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Response: We did not brand Mukerjee an animal rightist, but said that her so-called overview has a strong animal rights bias. We have not changed our view. We do not favor using animals in experiments that do not require their use, nor are we aware of anyone in the scientific community who holds this extreme view. Far from attacking mainstream views, as Rennie asserts, we are expressing the mainstream view, as evidenced by the accompanying letters. We agree with Mallow that middle- and high school students should visit research laboratories, with one clarification added. When given the appropriate factual tools and background to help them understand what they are seeing, students can decide for themselves what constitutes a deception. This is what the Science for Life project is doing.—Deborah Runkle and Ellen Granger

Insulin Gene Patent Litigation

I found Eliot Marshall's article (News, 22 Aug., p. 1028) about the 1977 cloning of the rat insulin gene and the subsequent patent litigation engrossing. However, some remaining uncertainties need to be resolved.

acterized as "smoking guns" were intended to make a record of what transpired, why were the letters and the events they recorded never mentioned in the 14 October 1977 memo by William Rutter and Howard Goodman? The University of California, San Francisco, biosafety committee and the National Institutes of Health (NIH) administrators investigating the events surely would have found the letters directly pertinent. The statement by Rutter and Goodman in the NIH files says nothing about retaining DNA.

2) Was DNA from the original pBR322 experiment retained or not? If so, what was done with it? The chronology is puzzling. It seems that destruction of the original pBR322 clones happened on 19 March and registered letters saying that not all the DNA was destroyed were dated several days later. But the claim seems to be that DNA was neither retained nor used.

3) What are the accession numbers of the pMB9 deposits at the American Type Culture Collection (ATCC) mentioned in the final paragraph of the article? Sequencing the original pMB9 clones might indeed resolve some of the controversy (although, depending on any subcloning process details, it might not). I asked ATCC staff about these, but to date they have not been able to identify any such deposits.

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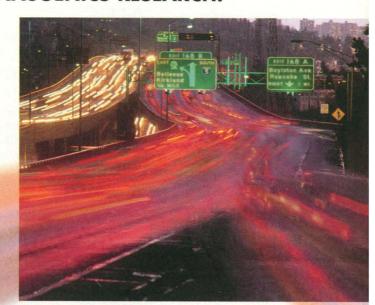
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LETTERS

Robert Mullan Cook-Deegan Director, National Cancer Policy Board, Institute of Medicine, and Commission on Life Sciences, National Academy of Sciences, Washington, DC 20418, USA

Response: After Science went to press, Rutter's attorney called to correct Rutter's suggestion that the 1977 cloning work could be verified by examining the original pMB9 clones, said to be on deposit at the ATCC. Rutter later explained in a note to Science that no such clones exist:

After our conversation, I thought I should check on this point to be sure. Having looked into the matter further, I learned that, in fact, we had combined two pMB9 clones, pAU-1 and pAU-2, into a composite subclone and had used pBR322 as a vector, since this work was done after pBR322 had been certified [as safe] by NIH. Thus, the deposit made with the ATCC was this subclone in the pBR322 vector.

This indicates how rapidly Rutter and his colleagues were working in the spring of 1977. In January, they put the insulin gene into a nonpermitted pBR322 vector, and in March they destroyed that material after being told its use was not allowed by NIH. They recloned the gene into a permitted vector (pMB9), publishing a paper on it in Science (17 June 1977, p. 1313). But in connection with a May patent application, they recloned the insulin into the (then permitted) pBR322 vector, which they deposited at the ATCC. Thus, the vector on deposit is not from the experiment described in Science and cannot be used to answer lingering questions about it.—Eliot Marshall

Troubling Matters

In his profile of Herbert Benson, founder of Harvard's Mind/Body Medical Institute (Research News, 18 Apr., p. 357), Wade Roush notes that "there have been critics who call Benson a better showman than a scientist." We see Benson as a publicizer of therapeutic claims that appear not to be supported by the data.

For example, according to Claudia Wallis (1), Benson has said that, by routinely eliciting the relaxation response, "75% of insomniacs begin to sleep normally, 35% of infertile women become pregnant and 34% of chronic-pain sufferers reduce their use of painkilling drugs"; Benson himself essentially repeats these claims in the 1996-1997 brochure advertising his continuing medical education course "Spirituality and Healing in Medicine-II" sponsored by Harvard Medical School. But what do the data show?

Concerning insomnia, the relevant paper (2) explicitly states that it is not possible to say whether the relaxation response contributes to the therapy, because a multifactor approach was used. In a separate study involving small numbers of patients (10 test versus 10 control) and labeled as "preliminary" (3), evidence of a small contribution of the relaxation response is made problematic by large standard errors of the means.

Concerning infertility, the relevant paper (4) disavows Benson's claim. The paper states, "the conception rate may be within the normal range for women who aggressively seek treatment from experienced infertility specialists" (4, p. 147), which is what most of the patients were doing in addition to receiving the behavioral treatment.

Concerning chronic pain, again the relevant paper (5) explicitly states that it is not possible to say whether the relaxation response contributes to the therapy because they used a multifactor approach. And nothing at all is said about any reduction in the use of painkilling drugs.

We are troubled by the discrepancies between the claims and the data.

Irwin Tessman

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Letters to the Editor

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