classic morphological features are absent in the smaller African apes that nonetheless habitually engage in knuckle-walking behavior, then clearly such morphologies are neither biomechanically required for that behavior, nor can their absence in comparably sized fossil forms be taken as definitive evidence of a lack of this locomotor behavior.

Why has this pattern of morphological variance become misconstrued into the frequently heard statement that the "knuckle-walking complex of features" are all functionally correlated and always occur together in identical expression in chimpanzees and gorillas? The problem may lie in poor character analysis and the difficulties inherent in describing features qualitatively, particularly when these features exhibit variable expression. Continuous features are better suited to a quantitative perspective such as that provided by allometry or other approaches (*5*).

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2. J. T. Stern and R. L. Susman, *Am. J. Phys. Anthropol.* **60**, 279 (1983).
3. R. L. Susman, *ibid.* **50**, 215 (1979).
4. _____ and N. Creel, *ibid.* **51**, 311 (1979).
5. P. S. Cranston and C. J. Humphries, *Cladistics* **4**, 72 (1988).

Response: Shea and Inouye provide interesting additional information relevant to the question of African ape-human relations. Knuckle-walking is a shared behavior of African apes, which, if my phylogenetic hypothesis is correct, must have evolved before the gorilla clade diverged from the chimp-human clade, or in parallel in each African ape. Shea, Inouye, and I agree that there is no evidence for the absence of knuckle-walking in the common ancestors of African apes and humans. That knuckle-walking "characters" are variable in their expression in extant knuckle-walkers means not only that the absence of these features in australopithecines does not rule out the possibility that they were knuckle-walkers, as Shea and Inouye suggest, it also reinforces the idea that knuckle-walking has a long ancestry and may have evolved idiosyncratically in separate lineages (*1*). However, it does not follow, in contrast to what Shea and Inouye seem to be suggesting, that knuckle-walking may have characterized the australopithecines. Variability in these traits simply means that they can be misleading as predictors of positional behavior. Much evidence exists indicating that australopithecines were not knuckle-walkers but bipeds, independent of the fact that they lack characters seen in some knuckle-walkers. I have suggested that some form of knuckle-walking or proto–knuckle-walking characterized the last common ancestor of African apes and humans. African apes diverged minimally but independently from this pattern, which could account for the variability in knuckle-walking characters Shea and Inouye note.

There are several reasons to think that humans evolved from knuckle-walkers. Humans and African apes share an os centrale fused to the scaphoid, more robust metacarpal and phalangeal shafts, larger and longer intermediate relative to proximal phalanges, and greater morphological and functional differentiation of manus and pes, all compared with orangs and most other primates. These characters are arguably synapomorphies and plausibly related functionally to both knuckle-walking and power grip. In addition, if one assumes that knuckle-walking is homologous at some level in African apes (the null hypothesis), it is more parsimonious to suggest that it is primitive for the African ape-human clade than to posit a third, unknown positional behavior from which both knuckle-walking and bipedalism arose independently.

A note of correction: In my report I cited a paper by J. Rogers (reference 6) in support of the conclusion that chimps are more closely related to humans than to gorillas. In fact, in this paper on molecular systematics, Rogers indicates that this trichotomy cannot currently be resolved (*2*). A sentence to that effect was inadvertently left out of the final manuscript. In addition, reference 35 should have read, "P. Andrews and L. Martin, *Philos. Trans. R. Soc. London Ser. B* **334** (1270), 199 (1991)."

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Conservation: Should Drug Companies Share in the Costs?

The argument has been made that pharmaceutical companies owe compensation to Third World countries for drugs derived from organisms initially provided by these

countries and specifically that such industries owe back "royalties" to source nations from drugs already on the market. It has been implied, for example, that Eli Lilly and Co. have an outstanding indebtedness to the original providers of the *Vinca* plant from which the company derived the profitable anticancer drugs vinblastine and vincristine (News & Comment, 19 June, p. 1624). Such implication has been persuasively refuted by Irving S. Johnson (Letters, 14 Aug., p. 860) and Carl Djerassi (Letters, 9 Oct., p. 203).

In our view the debate is improperly focused. The question is not whether the pharmaceutical industry is retroactively indebted for its successful development of natural products, but whether, as a matter of self-interest, it should henceforth help bear some measure of the cost of the custodianship of nature. As exemplified by the discovery of "miracle" drugs such as ivermectin, cyclosporin, and taxol, organisms are continuing to yield compounds of unforeseen structure and activity, such as could not, on the basis of existing knowledge, have been "invented" by design. Yet organisms are being lost to extinction faster than they can be studied chemically. If biodiversity is to be preserved for future chemical prospecting, the tide of extinction will need to be stemmed. The pharmaceutical industry, as a major beneficiary of the prospecting effort, should participate in the financing of conservation. Following the lead of Merck & Co., which in exchange for prospecting rights in Costa Rica is helping support the broadly conceived Costa Rican conservation program (Research News, 22 May, p. 1142), pharmaceutical companies should enter into regional agreements worldwide aimed at integrating chemical exploration with biotic preservation. Governments should provide tax incentives to encourage the industries' involvement in such ventures.

President-elect Clinton, in his 1992 Earth Day speech at Drexel University, proposed what will one hopes provide the basis for an initiative of his Administration. "We should explore establishing the international equivalent of The Nature Conservancy," he said, "a fund contributed to by developed nations and pharmaceutical companies to purchase easements in the rainforests for medical research. These easements and the profits from new drugs could make not developing the forests more profitable than tearing them down."

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Addendum

It has come to our attention that we inadvertently omitted an important condition from our description of the crystallization of HIV-1 reverse transcriptase in our Research Article "Crystal structure at 3.5 Å resolution of HIV-1 reverse transcriptase complexed with an inhibitor" (26 June, p. 1783) (*1*). Reverse transcriptase crystals were grown at 4°C. We apologize for any confusion our omission may have caused.

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Alliteration Accidental?

With regard to the Meeting Briefs "Chemists cluster in Chicago to confer on cagey compounds" by Anne Simon Moffat (Research News, 16 Oct., p. 400): Chemists clustering in Chicago? Conferring on cagey compounds? Common citizens complain! Closemouthed chemistry colleagues can cogitate on circular clustering, but care not for cagey compounds. Chicago? Were congressing chemists considering cagey clustered carbon compounds created in the Chicago conflagration? C's confuse! Call the copywriter!

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Corrections and Clarifications

The explanation of the images in the Table of Contents for the issue of 27 November (p. 1415) keyed to the report by D. S. Faber *et al.* (p. 1494) should have read "Stochastic behavior of glycine receptors," not "glutamate receptors."

The image in the Table of Contents for the issue of 23 October (p. 523) illustrating the report by G. D. Schellenberg *et al.* (p. 668) and the Research News article by Jean Marx (p. 550) about early-onset familial Alzheimer's disease being linked to chromosome 14 was incorrect. The correct figure appears on page 668 of the same issue.