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A metric for the study of evolutionary trends in the complexity of serial structures

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Little empirical work has been done to see what sort of patterns of change in morphological complexity occur in evolution, mainly because the complexity of whole organisms has been so hard to define and to measure. For serial structures within organisms, there are fewer difficulties; this paper introduces a set of complexity metrics that are designed especially for serial structures, and then explores some of the properties of the new metrics. Also, a principle proposed in the last century by Herbert Spencer, and offered recently in a new form by the thermodynamic school of evolutionary thought, predicts that complexity should increase in evolution as a consequence of the accumulation of perturbations. Here, simulations in which perturbations are introduced to ideal and real series of vertebral measurements show how the complexity increase predicted by Spencer's principle would be captured by the new metrics.

KEY WORDS: Complexity – serial homology – Herbert Spencer – evolutionary trends.

CONTENTS

INTRODUCTION

The notion that organismal complexity increases in evolution seems to be part of the conventional wisdom in evolutionary biology. The existence of a trend has been noted explicitly by many including Lamarck (1809 [1984]), Spencer (1860 [1890]), Rensch (1960), Stebbins (1969), Saunders & Ho (1976, 1981),

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Wicken (1979, 1987), Bonner (1988) and Brooks & Wiley (1988). Nevertheless, many have expressed scepticism. Williams (1966) and Hinegardner & Engelberg (1983) have questioned whether complexity has increased significantly since the Cambrian. McCoy (1977), Wake *et al.* (1986), Maynard Smith (1970), Simpson (1949) and others acknowledge that increases have occurred but emphasize that they have been restricted to certain groups and certain times and that decreases are common as well.

Little empirical work that might help settle this controversy has been done (although see studies by Cisne, 1974 and Bonner, 1988). This is partly because a trend has seemed so obvious to many of its proponents as to not require demonstration. A more important reason is that complexity has been hard to define and to measure.

Useful tools or metrics for measuring biological complexity have been devised for whole organisms by Schopf *et al.* (1975) and Bonner (1988), for complex organic molecules by Papentin (1982) and Yagil (1985), for behaviour by Cole (1985) and for nucleotide sequences by Gatlin (1972). Here I add another complexity metric (more precisely, a set of metrics) to the tool chest, one designed especially for serially homologous structures.

In this paper I first explain the metrics and explore some of their properties. Then, in a series of simulations, I show how the metrics would capture the increase in complexity in evolution that has been predicted on theoretical grounds.

COMPLEXITY METRICS FOR SERIAL STRUCTURES

The standard approach to measuring complexity

Complexity is usually understood to mean something like heterogeneity or degree of differentiation. And among modern students of complexity, there is some consensus that the differentiation of a system is measurable as some function of the number of different parts it has and the irregularity of their arrangement. A complex system is therefore one with many different parts arranged in a patternless configuration (Wicken, 1979, 1987; Saunders & Ho, 1976; Papentin, 1980; Hinegardner & Engelberg, 1983; Kampis & Csányi, 1987). Junk heaps, glacial till, and jigsaw puzzles are complex. (For discussion of 'order' and 'organization' and their relation to complexity, see Wicken, 1979, 1987 and McShea, 1991.)

Note that the concern here is only with morphological complexity, by which I mean the degree of physical differentiation in a system of comparable parts of about the same size. Excluded are the many other kinds of complexity, such as hierarchical (the degree to which a system is composed of nested subsystems), functional (the number of different tasks a system can perform), generative (the number of steps or instructions required to generate a system), and so on.

Serial structures

Measuring the complexity of whole organisms is difficult using the standard approach, because it is hard to know what to call a part and hard to find a simple way to characterize three-dimensional arrangements. Bonner (1988) has

made some headway using cells as parts and measuring complexity as number of different cell types. The approach is apt, but only rough estimates of numbers of cell types are possible for organisms with more than about ten. Also, the problem of characterizing pattern remains.

For serially homologous structures, such as body segments in annelids, vertebrae in vertebrates and limb segments in arthropods, the difficulties are fewer. Each element in the series is a part, and in arthropods, for example, the types of limb segments are different enough that they can be readily distinguished and the number of each type can be counted. Further, the arrangement of structures is linear, so the complexity of their arrangement is easy to measure using the Boltzmann/Shannon equation. Cisne (1974) took advantage of both of these features in his pioneering study of complexity changes in aquatic, free-living arthropods.

This method works for some serial structures, but not for others. In the vertebral column, for example, change along the sequence is continuous and the elemental types intergrade, so types cannot be reliably distinguished and counted. (It is possible to characterize vertebral types loosely—mammals have cervical, thoracic, lumbar, sacral and caudal types—but for within-class comparisons greater resolution is needed.) The solution is to give up trying to count parts and instead to measure differentiation in some other way, such as the following.

