Letters

Misuse of the Freedom of Information Act

Three times within the last several years individuals, through the Freedom of Information Act, have obtained the narrative of my grant proposals funded by the National Institutes of Health. One request was from a senior investigator at a major university, one from an industrial scientist at a large commercial firm, and one from a junior staff member of a national laboratory. The requesters did not send their requests to me directly nor did they inform me of their interest in my work. Were it not for the policy of NIH to inform the grantee when such requests are received, I would not have known that my grant narratives had been requested.

I strongly support the Freedom of Information Act. I can also appreciate the rationale that grants, once funded, become part of the public domain and should be available for scrutiny by interested parties concerned with the appropriate spending of federal moneys. In the three instances of my personal experience, I strongly doubt that this is the case. Rather, it seems more likely that individuals have sought this information for their own purpose and not to ensure that governmental processes are carried out under public observation. Although I am usually eager to share my thoughts and ideas, I do not believe that grant proposals are an appropriate vehicle for scientific dialogue.

The misuse of the Freedom of Information Act, as I believe these instances are, should be a matter of concern for the scientific community. Not only does this increasing misuse contribute to a degradation of the collegiality ostensibly underpinning our scientific communication, it also represents a practice that could perturb the integrity of the peer-review process for research proposal funding. I suggest that a policy might be adopted whereby requests for grant narratives would be supplied by NIH only if the requester has not been successful in obtaining these documents directly from the originator of the grant proposal. This would ensure that the requester would be required to justify to the author the need for the information. If agreement cannot be reached between author and requester, then it seems appropriate to obtain the grant from NIH.

JERRY R. WILLIAMS
Johns Hopkins Oncology Center, 600 North Wolfe Street, Baltimore, MD 21205

Rüdenberg's Patents

The statement in Arthur L. Robinson's excellent account of the Nobel awards for the electron microscope (Research News, 14 Nov., p. 821), that the German patent office did not grant a patent to Reinhold Rüdenberg, is incorrect. Eight German patents that bore Rüdenberg's name were issued after World War II to the German company Siemens-Schuckertwerke.

Rüdenberg's U.S. patent was found to be adequate in litigation, where he successfully won ownership of two U.S. patents from the Alien Property Custodian after wartime confiscation from Siemens (*1*). I can find no record of any Rüdenberg patent infringement suit against RCA (*2*), as mentioned in the article.

The award of the Nobel Prize a half-century later and many years after all of the Rüdenberg electron microscope patents (*3*) had expired does not support Robinson's speculation that the U.S. patent office made a mistake in its grants to Rüdenberg. It appears that Siemens and Rüdenberg complied with the patent laws of six countries. At the same time there is no doubt that Ruska deserves the Nobel honor for his fundamental work in electron optics and for his independent invention, design, and building of the first electron microscope.

JOHN L. HUMMER
Post Office Box 2160, Reston, VA 22090

REFERENCES AND NOTES

1. Rüdenberg v. Clark, 72 Fed. Suppl. 381 (Mass. Dist. Ct., 1947).
2. See Rüdenberg v. Clark, 81 Fed. Suppl. 42, 43–46 (Rüdenberg license proffer to RCA) (Mass. Dist. Ct., 1948).
3. The Rüdenberg patents are listed in M. M. Freundlich's article [*Science* **142**, 185 (1963)].

Quality of Biomedical Literature

In view of the current controversy as to whether scientific fraud is increasing, let me suggest that the insidious rise in publication costs and subtle changes of editorial policy and attitude are having serious effects on the quality of the biomedical literature.

As an occasional reviewer for several journals, I frequently find myself requesting additional data and controls. I am aware that my suggestions are often forwarded to the authors accompanied by a recommendation from the editor, understandably concerned over publication costs to the journal, for an abbreviation of the text. The author is thus faced with the impossible chore of supplying more data in less space. In general, he opts for cutting the text and assuring the editor that the requested controls have been performed to an extent that would satisfy even the most critical. But the data are not shown. The reviewer is then presented with the unenviable task of accepting the revised manuscript or imputing the integrity of the author.

As a separate issue, it would appear that some of our leading journals have established as policy to accept frankly incomplete manuscripts if they are judged scientifically exciting. These same journals often reject well-documented work under the pretext that it lacks sufficient general interest, particularly when a preliminary report on a similar topic has appeared elsewhere.

