Estimates of Coastal Populations

workers has dropped steeply. If this trend continues, the entire discipline will dwindle rapidly to extinction. The same article proposes a way to help rejuvenate the discipline: an international program to support 20 up-and-coming young paleontologists a year, at an estimated annual cost of \$1 million. I note with irony that according to the article on the facing page (News & Comment, 10 Oct., p. 218), McDonald's Corporation has just donated funds for a fossil-preparation lab at the Field Museum of Chicago, where Sue, an exceptionally well-preserved Tyrannosaurus rex, will be readied for display. The price the museum paid for Sue-\$7.6 million-could have provided the entire discipline of paleontology with a much-needed shot in the arm for almost a decade. The message of this juxtaposition of articles rings clear. There are potentially huge sources of funding out there. It is up to us to convince them that their money is better spent, not on the rocks, but on the people who find them, interpret them, and give them their scientific and cultural value.

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The excellent article by Peter M. Vitousek et al. (25 July, p. 495) states: "The human population is concentrated near coastsabout 60% within 100 km [kilometers]. . . ." This assertion comes from a recent World Resources Institute report (1, p. 254): "An estimated 60 percent of the global population lives within roughly 100 kilometers of the shore." This statement comes from a 1990 book (2, p. 7): "Probably 60 percent of humanity, or nearly 3 billion people, live on or within 100 km of a sea coast." The author of the latter book, Don Hinrichsen, an environmental journalist, informed one of us (J.E.C.) (3) that this statement was his "own assessment based on extensive backof-the-envelope estimates using the best available data at the time."

More recently, Hinrichsen reported (4, p. 39): "Nearly two-thirds of the world's people make their homes within 150 kilometers of the shore."

To our knowledge, the first public digital map of the world's human population appeared in 1995 (5). The data are freely available on the World Wide Web (6). These data make it possible to obtain more precise estimates of coastal populations as of 1994. As part of larger research pro-

grams, two groups with interests in the interactions between the earth sciences and the social sciences have independently co-registered the global digital population map on digital maps of coastlines. Using the digital World Vector Shoreline (7), two of us (C.S. and J.E.C.) estimate that approximately 37% (2.07 billion) of the 1994 population (5.62 billion) lived within 100 km of a coastline, and approximately 44% (2.45 billion) within 150 km of a coastline. These percentages are lower than those of Hinrichsen. In addition, C.S. and J.E.C. estimate that 49% of the 1994 population lived within 200 km of a coastline, and 66% within 400 km.

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Using the boundaries (compounded from various sources) provided by the gridded population of the world (6) and a different algorithm for computing distance to coastline, three of us (A.M., J.G., and J.S.) estimate that 37% lived within 100 km of a coastline. This estimate agrees with that of C.S. and J.E.C. Both groups anticipate that these estimates could be refined by the use of better data and methods.

While our estimates of coastal population size are considerably smaller than Hinrichsen's, we agree that very large numbers of people affect and are affected by coastal zones.

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Response: We thank Cohen *et al.* for their analysis, which should become the standard statement on coastal populations.

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Genetics of Parkinson's Disease

The identification by Mihael H. Polymeropoulos et al. of an Ala53Thr alteration in the α -synuclein gene in persons with autosomal dominant Parkinson's disease (PD) provides support for the genetic basis of PD (Reports, 27 June, p. 2045). Because the identical alteration was found among four "unrelated" families [one Italian (Contursi) and three Greek kindreds], Polymeropoulos et al. suggest that this genetic alteration is causative. This mutation nevertheless appears to be rare in familial PD, as others have not detected linkage to 4q21-q23 in sizable series of PD pedigrees, except for one (family K), where it remains unclear whether or not family K is linked to 4q21-23 (1). Assuming that the linkage of the Contursi kindred to 4q21-q23 is valid, we are concerned that this molecular alteration may not be the disease-causing mutation, but represents a neutral variant in linkage disequilibrium with a neighboring PD disease gene.

Factors including selection, admixture, finite population size, migration and mutation, co-ancestry, genetic hitchhiking, and growing population can affect linkage disequilibrium (2). Contursi, in the Salerno province, lies close to the port of Naples on the west coast of Italy. Close contact between Greece and Italy has occurred through the port of Naples for centuries. Thus, it is possible that these four kindreds are distantly related (co-ancestry) and that the Ala53Thr alteration represents an α -synuclein polymorphism in allelic association with a neighboring PD disease gene. Other neurological disorders, such as idiopathic torsion dystonia and Machado-Joseph disease, demonstrate linkage disequilibrium between microsatellite markers and the disease gene among different national populations (3).

The mutated residue is not evolutionally conserved, in contrast with adjacent residues, which are conserved between species. The "mutant" human sequence has a threonine at residue 53 like the wild-type rodent sequence. Thus, the sequences are identical in this domain of the protein. We know of no precedent for a pathogenic mutation to result in conversion of the human amino acid sequence "back" to the rodent amino acid sequence.

Notably, α -synuclein is found in Lewy bodies, the pathological hallmark of PD (4). However, as many other proteins (for example, neurofilament, and ubiquitin) are present in Lewy bodies (4), the presence of α -synuclein, although intriguing, does not prove that α -synuclein is a candidate PD disease gene. The report by Polymeropoulos