The metrics

The metrics use as raw data a sequence of measurements of a single dimension of each element along a homologous series (for example, the sequence of vertebral centrum lengths in a squirrel specimen shown in Fig. 1A). They are thus univariate in the present discussion, although generalization to the multivariate case would be straightforward.

The metrics consist of three measures of complexity and two measures of its opposite, order or constraint:

$$R = \log\,(X_{\max} - X_{\min}) \qquad \text{(range)}$$

$$C = \log\left(2\sum_{i=1}^{N} |X_i - \bar{X}|/N\right) \qquad \text{(polarization)}$$

$$Cm = \log\left(\left(\sum_{i=1}^{N-1} |X_{i+1} - X_i|\right)\Big/(N-1)\right) \qquad \text{(irregularity)}$$

$$E1 = R - C \qquad \text{(concentration)}$$

$$E2 = C - Cm \qquad \text{(smoothness)}$$

where X_i is the measurement taken from the ith element and N is the number of elements.

R is the range of variation along the series. For centrum length this is just the log of the difference between the longest centrum and the shortest.

C is the polarization of the column. For centrum length, this is the log of twice the average absolute difference between each centrum length and the mean for

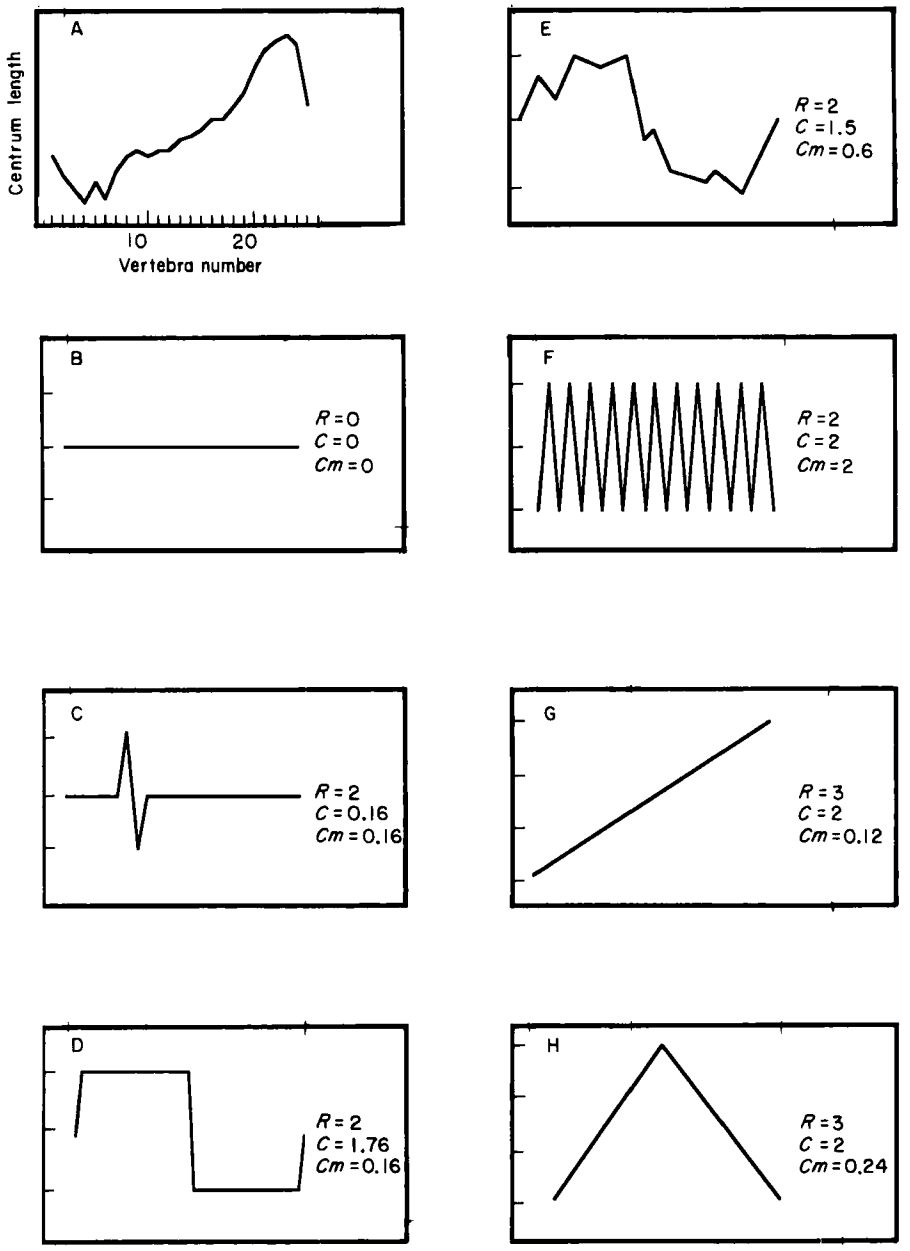

Figure 1. A, Centrum length in a squirrel specimen from axis to last presacral vertebra. B–H, Plots of centrum length (in arbitrary units) using artificial data to show how the metric works. Anti-logs of complexity values are shown adjacent to each plot; anti-logs are used in order to make the connection between the shape of the data curves and their complexity values easier to see. B, The extreme case in which all elements are identical and all complexity measures are equal to zero. C, Two elements in the ideal series in Fig. 1B become differentiated, causing a substantial increase in *R*, but only small increases in *C* and *Cm*. D, Polarization of most of the elements in Fig. 1C to the extremes of the range increases *C* to the point where it approaches *R*, but leaves *Cm* virtually unchanged. E, The addition of numerous perturbations along the series in Fig. 1D (with the range held constant) has some effect on *C* and increases *Cm* substantially. F, *Cm* is maximal, and equals *C* and *R* (that is, *E*1 and *E*2 equal zero) in a regular alternating series. G, H, *Cm* is larger when the source of a developmental gradient is located in the middle of a series (H) that when it is at either end (G).

the series. (The average difference is doubled so that when *Cm* is maximal, *R*, *C* and *Cm* are all equal.)

Cm is the irregularity of the column. For centrum length, this is the log of the average difference in length between adjacent vertebrae.

*E*1 measures the degree to which elements are concentrated at the mean, as opposed to the extremes, and *E*2 measures smoothness or the degree to which each vertebra is constrained to be like the one before it. The properties of the metrics in certain ideal cases are discussed in the caption to Fig. 1A–E.

Rationale

The main justification for the metrics is that they are simple and give results that agree reasonably well with our intuition (although some exceptions are discussed below). As in the standard approach, complexity is understood as a measure of differentiation, but rather than using counts of parts, these metrics use differences among parts (or elements, in the case of serial structures). The thinking is that the more different the elements get from each other, the more complex the series.

