Letters

Cystic Fibrosis Research

Scientific features which focus on personalities invariably cause offense, however balanced the presentation. Leslie Roberts' articles on cystic fibrosis research (Research News, 8 Apr., p. 141; 15 Apr., p. 282) are well written, but I believe some of the quotes in the articles give misleading impressions.

We have published more than 50 refereed papers on the molecular genetics of cystic fibrosis since 1980; if those who allege "shoddy" work were correct, this might be apparent in rejections or referrals. With respect to collaboration, as Roberts states, we have supplied our cystic fibrosis probes for diagnosis, free, to more than 250 noncommercial laboratories (every noncommercial laboratory which has requested them). We have also collaborated (and published refereed papers with) 32 other research groups during the past 8 years. These include the Toronto, Houston, Salt Lake City, and Integrated Genetics laboratories. We value these collaborations greatly; anyone who knows our lab "style" knows that we encourage joint research programs.

We called the gene which is in approximately the right genetic position a "candidate gene" because this has a very specific meaning in human molecular genetics; it is not a euphemism for "the gene," but just what it says, a candidate that must be tested. In 6 months, we sequenced this gene twice, in both directions, from patients with cystic fibrosis and from controls, and did several hundred linkage, Northern, and pulse field blots and many other experiments. As soon as it was clear that the candidate gene is not the one which is mutated in cystic fibrosis, we reported this at the Paris International Gene Mapping Congress, only 4 months after the original paper appeared in *Nature*. Surely this is not an unreasonable time.

Roberts states that "tensions are so bad that a few speculate that [I] misled people intentionally to scare off the competition, an accusation Williamson finds appalling." We are not responsible for the media hype, or for rumors, or for what the National Institutes of Health or the Cystic Fibrosis Foundation do; I am on no grant committees and play no part in any such deliberations.

We are currently engaged in ten major collaborations, mostly with U.S. groups, using our markers to attempt to isolate the real gene. Any group (including ours) tries to avoid working with several other groups on precisely the same project. Collaborations are most difficult between groups which share the same techniques and objectives, such as the Toronto group and ourselves, but we talk to each other often, and relations between the groups are good.

Commercial companies bring problems as well as cash. We do not think that they should profit from public research, and so we ask them for large contributions (to the Cystic Fibrosis Research Trust, not ourselves) if they use our probes or knowledge. This is particularly relevant for cystic fibrosis, since the funding for much of our work comes from families in which the disease occurs, while the companies are interested in population screening—not in developing new methods of treatment, which is unlikely to be profitable. We regard what we obtain from companies for the Cystic Fibrosis Research Trust as a tax, which will be used to develop new forms of treatment for those with the disease, an area which might otherwise be neglected in the scramble for the lucrative screening market.

The publicity which surrounded recent research reports (whether ours or from other labs) often came from the cystic fibrosis charities, who used it to increase interest, awareness, and funds for research and treatment. This is legitimate, and moreover (at least in the United Kingdom) has succeeded: there is far more awareness of cystic fibrosis now than there was 5 years ago, and more money raised for research. The press (including *Science*) personalizes these matters, and while those of us who work in science cringe at this, there are times when we cannot stop it.

All of the groups—Toronto, Salt Lake City, Boston, Houston, London, and so on—have made real contributions to the work; there is a good spirit of collaboration between most of us, most of the time. When the nature of the gene, and the mutation, is finally determined, everyone will be pleased for the sake of those with cystic fibrosis, and their families, whoever has their names on any particular paper.

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I was disturbed by the articles on the scientific "Race for the cystic fibrosis gene."

The Cystic Fibrosis Research Trust, a charity whose funds are raised almost entirely by the families of those suffering from this dreadful disease, has given total support to Williamson's work for the past 9 years and has been greatly heartened by the steady progress towards the gene. We have kept in touch with his progress throughout and are aware of the close international collaboration there has been over the years—indeed, we have funded international meetings in this country specifically for the purposes of exchanging data with many of those mentioned in the articles.