Add a growing public perception that truth encompasses all that is not explicitly false, and the message to young investigators is clear. Give us your half-baked ideas and spare us the boring details. At least 10 percent of what I read today in our leading journals, while certainly *not* fraudulent, is, however, incomplete, inadequate, and even incompetent.

In this milieu, if scientific fraud is not increasing, it will be. The victims will be all of us.

ROBERT G. MARTIN
Section of Microbial Genetics, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD 20892

Quantitative Risk Aspects of the "Woburn Case"

I was amused to read Daniel E. Koshland, Jr.'s editorial calling for reason in the area of toxic substances and the environment (24 Oct., p. 409), not only because of its usual tongue-in-cheek humor, but also because it was followed a few pages later by a discussion of an excellent example of irrationality in an environmental health issue, namely the "Woburn case."

In his well-balanced article, Eliot Marshall (News & Comment, p. 418) describes the background, outcome, and scientific issues of this case. Not discussed, however, are the quantitative risk aspects, which show (i) that it is highly unlikely that the reported levels of pollution in public wells "G" and "H" could have caused the elevated leukemia rate in Woburn, Massachusetts, and (ii) that drinking the well water presented no more hazard than consuming ordinary chlorinated U.S. tap water.

The calculations that allow one to reach these two conclusions are based on the measured levels of trichloroethylene, perchloroethylene, and chloroform in well G

(the most contaminated well) (*1*), Environmental Protection Agency carcinogenic potency values (*2*), and an assumed consumption of 2 liters per day. The "hazard index" (*3*) for well G water is 9.1 from the organohalogens present versus 11.6 for U.S. tap water due to chloroform alone. Thus, unless the contamination of the wells before 1979 was orders of magnitude higher than that reported in (*1*) there seems little likelihood that the contamination could have caused the elevated leukemia rate in Woburn.

As for the arguments quoted in the article relating toxic substances to immunosuppression and human cancer, they are clearly ludicrous with respect to such low concentrations. If we are to stop the nonsense that is now persisting in toxic substances litigation, it is time for respected toxicologists and public health professionals to work together with engineers and other interested parties to develop a rational plan to deal with and prevent the potential health risks caused by the contamination of ground water and drinking water with toxic substances.

MARTYN T. SMITH
Department of Biomedical and Environmental Health Sciences, School of Public Health, University of California, Berkeley, CA 94720

REFERENCES AND NOTES

1. G. S. Parker and S. L. Rosen, *Woburn: Cancer Incidence and Environmental Hazards 1969–1978* (Department of Public Health, Commonwealth of Massachusetts, Boston, 1981).
2. *Health Assessment Document for Trichloroethylene, Final Report* (EPA/600/8-82/006F, Environmental Protection Agency, Washington, DC, 1985), pp. 8–133.
3. The "hazard index" is a comparative risk number generated by multiplying the daily dose in micrograms from drinking 2 liters of water by the carcinogenic potency of the chemical in rodents. Average concentrations of trichloroethylene, perchloroethylene, and chloroform in well G were 262, 26.4, and 4.3 μg per liter, respectively (*1*). U.S. chlorinated drinking water supplies contain chloroform at a mean concentration of 83 μg per liter [S. J. Williamson, *Sci. Total Environ.* **18**, 187 (1981)].

Moonlight and Circadian Rhythms

Charles A. Czeisler *et al.* (Reports, 8 Aug., p. 667) may have used much more light than necessary to affect human circadian rhythms. There is evidence that significantly lower light levels, under the proper conditions, can have noticeable effects. Consider, for example, a folk belief still prevalent in the Shetland Islands around the turn of the century that illustrates this.

These islands lie between 60 and 61 degrees of north latitude (200 kilometers north of the Scottish mainland), or only 6 degrees south of the Arctic Circle. In the winter, daylight is only a few hours long and, until recently, artificial light was a luxury. The winter moon, as high in the night sky as the sun is in the summer, is particularly prominent there. A strongly held belief of the Shetlanders was that moonlight should never fall on the face of a sleeping person (*1*). Being unused to having unexpected periods of light during the long winter nights, they had presumably come to notice the unsettling effect such light could have when it happened to shine at a "sensitive" time in the circadian cycle. The full moon (about 0.3 lux) is 3×10^{-6} as bright as the midday sun (*2*). It would thus supply about 4×10^{-5} as much light as that employed by Czeisler *et al.* If it shone on a sleeper for (typically) an hour, the integrated intensity of nocturnal light that so worried the Shetlanders would be no more than 10^{-5} of that employed in these recent experiments.