Importantly, no claim about the genetic basis of differentiation in serial structures is implicit in the metrics. They are simply devices for expressing quantitatively the morphological differentiation that we observe, just as a ruler expresses length quantitatively. Of course, if empirical studies do reveal the actual genetic mechanisms behind serial differentiation, quantitative metrics such as these will make it easy to discover what relation such mechanisms have (if any) to observed complexity.

Having made this disclaimer, I would like to speculate. We seem to have two disparate kinds of complexity, one based on counting parts and the other on measured differences, but it is possible that they are really the same. To see how this might be so, consider a view of morphological differences as constructed or built up from small, standard-sized difference increments, with each increment understood as a different part. Thus, a large difference would be built up from proportionally more standard increments than a small difference; it would have proportionally more parts and thus proportionally greater complexity.

Further, the standard increment might have a physical basis. Quantitative genetic theory for continuous or quantitative characters assumes that such characters are controlled by a large number of genes, that the morphological effect of each gene is small and of about the same magnitude as all others, and that the effects of the genes are additive (Mather, 1979; Lande, 1981; Mather & Jinks, 1982). In the scheme above, one gene might specify one difference increment. Accordingly, a large morphological difference within a series might require proportionally more genes to specify than a small one and thus might correspond to proportionally greater genetic complexity.

I must emphasize that the metrics do not depend on these speculations for their justification. There is no reason why we should not have two kinds of complexity, one for discrete and one for continuous variation. Further, no correlation between genetic and morphological complexity is required. The metrics are tools for investigating patterns of *morphological* complexity change in evolution. If we discover that genetic complexity is independent, that will be

very interesting but will not undermine any evolutionary patterns that emerge from purely morphological studies.

PROPERTIES OF THE METRICS

Gradients

The graded changes observed in serial structures has led to the suggestion that differentiation is controlled by developmental gradients (Danforth, 1930; Huxley, 1932; Butler, 1939; Van Valen, 1970; Sawin & Hamlet, 1972 and earlier papers in the series). The idea behind most gradient theory is that a source of a diffusible substance, a morphogen, occurs at some point along a developing series, and at that point concentration of the substance is highest and its morphological effect (whether to increase or decrease some feature) is greatest. On either side of the source, morphogen concentration and the effect on morphology falls off monotonically. In a series (such as that in Fig. 1A), each local maximum and/or minimum might be interpreted as the product of a single source and gradient (Sawin & Crary, 1964; Van Valen, 1970).

The metrics capture the effects of gradients as constraints on *Cm*, measured by *E*2. To the extent that variation follows a gradient, each element is constrained to be similar to its neighbour, and *Cm* will be low relative to *C* (high *E*2). Conversely, *Cm* is most sensitive to reversals of the direction of change, to numbers of peaks and troughs, and thus a large number of independent gradients producing many reversals will result in a high *Cm* value relative to *C* (low *E*2).

