crease the benefits of compact fluorescents in some applications in cold climates, there is no doubting their benefits in warm climates, since the decreased air-conditioning load for indoor bulb substitutions is a fringe benefit. Regardless of climate, a great advantage of compact fluorescents is in replacing outdoor lighting (for example, night lights) where a 13-watt fluorescent bulb can be substituted for a 100-watt incandescent bulb with no noticeable degradation of light quality or quantity.

Five years ago, over 500 13-watt fluorescents were used to replace 100-watt incandescent outdoor lights at a condominium office complex in San Antonio, Texas. The electric bill dropped dramatically, from about $1100 per month to $200 per month; the investment paid for itself in about 6 months; and the complex has saved over $50,000 on electric bills and incandescent bulb replacement costs.

When one takes a systems viewpoint, the value of compact fluorescents is even more impressive. If every one of the approximately 100 million households in the United States replaced one 100-watt incandescent night light with one 13-watt fluorescent (assuming they are on 8 hours a night), the annual electric demand reduction would be the equivalent of 17 coal plants (500 megawatts each); the annual cooling water saved would be about 136,000 acre feet (5.46×10^{16} liters, enough to supply the water for a city of about 750,000 people); and the annual atmospheric release of at least 2.4×10^7 tons (2.18×10^{10} kilograms) of carbon dioxide would be prevented (estimate based on western coal; more carbon dioxide would be released for eastern coal, which has a higher carbon content).

GEORGE M. BARNWELL
University of Texas Health Science Center, San Antonio, TX 78284

EPA Committee

I was concerned to read the description published in ScienceScope of 22 February (p. 863) regarding the Environmental Protection Agency (EPA) subcommittee reviewing the EPA document on electromagnetic fields (ELF) and cancer. The data in the EPA report consist primarily of epidemiology, animal studies, and possibly related cell biology studies. The article indicates that physicists are not welcome on the committee and that the committee is made up of epidemiologists, cancer specialists, and biostatisticians. The article quotes the director of the EPA Science Advisory Board, an unidentified EPA staffer, and Ken Foster, an admitted skeptic about ELF effects.

Several of the committee members have extensive interdisciplinary experience in bioelectromagnetics, while others are new to the topic. The composition of the committee by doctoral discipline is as follows: engineering, four; epidemiology, three; cancer, three; biostatistics, two; empirical biology, two; biopsychology, one; environmental chemistry, one; and theoretical physics, one. Four engineers and one physicist represent physical sciences on the committee (29%). Seven of the members (three engineers, two biologists, one epidemiologist, and one biopsychologist) have experience in nonionizing ELF research. Five members of the committee (two engineers, one epidemiologist, and two laboratory biologists) are cited in the EPA report.

MARY ELLEN O'CONNOR
Department of Psychology, University of Tulsa, Tulsa, OK 74104-3189

Crystal Structure of Bee-Venom Phospholipase A_2: Correction

We would like to call attention to some errors in our report "Crystal structure of bee-venom phospholipase A_2 in a complex with a transition-state analogue" (14 Dec., p. 1563).

1) The legend to figure 2B should have read ". . . bee-venom PLA_2 (blue) superimposed upon that of the uninhibited form of the bovine pancreative PLA_2 (red)."

2) The sequence used to interpret the structure of the bee-venom phospholipase A_2 and cited in figure 2A is in error and differs from that inferred from the published cDNA sequence as follows. Leu should have been Glu at position 20, Arg should have been Lys at positions 47 and 72, Thr should have been His at position 56, Ala should have been Asp at position 92, and Arg should have been Thr at position 57. Lys was typographically omitted at position 124. Re-refinement of the structure with the corrected sequence neither alters the backbone conformation nor affects the disposition of surface residues 20, 47, 72, and 92. Thr57 replaces Arg57 in forming the hydrogen bond with the nonbridged oxygen of the inhibitor's *sn*-3 phosphate, and His56 replaces Thr56 at the opening of the hydrophobic channel. These changes do not alter our conclusions or those of its companion papers on phospholipase A_2. Corrected stereopairs (figures 3 and 4) are available from the authors on request, and the revised coordinates have been deposited in the Brookhaven Protein Data Bank.

DAVID L. SCOTT
ZBYSZEK OTWINOWSKI
Department of Molecular Biophysics and Biochemistry, and
Howard Hughes Medical Institute, Yale University, New Haven, CT 06511
MICHAEL H. GELB
Departments of Chemistry and *Biochemistry, University of Washington, Seattle, WA 98195*
PAUL B. SIGLER
Department of Molecular Biophysics, and Biochemistry, and
Howard Hughes Medical Institute, Yale University

NOTES

1. We thank T. P. King, Rockefeller University, New York, and G. Kreil, Austrian Academy of Sciences, Salzburg, Austria, for pointing out error 2.

Membrane-Bound Phosphotyrosine Phosphatases

Jean L. Marx reviews the discovery of integral membrane phosphotyrosine phosphatases (Research News, 15 Feb., p. 744) and rightly describes it as a tale of two sequences. However there are omissions on the CD45 side of the story. The sequencing of CD45 was first done for the rat thymocyte molecule at our laboratory (*1*), and the unusual features of the cytoplasmic domain, including the presence of two tandemly repeated similar domains, constituted a major point of interest. In parallel work, cDNA clones encoding extracellular sequence of mouse CD45 were reported by the laboratory of Ted Boyse (*2*), and the subsequent work on CD45 and its related molecules has been done on the basis of the rat or mouse cDNA clones.

CD45 was worth sequencing to resolve questions of molecular heterogeneity, but above that we thought it must have a key function and that having the sequence might bring about a convergence of fields. This happened in 1988, when Charbonneau *et al.* (*3*) sequenced a placental phosphotyrosine phosphatase. Long live serendipity!

ALAN F. WILLIAMS
Cellular Immunology Unit, Medical Research Council, Sir William Dunn School of Pathology, University of Oxford, Oxford OX1 3RE, England

REFERENCES

1. M. Thomas, N. Barclay, J. Gagnon, A. F. Williams, *Cell* **41**, 83 (1985).
2. N. B. Shen *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **82**, 7360 (1985).
3. H. Charbonneau, N. K. Tonks, K. A. Walsh, E. H. Fischer, *ibid.* **85**, 7182 (1988).