ral History, vol. 29, no. 4, and vol. 30, no. 1 [1978]), which, though its estimates are based on the more complete specimens rather than on entire assemblages, gives MNI estimates for Olduvai bovids. Nor apparently did he consult a thorough review by Isaac and Crader (written in 1977 and widely circulated; published in 1981 in *Omnivorous Primates*, R. Harding and G. Teleki, Eds., Columbia University Press) of published reports on Early Pleistocene African bone assemblages and their implications for hominid carnivory. Isaac and Crader reach many comparable conclusions (for example, they reject vertically diffuse accumulations as representing living floors) without MNI estimates or the numerical acrobatics conducted by Binford.

Many of the methods by which Binford converted Mary Leakey's data for analysis involve highly risky assumptions, and errors of great magnitude are consequently inherent in his results. For example, for the famous FLK *Zinj* site Binford calculates an MNI estimate of 13.07, based on teeth. He then calculates MNI estimates for other skeletal parts at FLK *Zinj* and other Olduvai sites and uses these as fact. The dangers of error in such a superstructure will be well understood from the fact that my own recently completed archeological analysis of the bones making up the FLK *Zinj* assemblage yields an MNI estimate, based on teeth, of 40 individual animals identifiable as bovid, suid, equid, and giraffid. Many more identifiable bone specimens are present than Binford's numbers indicate. Moreover, the presence in the FLK *Zinj* assemblage of numerous cut-marked bones that are identifiable to skeletal part (H. T. Bunn, *Nature (London)* **291**, 574 [1981]; R. Potts and P. Shipman, *ibid.*, p. 577) indicates, on Binford's criteria, that skinning, dismemberment, and meat removal by hominids occurred. These facts strongly suggest bone accumulation principally by hominids, followed by carnivore scavenging, as the simplest interpretation of that site.

Binford raises the important question of natural background bone density resulting from normal animal mortality and other nonhominid factors, but he does not develop this line of reasoning adequately, even though it is critically important to interpretations of bone and artifact accumulations as indicating living floors and home bases. He makes some astute comments on the alleged hippo and elephant butchery sites at Koobi Fora and Olduvai. With justification he challenges the interpretation of these sites as representing butchery of large mammals by early hominids, arguing that some or all of the bones present may be part of a natural scatter. At many of the Olduvai and Koobi Fora sites the bone densities that Binford chooses to regard as background density do, however, stand out as strong high-density anomalies relative to the modern East African background bone-density figures of Hill and Behrensmeyer (*Fossils in the Making*, University of Chicago Press, 1980). That contrast does not support Binford's position, unless it can be assumed that the alleged background accumulations took place over a long period of time (or at a faster rate than is documented in modern analogue environments). In view of Mary Leakey's reports of fresh, unweathered, unabraded bones and artifacts and Hay's statements concerning depositional conditions (*Geology of the Olduvai Gorge*, University of California Press, 1976), such assumptions seem improbable to say the least. Though it is true that anomalously high bone densities either with or without stone artifacts in physical association do not in themselves necessarily indicate hominid involvement, recognizing that the sites are high-density anomalies does permit a fairer understanding of Mary Leakey's reasons for viewing the bones as hominid food debris.

There are also problems with Binford's use of data derived from modern carnivores. One of these has to do with the transferring of diagnostic criteria on bone fragmentation from modern human and carnivore assemblages to the Early Pleistocene. The problem is not that uniformitarian assumptions about these properties are unwarranted; rather, premature application overlooks other potential agents of bone fragmentation, including fragmentation resulting from trampling of exposed bones by large mammals. This process may operate in a manner that is analogous to attrition from animal gnawing, which Binford emphasizes may cause initially different bone assemblages to appear more similar. Moreover, for his generalizations Binford, though he uses some African data, relies principally on his own relatively small set of data on Alaskan wolves. In so doing, he implicitly equates wolves with hyenas and other large African predator-scavengers. Yet, as Binford documents, wolves are at the top of the carnivore hierarchy and can afford to lounge around and sleep beside partially eaten carcasses. African hyenas often do not share that luxury, and faced with more competition hyenas exhibit considerable behavioral variability both within and between species. Ongoing research has shown that hyenas are capable of a broad spectrum of bone transport and modification. In mechanical terms, spotted hyenas are probably stronger than wolves as bone breakers and thus are probably capable of considerably more bone destruction.