Cm can be fooled, so to speak, in one way. A regular, alternating series (Fig. 1F) will have a very high *Cm* value, as indeed it should if each peak and trough represents an independent gradient. Such alternating series occur, for example, in the heights of neural processes of fossil captorhinomorph reptiles (Sumida, 1987). Such series do not seem very complex, however, and it is not hard to imagine how such a pattern might be produced quite simply, if pairs of elements are developmentally linked in some fashion. To accommodate series that alternate or have even longer repeating patterns, additional metrics—variations on *Cm*—could be easily developed.

Fig. 1G, H shows a case in which *Cm* appears to be fooled but may not be. The series in Fig. 1H has the higher *Cm* value, because *Cm* weighs, so to speak, the effect of both peaks and troughs: both have one peak, but Fig. 1H has two troughs (local minima) while Fig. 1G has one. Clearly, however, both patterns could be produced by a single source lying at the centre in Fig. 1H and at one end or the other in Fig. 1G. The problem could be avoided by artificially linking the two ends of each series and computing the *Cm* of each as if it were circular, in which case the two would have the same *Cm* values. On the other hand, Fig. 1H may actually be the result of two gradients, one at each end, in which case the difference in *Cm* values is appropriate. In the absence of knowledge of source placement, it is impossible to know what to do in concrete cases, and the choice of *Cm* protocols (the non-circular or the circular version) is difficult. The non-circular protocol seems preferable here, because although it is more likely to exaggerate complexity differences, it is also less likely to overlook them.

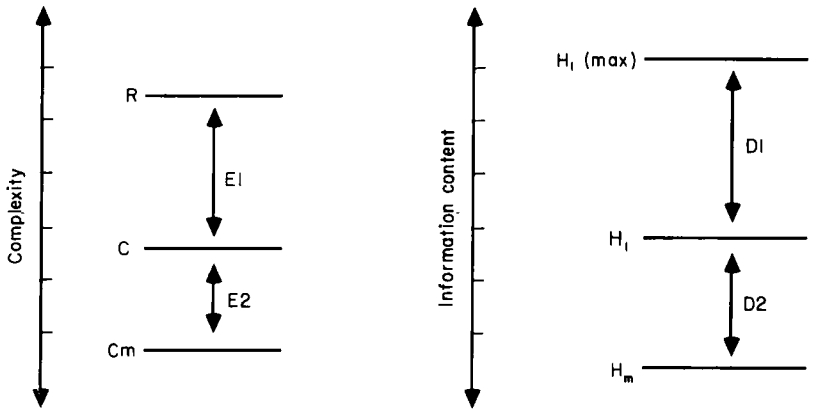

Figure 2. Relations among the complexity measures proposed here (left) and analogous measures of information content from communications theory (right). *R* is always greater than *C*, and *E*1 measures the difference between them; similarly, H_1 (max) is always greater than H_1, and D_1 is their difference. *C* is always greater than *Cm*, and *E*2 measures their difference; likewise, H_1 is always greater than *Hm*, and *D*2 is their difference.

Differences in size may also confound the metrics in that larger organisms will tend to have larger values even when their homologous series are not more complex. Size differences do not affect the conclusions from the simulations below, but for comparisons among real taxa the problem is significant and will be addressed in a subsequent paper.

Similarities with communications-theory metrics

R, *C*, *Cm*, *E*1 and *E*2 share some properties with the standard information-content metrics from communications theory: H_1 (max), H_1, *Hm*, *D*1 and *D*2 (from Gatlin, 1972). H_1 (max), like *R*, measures the maximum information possible per symbol (per element, for *R*). H_1, like *C*, measures the average information actually represented by each symbol (element). And *Hm*, like *Cm*, measures the information represented by each symbol (element), taking into account the constraint introduced by the preceding element or symbol.

There are also significant differences. H_1 is maximal and equals H_1 (max) when all symbols are equally probable; *C* is maximal and equals *R*, however, when all elements lie at the extremes of the range (half at each extreme). *Hm* is maximal and equals H_1 when the probability of each symbol is independent of the one before it; *Cm* is maximal and equals *C* when each element is as different as possible from the one before it (Fig. 1F). Figure 2 shows the relationships among the variables in each set of metrics.

SPENCER'S PRINCIPLE

There are few data on complexity change in evolution but there is no shortage of theoretical work. A great variety of rationales have been devised to explain why complexity ought to increase over time (McShea, 1991). For example, complexity increase has been said to be driven by invisible fluids (Lamarck, 1809 [1984]), natural selection (Rensch, 1960; Maynard Smith, 1970; Bonner, 1988),

size increases (Cole, 1985; Bonner, 1988), increasing heterogeneity of the biosphere (Waddington, 1969) and 'diffusion' in a morphospace with a fixed lower boundary (Fisher, 1986).

One argument that has drawn much attention recently comes from what might be called the internalist school. Today's internalists argue that complexity increase is driven not by external forces such as selection, but by an internal randomizing tendency, specifically entropy or the Second Law of Thermodynamics (see especially Maze & Scagel, 1983, Wicken, 1987, and Brooks & Wiley, 1988). The internalists have glimpsed the mechanism behind what could, in principle, be a powerful force driving complexity increase, but the language of thermodynamics is awkward and unfamiliar in biology. It may also be unnecessary: what appears to be essentially the same mechanism was described in more general terms in the nineteenth century by Herbert Spencer. I adopt Spencer's version here.

