dle group over which the outside expert referees have disagreed. In the face of such controversy, the *Journal's* Board, acting as advocate for a general readership (and believing that it need not regret so much the rejection of a good paper as the publication of a poor one), frequently responds to the challenge by declining to go out on a limb. This conservative posture leads more often than desired to the publication of "safe" (not very exciting) pieces, by "safe" (established) authors. It seems likely that the same sin is shared by our great lay publishing houses.

The concept of the "established author" has recently been analyzed by a sociologist considering the literature of "hard science" (chemistry and physics) and crystallized under the title of "The Matthew effect,"[1] which, paraphrasing the parable of the talents (according to St. Matthew[2]), notes simply that the big get bigger and the small get nowhere. The same principle had previously been demonstrated in the fine arts by a contemporary painter whose work was sold (at handsome prices) in the style and over the name of Vermeer. Recognizing this effect also, the *Journal* for some years attempted to review all submissions under a double-blind system whereby author and reviewing referee were mutually anonymous. This system inevitably broke down under the persistent weight of authors whose identity was regularly disclosed by their subject matter, bibliographic citations and well labeled illustrations as well as of those referees who considered their objective integrity impugned thereby. The Matthew effect thus remains a grammar that cannot be fully parsed.[3]

But what of the *un*-established author *sans patron* who has no Rubens to apply a final brush stroke and announce the work as being from an accepted *atelier?* And what of the unknown monk, counting peas, who may be ahead of his time? How can he be heard?

Occasionally, the Board of the *Journal* is treated to a contribution so fresh and yet so outlandish as to cause consternation about its possible publication. Mostly, the idea submitted is sufficiently removed from medicine's mainstream that the author can be dismissed with the relieved but uncertain comment, "He's a nut (or he's right)," and forgotten.

Sometimes, however, as is apparent in the article by Tullis published in this issue of the *Journal*, the question is simply too important not to be brought out into the daylight of public scrutiny. Such illumination soon forces confirmation or denial by others of the reproducibility of the data and validity of the allegation.

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## GOOD LIGHT AND BAD

WISER prophets than we have proclaimed that the 1970's will be the decade when human ecology attains its rightful place in the hierarchy of medical sciences. Spurred by mounting concern about the long-term consequences of smog, food additives, water pollution and, most recently, noise, broad public support is becoming manifest for programs to explore the effects of environmental inputs on human health, and to limit the ability of individuals to modify the environment in which all of us live.

Coinciding with this general growth of interest in environmental factors, recent years have also witnessed a striking increase in our knowledge of the biologic effects of one particular factor: light. It is now recognized that visible and ultraviolet radiations influence a variety of physiologic processes in humans. Moreover, light exposure can generate pathologic states in some people (for example, those subject to photosensitive eruptions), but it can also be applied with great effectiveness in certain diseases. Phototherapy is now widely used to treat hyperbilirubinemia in premature infants.[1] Papers appearing elsewhere in this issue present compelling evidence that light exposure will also lower serum bilirubin levels in full-term infants with nonobstructive, nonhemolytic jaundice. The patients are exposed intermittently to "daylight" or blue fluorescent bulbs at relatively low intensities (fewer than one tenth the footcandles present in the shade of a Boston afternoon). One infant so treated has continued to respond after five months of therapy and has displayed no neurologic or ophthalmic complications during this period. Indeed, to date there have also been no reports of toxicity attributable to phototherapy in jaundiced premature infants.