Still another weakness of Binford's analysis is that in viewing sequentially modified assemblages first in terms of canid predator-scavenger data and attributing only residual variations to other factors without considering alternative lines of analysis and explanation he is guilty of the same type of single-mindedness for which he relentlessly criticizes others. Why should the opposite sequence—hominids as principal agents of bone transport and modification, followed by attrition due to scavenging carnivores—escape Binford's serious consideration?

Binford's presentation is marred by many unnecessary errors, including mislabeled or incompletely labeled graphs and tables, erroneous text references to his own and other researcher's tables, and misquotation of other researchers' published and even unpublished writing. Patient detective work can resolve most of these discrepancies, but that is asking a lot of the reader.

Binford's book will have a major impact on future archeological research by stimulating additional middle-range research. Despite what I consider to be serious shortcomings, the book is essential reading for archeologists and others with an interest in past human subsistence activities, but it should be read with the full understanding that some of the modern myths are being generated by Binford.

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Endosymbiosis vs. Autogeny

Origins and Evolution of Eukaryotic Intracellular Organelles. Papers from a conference, Jan. 1980. JEROME F. FREDERICK, Ed. New York Academy of Sciences, New York, 1981. x, 512 pp., illus. Cloth or paper, $99. Annals of the New York Academy of Sciences, vol. 361.

The further back in time we attempt to trace phylogenies the less reliable is the evidence and the more conjectural are the conclusions. Facts become especially hard to find, and hypotheses are correspondingly easy to make, when we ask about the origin of eukaryotes (animals, plants, fungi, protists) and their distinc-

tive cellular organelles. The fossil record is sufficient only to suggest that they arose somewhere in the vicinity of one to two billion years ago. Their ancestors were presumably simpler bacteria-like cells without nuclei, mitochondria, or chloroplasts and had been around for one or a few billion years previously. Those ancestors also gave rise to the modern prokaryotes: bacteria and the recently "discovered" archaebacteria. The differences between the eukaryotic and prokaryotic lines of descent are striking and important: eukaryotes have elaborate internal membranes that function in transport of materials as well as compartmentalize the cell into nuclei, mitochondria, chloroplasts, endoplasmic reticulum, and so on; a cytoskeleton involved in cell division, motility, shape, and transport; and nuclear genomes that are many times richer in genes for specialized functions.

It is small wonder that the question of the origin of the eukaryotic cell and its organelles has generated rival schools and as much polemic as thoughtful discussion. The endosymbiotic school holds that some of the cytoplasmic organelles, in particular the mitochondria and chloroplasts, were originally separate organisms that took up residence as symbionts inside a host cell whose own native genes ended up in the nucleus. The autogenous school has advanced several different theories in which a single cell with its genes was subdivided by gradual steps into nucleus, mitochondria, and chloroplasts.

Origins and Evolution of Eukaryotic Intracellular Organelles is a collection of papers from a conference. Both the autogenous and endosymbiotic schools are well represented. Their contributions are complemented by well-balanced reviews written by fence-sitters and by interesting papers on contemporary intracellular symbioses. Publication of the book was fast enough that it provides a nearly current view of the subject. The majority of papers are clearly written and a number are exceptionally good. However, the book is probably not a good introduction for the nonspecialist, for these are experts talking to each other. Taxonomic nomenclature is a problem; it is difficult to compare phylogenies and follow arguments unless one is familiar with the confusing variety of synonymous or partly synonymous names attached to various groups of algae and bacteria. Many of the papers are followed by discussion. The discussion produces some new and interesting ideas and helps to give the flavor of the conference and of the field as a whole.

The symbiotic and autogenous schools are both alive and well and doing research, as this book shows. But textbooks of biology most often take the endosymbiont theory as proved, at least for chloroplasts, and this probably reflects the view of the majority of biologists both within and without the field. How did a theory about a historical event for which there is no fossil record become dogma? Both the endosymbiotic and the autogenous theories are as old as this century but were not well developed until the 1960's Interest in the problem was stimulated by the discovery that mitochondria and chloroplasts are genetically autonomous, containing their own small sets of genes and apparatus for protein synthesis and reproducing by growth and division. Their DNA molecules are more like those of bacteria than like true nuclear chromosomes, and their ribosomes resemble bacterial ribosomes in several important respects. Moreover, it is apparent that intracellular symbioses involving prokaryotes (in eukaryotic hosts) are common and easily established. Intermediate stages have been found between obvious symbionts capable of independent existence and true organelles dependent on nuclear genes for many functions; it is not yet clear whether the cyanelles of some algae should be called symbionts or chloroplasts.