This suggests that much lower light levels could be used to probe the effects on humans. These lower levels are, interestingly enough, those already found ample to affect circadian pacemakers in a variety of creatures (*3*) and to show an effect on the human menstrual cycle (*4*). It is possible that the accumulated experience that led to this "folk wisdom" could have contributed to the widespread importance accorded the moon in prehistoric Britain (*5*).

ROLF M. SINCLAIR
Physics Division, National Science Foundation, Washington, DC 20550

REFERENCES AND NOTES

1. This belief was voiced on numerous occasions by my mother, the late Elizabeth Stout of Shetland.
2. See, for example, G. Abell, *Exploration of the Universe* (Holt Reinhart, New York, ed. 2, 1969), chap. 20.
3. A. Winfree, *Phys. Today* **28**, 34 (March 1975) cites the critical time and intensity as "close to (subjective) midnight and . . . equivalent to a few minutes' full moon!"
4. E. M. Dewan, *Sci. Technol.* (No. 85) (January 1969), p. 20.
5. E. Hadingham, *Early Man and the Cosmos* (Walker, New York, 1984), chap. 5.

Response: Light of very low intensity can indeed affect circadian pacemakers, particularly in plants and insects (*1*). In fact, exposure to light with a mean flux of 5 photons per second per eye for 12 hours alternating with 12 hours of darkness is sufficient to entrain wheel-running activity rhythms to a 24-hour period in the nocturnal cockroach (*Periplaneta americana*) (*2*). As we stated in our report, it had originally been reported that a light-dark cycle of ordinary indoor illumination (200 to 500 lux) was insufficient to similarly entrain human circadian rhythms (*3*), but we had subsequently shown that when such human studies were conducted in a manner comparable to the animal studies, entrainment to a 24-hour day could be achieved with ordinary room illumination (*4*). However, studies in other species indicate that the amplitude of the response of the circadian system to light signals is related to the intensity, duration of exposure, and circadian phase of administration. On the basis of prior studies in humans, it was difficult to determine whether indoor light had a direct synchronizing effect or a behaviorally mediated one. We therefore attempted to determine whether relatively brief exposure to bright (about 10,000 lux) light comparable in intensity to natural sunlight (which reaches over 100,000 lux at midday) could reset the human circadian pacemaker, even when the timing of the sleep-wake cycle was held fixed. The data from the case study we reported, which we have subsequently confirmed and extended in eight trials in three other subjects, demonstrates that the effect we observed requires bright light, since it did not occur in control studies when these subjects were instead exposed to ordinary indoor light of 50 to 250 lux. Thus, we would not expect moonlight [about 0.3 lux (*5*)] to have effects similar to that of the bright light used in our study in individuals living in temperate or equatorial latitudes. It is conceivable that the circadian system of individuals living in constant darkness might become more sensitive to resetting by lower intensity light, as has been reported in *Drosophila* (*6*). However, the occurrence of midwinter sleep-onset insomnia during the "dark period" in Tromsø, Norway (about 69° latitude), when people depend completely on artificial indoor room lighting, and its reported improvement following exposure to morning bright light (2500 lux) (*7*) again suggests that in humans, ordinary indoor illumination is a weaker synchronizing cue than light of greater intensity.

CHARLES A. CZEISLER
JAMES S. ALLAN
Neuroendocrinology Laboratory, Division of Endocrinology, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 221 Longwood Avenue, Boston, MA 02115

REFERENCES

1. E. Bünning, *The Physiological Clock* (Springer-Verlag, New York, ed. 3, 1973), pp. 94–97.
2. M. I. Mote and K. R. Black, *Photochem. Photobiol.* **34**, 257 (1981).
3. R. Wever, *Pfluegers Arch.* **321**, 133 (1970).
4. C. A. Czeisler, G. S. Richardson, J. C. Zimmerman, M. C. Moore-Ede, E. D. Weitzman, *Photochem. Photobiol.* **34**, 239 (1981).
5. L. Thorington, *Ann. N.Y. Acad. Sci.* **453**, 43 (1985).
6. A. T. Winfree, *J. Comp. Physiol.* 77, 418 (1972).
7. O. Lingjaerde, T. Bratlid, T. Hansen, Å. Sykehus, *Acta Psychiatr. Scand.* **71**, 506 (1985).