Spencer (1860 [1890]) argued that given a diffuse collection of particles, all more or less identical, the collection will begin to condense, perhaps from gravity if the system is a physical one. As it condenses, the particles become distinguishable from each other by virtue of their differing positions within the whole, those outside from those inside, for example. Particles in different positions occupy distinct environments and thus are acted upon by different forces (or are acted upon differently by the same forces), causing them to differentiate into distinct particle types. The whole, formerly homogeneous, becomes heterogeneous.

Biological systems do not seem to 'condense' in Spencer's sense (although Denbigh, 1975 and Wicken, 1987 suggest that aggregative forces dominate in biological systems), but aspects of his logic are familiar in biology. In combination with natural selection, we use it to account for the breaking of primitive symmetries, for the evolution of features defining front and back in organisms that move, or inside and outside in those too large to depend on diffusion. The mechanism is general, Spencer argues, and he invokes it to explain the differentiation of the planets in the Solar System from a homogeneous nebula, of social roles in advanced societies from their undifferentiated condition in primitive ones, of words in modern languages, of the organs in complex organisms and so on.

The argument has a corollary, which Spencer calls "the principle of the instability of the homogeneous" and which I will call Spencer's principle. Homogeneous systems are unstable, he argues, and they tend to differentiate by the passive accumulation of perturbations. The corollary does not mean that the homogeneous system is weaker, more prone to destruction, or selectively disadvantaged. Rather, it is unable to maintain the identity and internal relations of its parts. The instability, Spencer writes, is not that of a stick balanced on its end, but that of balanced scales becoming unbalanced due to rust, erosion, wind and so on.

In evolution, the passive accumulation of perturbations can be understood as a randomizing vector, a force tending to increase the complexity of organisms in a lineage. More specifically, the implication for serial structures is that variations or perturbations occurring along an initially uniform series should result in differentiation, and this differentiation should increase as perturbations accumulate. Darwin (1859 [1964]), Cope (1871), Williston (1914) and Gregory

(1935a, b) seem to have had something like this principle in mind for explaining the differentiation of serial structures, as did Ohno (1970) for gene differentiation in multigene families, and Pringle (1951) for increases in behavioural repertoires.

EFFECTS OF PERTURBATIONS

Some simple simulations suggest that, in agreement with Spencer's principle, increasing complexity (*R*, *C* and *Cm*) is the most probable result of the addition of perturbations. This is trivially true for an ideal, perfectly ordered series; complexity in such a series (for example, Fig. 1B) can only increase. More interestingly, it is also true for various real series which are already complex in some respects: the effect of perturbations on the complexity and constraint measures is demonstrated here using a series of homologous measurements along the vertebral columns of three very different mammals.

The first set of simulations models the effect of randomly perturbing elements individually in a perfectly ordered series; the second set does the same with three real series; and the third shows the effects of adding a more structured perturbation, again to three real series.

Random perturbations of an ordered series

Consider an ideal series of 20 elements that are initially the same in some dimension, say length, at time zero (Fig. 1B). In each time unit or step, 20 coins are flipped, one for each element, and the length of the corresponding element is increased by one small increment for a head, decreased by one increment for a tail. Thus the lengths of the elements follow independent random walks through time. (Random walks are prevented from crossing the zero-length line by setting the starting length at some very large value; this could also have been done by making the step sizes very small.)

Figure 3 shows the average trajectory of *R*, *C* and *Cm* for the entire series over 20 time intervals in 500 simulations. All three measures increase as the 20 independent random walks diverge from each other. So long as upper and lower boundaries on the length of elements are not reached, the measures increase without limit. Note first that the increase is not an effect of what might be called size, because the length of an element decreases as often as one increases, and the average length (a reasonable estimate of size) remains about the same.

Second, in real series, natural selection presumably places upper and lower limits on the dimensions of the elements, and therefore complexity should stop increasing when elements reach some high level of dispersion between the limits. Vertebral series, at least, are highly ordered and lie far from this high level of dispersion, however. Therefore, increases in complexity are always the expectation, as shown in the next set of simulations. Interestingly, the constraint measures (*E*1 and *E*2) quickly stabilize, raising the question of whether or not they do so in real evolving systems as well.