The advent of phototherapy for the treatment of hyperbilirubinemia has an exciting and more general implication: if light can be used to speed the destruction of one circulating compound – bilirubin – it seems not unlikely that light exposure will eventually be shown to influence plasma levels of other compounds. Light may be beneficial in many more situations than are now apparent, but also may already be impairing human health by destroying essential compounds or generating toxic ones. The current situation with respect to light may be analogous to that of x-irradiation at the turn of the century: in certain circumstances x-rays provided a life-saving adjunct to diagnosis and therapy. However, their indiscriminate and uncontrolled use also generated a considerable amount of disease. Currently, a major difference between medical x-irradiation and pho-

totherapy is that most healthy people have no trouble avoiding the former, but seem to have no way of escaping the latter. Most Americans spend much of their time indoors, exposed to artificial-light sources whose spectra differ appreciably from sunlight. For example, "daylight" fluorescent bulbs provide very little long-wave ultraviolet light, and emit yellow and red radiations in a ratio quite different from that present in sunlight. It does not seem wildly imaginative to speculate that prolonged exposure to this unplanned phototherapy might have physiologic consequences. Perhaps it is not too early to suggest that an appropriate federal body give thought to the ultimate necessity of regulating the spectral composition of commercially available light sources. It is certainly not too early to suggest that much additional research should be performed to define the biologic effects of spectra now in use.

Environmental lighting has two types of effects on humans: *direct* effects, mediated by the photochemical responses of molecules present in the skin or subcutaneous tissues; and *indirect* effects, mediated by retinal photoreceptors. The direct effects of light include the erythemal response to ultraviolet light, the stimulation of melanin synthesis, the photolytic decomposition of circulating bilirubin, and the activation of precursors of vitamin D. We have recently examined the effects on calcium absorption of a white-light source whose emissions simulate the solar spectrum in both its visible and its ultraviolet regions.[2] The study suggested that living under such lighting for as little as five weeks perhaps enhances the intestinal absorption of calcium in healthy people. It seems likely that the relatively small quantities of long-wave ultraviolet light emitted by the test lights caused enough additional vitamin D production to facilitate calcium absorption. The data suggest another possible use for phototherapy (or, more properly, photoprophylaxis): to stimulate calcium absorption among people who, for reasons of age, climate or habit, are chronically underexposed to sunlight.

The indirect effect of light about which most information is available is, of course, vision. Retinal responses to environmental lighting also mediate an expanding list of neuroendocrine effects.[3] These include control of pubescence, ovulation and a large number of daily rhythms. If rats, a nocturnally active species, are deprived of photic stimuli (by being blinded or maintained in an environment of continuous darkness), sexual maturation is delayed. Blind humans, in contrast, show an earlier menarche.[4] The mechanism through which light influences sexual maturation is not established; it probably involves the pineal organ but must also use other channels. The daily night-day light cycle generates some 24-hour rhythms in metabolic function (for example, the daily rhythm in the synthesis of melatonin by the pineal[5]) and acts as the dominant synchronizer of most or all daily rhythms. Essentially nothing is known about the identity of the portions of the light spectrum that cause the neuroendocrine and metabolic effects of light. Recent studies have shown, however, that rat gonads grow less rapidly when the animals are exposed to light from regular "cool-white" fluorescent bulbs than to spectra more similar to sunlight.[6] Here, too, therefore, spectra are important, and continued exposure to those differing from sunlight could have notable physiologic effects.

It seems safe to state that, whether we like it or not, light is another thing that physicians must now worry about. The light to which our patients are exposed may be useful, deleterious or, most probably, biologically neutral; however, we can no longer assume that it is any of these things without adequate evidence.

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## MASSACHUSETTS MEDICAL SOCIETY

ROBINSON — Hugh L. Robinson, M.D., of West Newton, died on November 14, 1969. He was in his seventy-second year.

Dr. Robinson received his degree from Harvard Medical School in 1922. He was formerly a medical missionary in North China from 1925-41 and a member of the staff of Newton–Wellesley Hospital. He was a member of the American Medical Association and the American Academy of Pediatrics.

Dr. Robinson is survived by his widow, two daughters, two sons and one brother.

SHERMAN — Joseph Donald Sherman, M.D., of Natick, died on October 20, 1969. He was in his forty-first year.

Dr. Sherman received his degree from Boston University School of Medicine in 1957. He was chief of hematology at Framingham Union Hospital, assistant professor of medicine at Boston University School of Medicine and research associate at Tufts University School of Medicine. He was a member of the American Medical Association and the American Society for Experimental Pathology.