Not only do I feel it unnecessary to set out the blow-by-blow account of the well-known phenomenon of scientific rivalry, as done in the articles I fear it could be positively counterproductive, for while it may be of vicarious interest to some of Williamson's fellow scientists, should it be taken up by the sensation-seeking press, it is likely to cause great distress to those for whom he has worked hardest—those with cystic fibrosis.

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The new *Science* style reflected in the "search for the cystic fibrosis gene" series provides background color usually lacking in the dispassionate review of scientific events. It is true that when an important target is clearly defined the competition can become intense and for a time the competitors may lose perspective. Reflection on the ethical questions raised may be appropriate. My own reflection, however, continues to find unfair the stinging comment from the anonymous reviewer who found our *Nature* manuscript "immoral, but not criminal," apparently because our article threatened the precedence of the work of Lap-Chee Tsui and Collaborative Research in locating the cystic fibrosis gene to chromosome 7. In fact, the existence of such a threat became known to us only very late as events unfolded in November 1985.

We had expected the *Science* article by Tsui *et al.* (*1*) to reveal the chromosome 7 location of the cystic fibrosis gene and to be published well in advance of our article. This would have provided an opportunity for complete reference, establishing scientific precedence. We learned only late in November 1985 that the authors had chosen not to reveal the chromosomal location of cystic fibrosis in that manuscript. As a result, our manuscript, which was intended primarily to describe the identification of a new, very tightly linked DNA marker for cystic fibrosis, did threaten to supercede, as our tightly linked marker had previously been localized

to chromosome 7. It added up to a difficult and awkward situation as we could not properly reference a rumor and the other group had not put their findings into print.

At no time, however, including our initial discussion with the editor of *Nature*, have we been less than candid and forthcoming in recognizing the precedence of the Lap-Chee Tsui–Collaborative Research group in localizing the cystic fibrosis gene to chromosome 7 (*2*). We were indeed relieved when a proper solution was constructed and much appreciated the clarifying *Nature* editorial (*3*) which accompanied the several manuscripts (*4*).

The experience stresses the importance of timely publication of scientific findings and highlights the risk taken in delaying publication in order to maintain a competitive advantage.

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2. R. White, *ibid.* **234**, 1054 (1986).
3. P. Newmark, *Nature* **318**, 309 (1985).
4. R. G. Knowlton *et al.*, *ibid.*, p. 380; R. White *et al.*, *ibid.*, p. 382; B. J. Wainwright *et al.*, *ibid.*, p. 384.

Response: Williamson and I apparently agree on the facts. Readers can judge for themselves whether or not the articles are misleading.

I am puzzled, however, by what Williamson means by the press "personalizing" scientific matters. My article recounts the history of the very important work under way to isolate the cystic fibrosis gene—work that is, after all, done by people.

Nor do I understand why Bentley believes that the families of cystic fibrosis patients will be greatly distressed by the articles. On the contrary, they might be interested in learning how the money they raise for research is spent.—LESLIE ROBERTS

Genesis 1:28

Times have changed, and not for the better. A short 21 years ago Lynn White, Jr., argued in the pages of *Science* (10 Mar. 1967, p. 1203) that the roots of the present ecological crisis could be traced to our unquestioning acceptance of the message in Genesis 1:28. White suggested (p. 1207) that we shelve the Old Testament myth once and for all and recognize as the "patron saint for ecologists" the nature-loving St. Francis of Assisi. And in 1971 Ian McHarg, an ecologist, called the message of Genesis 1:28 "the best guarantee of [our] extinction" (*1*). But now we read (22 Apr., p. 375) a letter from Jonathan H. Cilley, Sr., informing us that the "fundament" of biology is to be found not in the biological theory of evolution, which places human beings on a level with other living beings, but in the Old Testament, specifically in Genesis 1:28, where mankind, created separately by divine fiat, is commanded to "be fruitful and multiply, and fill the earth, and subdue; and rule over every living thing." This, he asserts, constitutes our "cultural mandate." *Quantum mutatus ab illo!*, as Aeneas said when the battered face of Hector returned to him in a dream.

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