Random perturbations of real series

Here, the effect of adding random noise to measurement series from real vertebral columns is illustrated. Figure 4A shows measurements taken from the

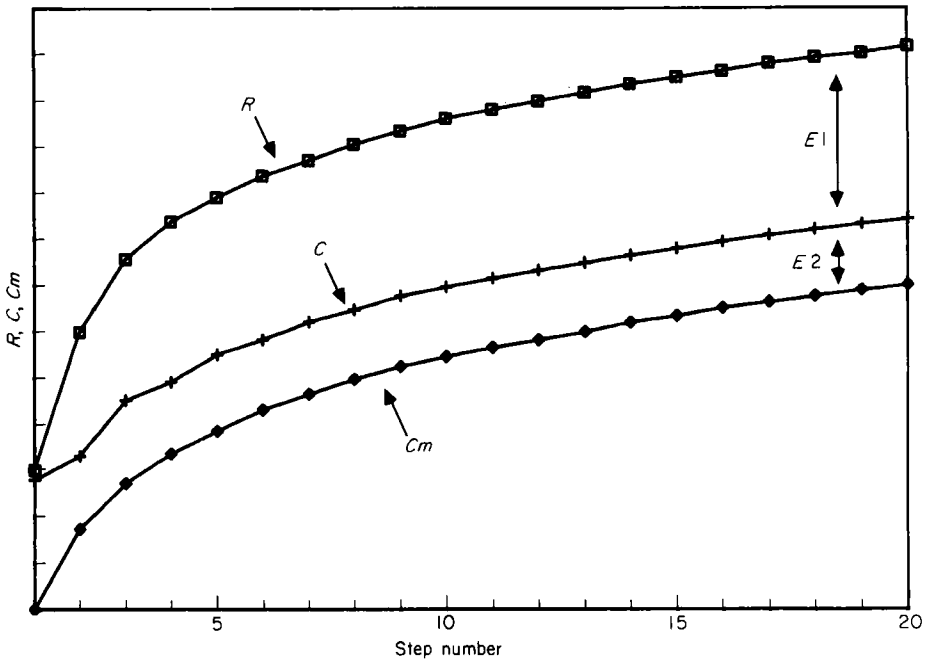

Figure 3. Random perturbations of a perfectly ordered series. Twenty originally identical elements underwent simultaneous, independent, one-dimensional random walks. Each step in the walks added one increment, with probability one half, or subtracted one increment, with probability one half. Complexity and constraint values were computed at each step, and the walks continued for 20 steps. The procedure was repeated 500 times and average values at each step computed. *R*, *C* and *Cm* all show a pattern of monotonic increase, as the elements dispersed from the mean, while *E*1 and *E*2 stabilized at constant values.

vertebral columns of three mammals, a squirrel, a seal and a chevrotain. The measurements are the altitudes of the neural processes (defined in Fig. 4 caption) from the third cervical vertebra (just behind the axis) to the last presacral vertebra. In the simulations, a number was selected at random for each vertebra from a normal distribution with a mean of zero and added to the neural process measurement for that vertebra (for example, Fig. 4B). *R*, *C*, *Cm*, *E*1 and *E*2 values for the entire column were recomputed, the procedure was repeated (always starting with the original data) 100 times, and the results averaged. The model was run with various standard deviations. Results appear on the left in Table 1.

The effect of adding random noise was almost always to increase *R*, *C* and *Cm*. *E*1 increased also, because noise tended to extend the range (*R*) more than it increased the average dispersion of the vertebrae (*C*). The original vertebral series were highly constrained, and the constraint was disrupted by the addition of high-frequency noise, causing *E*2 to decrease. That is, *C* increased but *Cm* increased even more. A slightly different experiment, in which the original data were *multiplied* by numbers drawn from a log-normal distribution, gave concordant results.