But for many biologists the most convincing evidence for the symbiont theory comes from the nucleotide base sequences of ribosomal RNA (rRNA) molecules and amino acid sequences of several proteins, notably cytochrome *c*. In the final analysis evolution consists of the substitution of one base pair for another in all the copies of a gene in a population of organisms. This in turn may lead to an amino acid substitution in the protein coded by that gene. Comparing the base sequences of the same gene in two related organisms tells how many substitutions have occurred since those organisms, and their genes, diverged from a common ancestor. From the available data, nuclear rRNA genes seem to be very different from those in either the mitochondrion or the chloroplast; the 5*S* rRNA shows some similarity to that of certain bacteria. Mitochondrial genes are more like those of a different set of bacteria, probably the purple nonsulfur photosynthetic forms. (Note that the gene for mitochondrial cytochrome *c*, which is most useful in this case, is actually in the nucleus; the symbiont theory holds that it was moved there from the premitochondrial symbiont.) The case is strongest for chloroplasts, whose genes are very different from those of the mitochondria or the nucleus but very similar to those of the cyanobacteria (blue-green algae). These data are clearly compatible with the origin of mitochondrial, chloroplast, and nuclear genes from three divergent lines of prokaryotes as required by the symbiotic theory, but their interpretation is still controversial. In their paper in this volume Uzzell and Spolsky argue that the 5*S* rRNA data are ambiguous and that the cytochrome data are better fitted to a phylogenetic tree involving an autogenous origin of organelles. The phylogenetic tree of cytochromes derived by Schwartz and Dayhoff actually requires two or three independent symbiotic origins each for the mitochondria and the chloroplasts. Schwartz and Dayhoff, who summarize their work here, consider this strong evidence for the symbiotic theory, but it is not clear why they regard the alternative, multiple autogenous origins, as less reasonable.

Suppose that the interpretation of the sequence data were unambiguous; would they then constitute proof of the symbiotic theory? I think not. The sequence data produce trees that show sequence homologies (phenetic relationships) between organisms, and nothing more. To interpret these trees as showing phylogenetic (cladistic) relationships requires several assumptions. Evolution must be divergent, not convergent, and the rate of base-pair substitution must be the same in all branches of the tree. These assumptions are approximately correct for animals, where they have been tested by comparing sequence trees with the fossil record. Even in animals, they may apply primarily to selectively neutral mutations. The rate of base-pair substitution will be constant only if both the mutation rate and the probability of fixation of a new mutation in the population are constant. As is shown by the data Brown reports here, mutation rates can be very different for nuclear and organelle genes. Mutation rates depend upon the efficiency with which cells repair errors in DNA molecules, which may have changed from time to time as primitive organisms experimented with different repair enzyme systems.

In fact, one can incorporate sequence data in a phylogenetic tree that assumes an autogenous origin of organelles, but the tree requires that the rate of base-pair substitution increased greatly in the genes that were packaged in the nucleus. This could be due to a temporary decrease in repair efficiency. More plausibly, it could reflect rapid evolutionary changes in nuclear genes under selective

pressure. Changes would certainly be necessary to adapt those genes to being separated by the nuclear membrane from the site of protein synthesis in the cytoplasm. Selection for the necessary sequence changes would increase the probability of fixation of mutations and thus increase the rate of base-pair substitution. It is also worth remembering that not all parts of a gene are subject to the same degree of selection, and thus to the same rate of substitution. Some regions of the mitochondrial 21*S* rRNA gene are very similar to that of *Escherichia coli* 23*S* rRNA whereas others have diverged widely (Dujon, *Cell* **20**, 185 [1980]); which regions should we use in making phylogenetic trees?

It is disappointing to see so little discussion of these and other problems of data interpretation in this book, or outside of it for that matter. The symbiont school passes over the problems lightly; one would think they would want to consider alternative interpretations of their data more carefully, in self-defense if for no other reason. In their attacks on the symbiont theory, members of the autogenous school mention the difficulties of interpreting sequence data but give surprisingly little consideration to what evolutionary geneticists tell us about the factors that influence rates of evolution. And surely no theory of the evolutionary origin of organelles can be considered complete and correct until it has explained not only the sequence data but also the numerous other aspects of cell and organelle phenotypes discussed in this book. Excellent reviews of genome structure and protein synthesis in chloroplasts (Gillham and Boynton) and mitochondria (Mahler; Locker *et al.*) show that these organelles are highly diverse and exhibit a remarkable mixture of prokaryotic and eukaryotic features. Why do mitochondria alone have a different genetic code? Why have the number and kind of different genes in mitochondria and chloroplasts changed so little in the evolutionary paths from protists and fungi to humans? Indeed, why should there be any genes at all in these organelles? The papers by Whatley and Gibbs show that some algal chloroplasts are surrounded by three or four membranes instead of the usual two. This is interpreted as representing separate symbiotic events, but the interpretations do not seem to fit well with the phylogenies deduced from sequence data. Could they also be explained by an autogenous theory?

