a linear projection from the Phanerozoic, the metazoans have been estimated to have originated near 680 Ma (Valentine 1993). This figure now needs to be revised downward, and is perhaps near 600 Ma. Thus, consideration of an early rapid increase in cell type number produces a prediction of the time of metazoan origin that is much later than commonly postulated (e.g., Durham [1970] and Runnegar [1982] estimated the origin of some living higher invertebrate phyla to be between 800–1700 Ma and 900–1000 Ma, respectively, and the origin of the metazoan kingdom would have to have been still earlier in each case).

A number of authors have ascribed evolutionary trends toward increased size or complexity to the “nowhere to go but up” situation when there is a floor (Maynard Smith 1970; Stanley 1973; Gould 1988; McKinney 1990); the trend was ascribed by Fisher (1986) and Gould (1988) to the increase in variance that would accrue through time. McShea (1992) also illustrated a rapid initial rise in the upper bound of a model variable that is rising above a floor, as is seen in the models of cell type number presented here. This relatively rapid early rate of increase of the upper bounds in the models implies that no single clade is likely to remain as the most complex (in cell type number) during the early stages of complexity increase. Rather, there is likely to be replacement of one clade by another at the upper bound of complexity,

assuming that there is a stochastic process of cell type addition or subtraction, and that there is a number of clades near that bound. When one clade finally achieves a significant lead in complexity, and other clades, formerly bidding to be most complex, happen to rise less rapidly or even to decline in complexity, then the track of the bound should become more variable, for it is now formed by a single clade. If metazoan history has followed some similar pattern, then it would not be surprising if, say, the arthropods contributed the most complex organisms in the Middle Cambrian, perhaps to be replaced at the upper complexity bound by mollusks (cephalopods), which might have been surpassed in turn by chordates, a group which then ruled the roost for the remainder of the Phanerozoic, though certainly with replacements within the group. The replacement of the most complex major clade should have been more common early in metazoan history.

There is no indication in the models that there should be a marked jump in morphological complexity at any particular time, except for the high initial rate of complexity increase. The Precambrian/Cambrian transition receives no support from either the available data or the models as a special time in the history of metazoan complexity rise. Aside from stochastic variations, the only notable change in the trend of complexity increase in the models is the *lowering* of the rate of increase during metazoan history. Although it is possible that the upper bound of complexity happened by chance to undergo a small upward fluctuation during the transition, there should have been a number of periods when such an event occurred, so that it would not have been a unique feature of the Cambrian diversification.

Not all new clades were near or at the maximum level of complexity of the times during the explosive appearance of body plans near the Precambrian/Cambrian boundary; e.g., early brachiopods are probably much less complex than early arthropods, yet both groups evidently appeared within the same stage. Furthermore, most new body plans were probably appearing at significantly less than the maximum complexities that they even-

tually achieved, as morphological adaptations to various environmental challenges or opportunities produced specialized structures in some descendant lineages. Probably each new Cambrian body plan that we see evolved from an ancestor that was just slightly less complex, and that we don't see. In the models, before one has, say, 40 cell types, an ancestor must have 39. While there is no definitive constraint of this sort in the evolution of cell differentiation, it is not unreasonable to assume that most increases in cell-type numbers have occurred gradually. There is no hint in the available data that such changes have gone on wholesale to produce major salutations in morphological complexity. This is not to say that the body plans of the Cambrian were not novel, just that they do not seem to have been very much more complex than their immediate antecedents; the complexity rise associated with the Cambrian explosion was probably simply part of the generally rising complexity of the time, slower than earlier Precambrian rates but faster than later Phanerozoic rates (fig. 4). This situation may well underly the abruptness and particularly the breadth of the metazoan diversification across the Precambrian/Cambrian transition, the breadth and rapidity of the diversification suggesting the creation of new morphogenetic patterns rather than the wholesale evolution of major new levels of tissue and organ complexity.