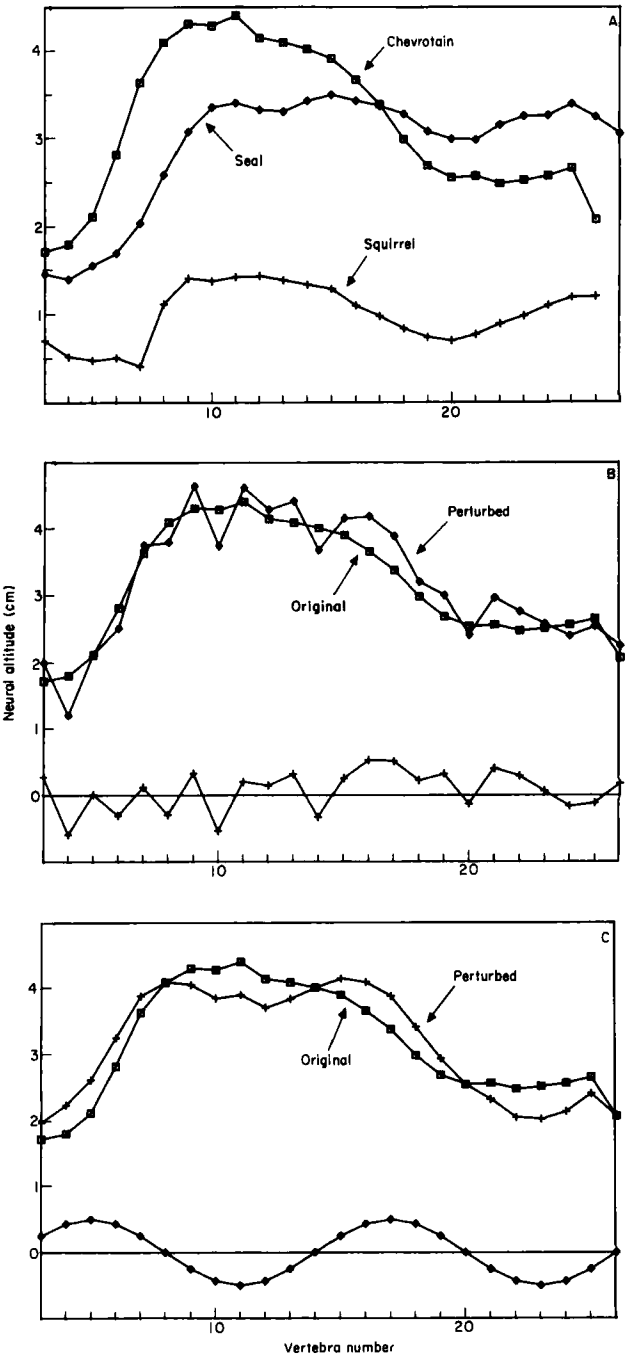

Figure 4. A, Measurements of neural altitude from the third cervical vertebra to the last presacral in three specimens: a squirrel, a seal and a chevrotain. Neural altitude is defined as the distance from the most anterior point on the dorsal median centrum (not including the epiphysis) to the anterior tip of the neural process. B, An example of the effect of the addition of random perturbations; here, the perturbations (lower trace) are added to the original chevrotain data to produce the modified or perturbed series. The perturbations are selected from a normal distribution with mean zero and standard deviation 0.5 cm. C, An example of the addition of a sine-wave perturbation; here, a sine wave (lower trace) with amplitude 0.5 cm and frequency 2 cycles (over the length of the vertebral column) is added to the original chevrotain data to produce the perturbed series.

TABLE 1. Results of adding random and structured perturbations to vertebral data (neural process altitude) from a squirrel, a seal and a chevrotain. Entries in the table indicate whether *R*, *C*, *Cm*, *E*1 or *E*2 increased (+) or decreased (−) for a particular combination of parameters. The single column on the left shows the consequences of introducing random perturbations drawn independently for each element from normal distributions with six different standard deviations (in cm). The remaining five columns show the results for sine-wave perturbations with five different frequencies (in number of cycles over the length of the column) and six different amplitudes (in cm). Complexity and constraint values are computed as averages of values from eight equal phase-angle shifts spanning one wavelength. Overall, *R*, *C*, *Cm* and *E*1 tended to increase while, at least for high frequency perturbations, *E*2 tended to decrease

Random perturbations			Sine-wave perturbations					
	Standard deviation		Amplitude	Frequency 0.25	0.5	1	2	4
Chevrotain								
R	0.01	+	0.01	+	+	−	+	+
	0.05	+	0.05	+	+	−	+	+
	0.1	+	0.1	+	+	−	+	+
	0.2	+	0.2	+	+	−	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
C								
	0.01	−	0.01	+	+	−	+	+
	0.05	−	0.05	+	+	−	+	+
	0.1	+	0.1	+	+	−	+	+
	0.2	+	0.2	+	+	−	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
Cm								
	0.01	+	0.01	+	+	−	+	+
	0.05	+	0.05	−	+	−	+	+
	0.1	+	0.1	+	+	−	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
*E*1								
	0.01	+	0.01	+	+	+	+	+
	0.05	+	0.05	+	−	+	+	+
	0.1	+	0.1	+	−	+	−	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
*E*2								
	0.01	−	0.01	+	+	−	+	+
	0.05	−	0.05	+	+	−	−	−
	0.1	−	0.1	+	+	−	−	−
	0.2	−	0.2	+	−	−	−	−
	0.5	−	0.5	−	−	−	−	−
	1	−	1	−	+	−	−	−

TABLE 1. (*continued*)

Random perturbations		Sine-wave perturbations					
	Standard deviation		Amplitude	Frequency 0.25	0.5	1	2	4
Seal R								
	0.01	−	0.01	+	−	+	−	−
	0.05	+	0.05	+	+	+	−	−
	0.1	+	0.1	+	+	+	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
C								
	0.01	+	0.01	+	−	+	−	+
	0.05	−	0.05	+	+	−	−	+
	0.1	+	0.1	+	+	+	−	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
Cm								
	0.01	+	0.01	+	+	+	−	+
	0.05	+	0.05	+	+	+	+	+
	0.1	+	0.1	+	+	+	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
*E*1								
	0.01	−	0.01	+	+	+	+	−
	0.05	+	0.05	+	+	+	+	−
	0.1	+	0.1	+	+	+	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	−	−	+	+
*E*2								
	0.01	−	0.01	+	+	+	−	+
	0.05	−	0.05	+	+	−	−	−
	0.1	−	0.1	+	−	−	−	−
	0.2	−	0.2	−	+	−	−	−
	0.5	−	0.5	+	+	+	−	−
	1	−	1	+	+	+	−	−
Squirrel R								
	0.01	+	0.01	+	+	−	−	+
	0.05	+	0.05	−	−	+	+	+
	0.1	+	0.1	+	+	+	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+

