Biochemistry. In the article "Reductive detoxification as a mechanism of fungal resistance to singlet oxygen-generating photosensitizers" by Margaret E. Daub, Gary B. Leisman, Rose A. Clark, and Edmond F. Bowden, which appeared in number 20, October 15, 1992, of *Proc. Natl. Acad. Sci. USA* (**89,** 9588–9592), Fig. 2 was poorly reproduced. The figure and its legend are shown here.

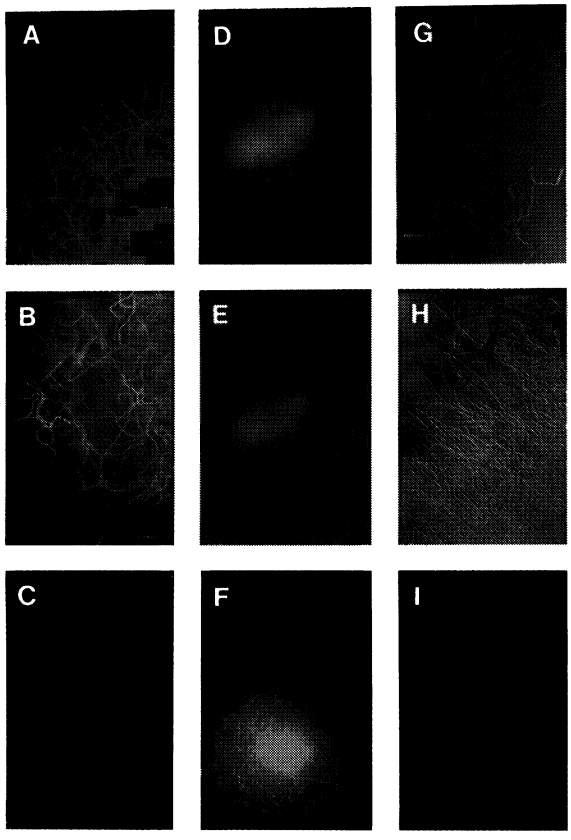

FIG. 2. Fluorescence microscopy of *C. kikuchii, C. nicotianae,* and *Asp. flavus* incubated with photosensitizers. (*A*) Cercosporin-producing culture of *C. kikuchii* viewed with the 530 filter (specific for reduced cercosporin); green fluorescence of hyphae indicates cercosporin reduction. (*B*) Same culture viewed with the 605 filter; yellow fluorescence indicates cercosporin reduction. (*C*) Same culture killed by exposure to chloroform vapor and viewed with the 605 filter; red fluorescence indicates lack of cercosporin reduction. (*D*) *Asp. flavus* incubated with cercosporin and viewed with the 530 filter; reduced cercosporin was present only at colony center. (*E*) Same as *D* except photographed with both brightfield and fluorescence, allowing visualization of nonfluorescing hyphae. (*F*) *Asp. flavus* viewed with the 605 filter; reduced cercosporin (yellow fluorescence) was present in the colony center but not (red fluorescence) at the actively growing periphery. (*G* and *H*) Fluorescence and interference contrast views of a non-cercosporin-producing culture of *C. nicotianae* incubated with eosin Y; lack of fluorescence indicates reduction of eosin Y. Arrowheads point to same hyphal strand in *G* and *H*. (*I*) *C. nicotianae* incubated with rose bengal; red fluorescence from rose bengal indicates lack of reduction of this compound. (A–C and G–I, ×50; D–F, ×25.)

Biophysics. In the article "Direct measurement of brain glucose concentrations in humans by ^{13}C NMR spectroscopy" by Rolf Gruetter, Edward J. Novotny, Susan D. Boulware, Douglas L. Rothman, Graeme F. Mason, Gerald I. Shulman, Robert G. Shulman, and William V. Tamborlane, which appeared in number 3, February 1, 1992, of *Proc. Natl. Acad. Sci. USA* (**89,** 1109–1112), a more complete fit of the data points in Fig. 3 results in $K_t = 4.9 \pm 0.9$ mM, $T_{max}/V_{gly} = 3.6 \pm 0.1$, and $T_{max} = 1.1 \pm 0.1$ μmol/g·min, which are not statistically different from the values given and do not affect the conclusions in the paper. Fig. 3 should be replaced by the figure shown below.

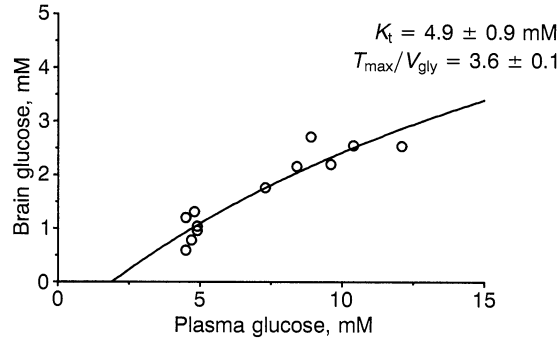

FIG. 3. Determination of the kinetic parameters from the brain glucose concentrations (μmol per ml of brain vol) measured at euglycemia and hyperglycemia in six normal individuals. The model requires that at steady state the rate of glucose influx across the blood–brain barrier, T_{in}, equals the sum of the rate of efflux, T_{out}, plus the rate of metabolism, V_{gly}. If T_{in} and T_{out} are assumed to have the same Michaelis–Menten kinetics (8, 15, 34), the steady-state relationship between brain and blood glucose concentrations is given by ($x = [\text{glucose}_{plasma}]/K_t$; $v = T_{max}/V_{gly}$)

$$[\text{glucose}_{brain}] = K_t \times \{[x(v - 1) - 1]/(x + 1 + v)\}. \quad [1]$$

This equation was fitted to the data points (solid line) as in previous studies (15, 16) of animals in order to calculate the apparent Michaelis–Menten constant of transport. K_t, as well as the ratio T_{max}/V_{gly} of the apparent maximal glucose transport rate, T_{max}, to the glucose consumption rate V_{gly}.