As we did not count nerve cell types and somewhat lumped cells into phenotypic categories, there is a question as to what the plot of figure 1 would look like if all cell types could be included. Certainly the numbers of cell types among more complex forms, chordates in particular, would be greatly increased. If that increase should be markedly disproportionate to the cell phenotypes categorized here for simpler forms, it would be evidence for a forcing mechanism, at least for aspects of complexity associated with the added cell types, such as neurological complexity. Relative cell type numbers among the simpler body plans would probably be little affected by complete cell data, and thus the inferences that we have drawn about the Cambrian explosion might prove to be little

biased. This is especially true because our object has been to examine the gross morphology of body plans, to which the cell phenotype categories contribute most directly. The information contents of genomes or of developmental programs of metazoans are very important but are not what we are attempting to investigate.

Although our data do not closely delimit the path of Phanerozoic complexity at narrow time intervals, there is no hint that maximum metazoan complexity ever suffered a major drop, that is, much of a setback other than those encompassed by stochastic variation, even though several mass extinctions and many regional extinctions are known to have punctuated Phanerozoic biotic history. It is possible that drops occurred in the upper complexity bound but are not revealed owing to the coarseness of the data; the Cretaceous/Tertiary extinction, for example, might have removed the most complex animals on earth at the time, though this is by no means certain, since mammals and birds survived. A reason for the persistence of complexity may be exemplified by the work of Foote (1992) on patterns of morphological diversity within evolving clades. Extinction events, unless they are selective against the morphological extremes, will not reduce morphological diversity by as much as the decline in taxonomic diversity, and if the extinctions are distributed over several events, morphological diversity may actually be permitted to increase even as taxonomic diversity falls. In the present case, this finding means that even large extinctions that greatly thinned the number of lineages, but that did not select against the more complex ones, would not necessarily greatly reduce the upper bound of complexity. Indeed, if the more complex clades were at all diverse they would tend to escape complete eradication (Raup 1975, 1981) and the surviving lineages would preserve the position of the upper complexity bound.

Conclusions

The behavior of the upper bound in stochastic models of change in cell-type numbers agrees well with the historical increase in the upper bound of numbers of categorized

cell phenotypes in metazoan body plans as inferred from the fossil record. The agreement suggests that the opportunities for increase in body plan complexity may have been random. The increase in the upper bound of morphological complexity implied by the data and by the behavior of the models is particularly important, for complexity increases are not simply quantitative but permit qualitative changes also, as particularly well exemplified by the evolution of intelligence. The behavior of the median complexity level is not as meaningful, its chief significance being statistical rather than biological.

The rate of increase of the upper bound of complexity may have been greatest early in the evolution of the metazoans, which may have originated near 600 Ma, far later than usually supposed. Perhaps the rate then declined across the Proterozoic/Phanerozoic transition, with no unusual increase in complexity during the Cambrian radiation of metazoans, although there were probably occasional replacements of the clade that lay at the upper complexity bound at least through the Cambrian. Chordates achieved a position at the upper bound of metazoan complexity probably during the Ordovician or Silurian and once attaining it did not relinquish it. Extinctions have rarely if ever reduced the maximum complexity, partly because extinctions do not seem to have been selective on clades with high complexities, and partly because the most complex clades have been diverse and therefore not easily eradicated.

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Appendix

The following are sources from which minimum cell type numbers were calculated, and from which the ages of origin of taxa were inferred, for a number of higher metazoan taxa including those in figures 1 and 4.

Taxa	Cell types	Age
Poriferans	Simpson 1984	Placed near origin of Metazoa because of sister-group relation with rest of Metazoa (Sogin in press).
Cnidaria	Chapman 1974	Inferred from ages in Conway Morris 1989.
Hemocoelic bilaterans	Valentine 1993	Inferred from traces (see Crimes 1992) and Conway Morris 1989.
Echinodermata	Sneath 1964*	Sepkoski 1992
Annelida	Westheide & Hermans 1988	Inferred from size of vertical traces (see Droser 1991).
Arthropoda	Valentine ms.*	Inferred from traces (see Crimes 1992).
Agnatha	Hardisty & Potter 1971–1982	Carroll 1988
Cephalopoda	Sneath 1964*	Sepkoski 1992
Actinopterygii	Sneath 1964*	Carroll 1988
Amphibia	Sneath 1964*	Carroll 1988
Diapsida	Sneath 1964*	Carroll 1988
Aves	Sneath 1964*	Carroll 1988
Hominidae	Alberts et al. 1989	Carroll 1988

* Estimates not documented by lists of cell types or by references to published histological descriptions.