TABLE 1. (*continued*)

Random perturbations			Sine-wave perturbations					
	Standard deviation		Amplitude	Frequency 0.25	0.5	1	2	4
C								
	0.01	+	0.01	+	+	−	+	+
	0.05	+	0.05	+	+	+	+	+
	0.1	+	0.1	+	+	+	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
Cm								
	0.01	+	0.01	+	+	−	+	+
	0.05	+	0.05	+	+	−	+	+
	0.1	+	0.1	−	+	+	+	+
	0.2	+	0.2	+	+	+	+	+
	0.5	+	0.5	+	+	+	+	+
	1	+	1	+	+	+	+	+
*E*1								
	0.01	+	0.01	+	+	+	+	+
	0.05	+	0.05	−	−	−	+	+
	0.1	+	0.1	−	−	−	+	+
	0.2	+	0.2	−	−	+	+	+
	0.5	+	0.5	−	+	+	+	+
	1	+	1	+	+	−	+	+
*E*2								
	0.01	−	0.01	+	+	−	+	+
	0.05	−	0.05	+	+	+	−	−
	0.1	−	0.1	+	+	+	−	−
	0.2	−	0.2	+	+	+	−	−
	0.5	−	0.5	+	+	+	−	−
	1	−	1	+	+	+	−	−

Structured perturbations of real series

In these simulations, sine-wave perturbations were added; the waves are highly structured perturbations such as might be introduced in real vertebral columns by the addition of one or more morphogenetic gradients. Figure 4C shows the changes in neural altitude effected by adding a sine wave to the chevrotain data. For a sine wave, three parameters can vary: amplitude, frequency and phase angle. Table 1 shows the effect on *R*, *C*, *Cm*, *E*1 and *E*2 of adding waves of five different amplitudes and five different frequencies. Values are computed as averages from eight equal phase-angle shifts spanning one wavelength.

R, *C* and *Cm* increase for most amplitudes and frequencies, although decreases do occur for some combinations. *E*1 increases and, at least for higher-frequency waves, *E*2 decreases, as in the random-perturbation model. Note that for low frequencies, especially those less than one cycle per column length, *E*2 tends to increase. This is not an anomalous result, because a low-frequency wave will

tend to disperse vertebrae from the mean (increasing *C*) without adding a great deal of vertebra-to-vertebra irregularity (a lesser increase in *Cm*). A multiplicative model again gave similar results. Other mammalian taxa and other vertebral dimensions, such as centrum height, width and length, show about the same degree of structuring and can be expected to yield the same pattern of results.

If the perturbations considered here were at all realistic, and if no other forces were at work, then Spencer's principle would make two predictions for real evolutionary lineages: (1) *R*, *C* and *Cm* should all increase; and (2) real columns are relatively unconcentrated (low *E*1) and consequently are likely to extend their ranges, to produce one or a few highly differentiated vertebrae, as a result of perturbations. Also, real columns are quite smooth (high *E*2), having low *Cm* relative to *C*, and consequently are likely to become relatively more irregular (lower *E*2) as perturbations accumulate.

EVOLUTIONARY EXPECTATIONS

In addition to Spencer's principle, natural selection must also affect complexity. Unfortunately, we do not know how strongly (relative to Spencer's principle), how frequently, or even in what direction it is likely to act. Regarding direction, some organismal functions must require simpler designs, and selection for these functions ought to reduce complexity. On the other hand, complex designs must also be favoured sometimes. Saunders & Ho (1976) and Stebbins (1969), among others, argue that complexity increases are more likely to be advantageous than decreases, while Castrodeza (1978) and McCoy (1977) doubt whether such a bias exists. The theoretical issue is at present unresolved, and therefore it is difficult to know whether to expect Spencer's principle and natural selection typically to act in the same direction or to oppose each other.

Still, the action of selection may be demonstrable in some cases. Most complexity decreases in single lineages, for example, are probably attributable to selection. (Increases, of course, may be the result of either Spencer's principle, selection or both.) Among many lineages, decreases will probably not predominate (the conventional wisdom is probably not completely wrong), but if they do, this will weigh in favour of the overall efficacy of selection. Finally, if increases predominate, or if neither increases nor decreases predominate, it will be difficult to assign causes to the pattern.

In any case, the discussion of causes may be premature. What we need is more data on complexity change in evolutionary lineages so that we will have some sense of what patterns actually occur that require explanation. The metrics developed here, along with the others listed earlier, provide means to that end.

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