One comes away from this book with the impression of having witnessed an argument or debate, but not a real discussion. Members of both schools tend not to think very hard and deeply about how their data would fit alternative hypotheses. The symbiotic school is emperor for the day, and the emperor does have clothes, but they seem rather transparent. The wide acceptance of the theory may owe a great deal to its novelty and psychological appeal, which are expressed clearly and poetically by Lewis Thomas (*The Lives of a Cell*, Viking, 1974). The notion that "we are shared, rented, occupied" by former symbionts is certainly appealing to the emotions, but I prefer to reserve judgment on its scientific validity.

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Epithelial Transport

Epithelial Ion and Water Transport. Papers from a workshop, Dunedin, New Zealand, Apr. 1980. ANTHONY D. C. MACKNIGHT and JOHN P. LEADER, Eds. Raven, New York, 1981. xx, 372 pp., illus. $42.

The study of epithelial transport has progressed considerably in recent years. Biophysical and biochemical techniques newly applied in this field have permitted in-depth examination of the mechanisms of epithelial salt and water transport. Among these techniques, sophisticated intracellular and transepithelial electrophysiological methods, electron microprobe analysis, light microscopy of living tissue, and studies in isolated membrane vesicles are or will eventually become widespread experimental tools in the study of epithelia. This multiplicity of experimental approaches has made it difficult to provide up-to-date overviews of the subject. Books such as this one are therefore useful complements to publications in research journals.

The volume consists of 35 papers presented at a workshop held in honor of James R. Robinson. The papers are organized in a progression from basic methodological aspects to more specific problems. There are sections on optical and biochemical techniques, intracellular microelectrode methods, measurements of intracellular ions, with ion-selective microelectrodes and electron microprobe analysis, leaky epithelia, tight epithelia, models of ion transport, and regulation of cell volume. The result is a well-organized book of overall high quality.

Most of the contributions are well written, concise, and of current interest. As a whole, they provide a good sample of the essential problems that are currently confronted. Reports on current research, very much in the style of regular research papers, are presented next to papers in which specific techniques or problems are reviewed from a broader viewpoint.

I have chosen, rather arbitrarily, to comment on some of the contributions that appear to be the most exciting because they represent important technical accomplishments or because of the significance of the results themselves.

Di Bona and co-workers discuss the use of differential interference contrast and fluorescence optics in the study of epithelial transport. The most impressive results are those obtained in gastric glands, where acridine orange emission at 624 nanometers allowed the investigators to identify the intracellular low *p*H compartment during stimulation of proton secretion. Additional experiments proved that adenosine triphosphate alone restores the cell capacity for proton secretion after adenosine triphosphate depletion.

Frömter *et al.* communicate successful determinations of cell-membrane resistances and capacitances in *Necturus* gallblader, obtained by transepithelial and intracellular impedance measurements. The data can be obtained rapidly with a single intracellular microelectrode, and they compare excellently with those obtained by flat-sheet cable analysis, which is slow and technically more difficult. Further development of this technique holds high promise for studies of leaky epithelia, including renal tubule segments.

Armstrong and Garcia-Diaz provide an interesting discussion of criteria for the use of microelectrodes to measure membrane potentials in epithelial cells. This paper is complemented well by one by Armstrong on ion-selective intracellular microelectrodes. Because both techniques are increasingly used in epithelial physiology, both papers will be useful to newcomers and to established investigators.

Excellent papers by Rick *et al.*, Thurau *et al.*, and Dörge *et al.* cover the use of electron microprobe analysis of epithelial function.

The sections on leaky epithelia, tight epithelia, and models of epithelial transport are more restricted in content and are heavily directed to ion transport, with only brief references to water transport mechanisms. An elegant study in the section on models of epithelial transport is by Schultz *et al.* on the mechanism of sodium entry across the apical