collegial discussion that occurred at that RAC meeting. Our protocol was not "nixed" by the RAC. In fact, the RAC has encouraged us to proceed with the clinical study after addressing recommendations that evolved as a result of our discussion at that meeting. We intend to move forward after incorporating these recommendations.

Garber's article seems to trivialize the important ethical and scientific questions that were raised in this forum. Pedro Lowenstein presented interesting unpublished findings concerning the administration of viral vectors into rat brain. Thaddeus Dryja had some important views on the therapy of retinoblastoma that went far beyond the statement that enucleation is "gratifyingly tolerable" for the treatment of this disease. These ad hoc reviewers along with members of our clinical research team were

Retinoblastoma, a malignancy of the eye and a target for gene therapy

asked to present their views to the RAC and did so out of a sincere commitment to better the lives of children with cancer.

The article makes it sound as if the meeting was rife with contention and disagreement. Nothing could be farther from the truth. The meeting was handled like any other NIH review committee meeting and strongly adhered to the peer-review process so vital to the scientific community. Members from our team have great respect for the members of the RAC, the ad hoc reviewers, and the review process. In fact, one of the members of our research team, Estuardo Aguilar-Cordova, is currently a member of the RAC, but was not involved in the review process for this protocol.

Finally, a statement is attributed to Jan Wolff implying that some members of the gene therapy community are "cowboys." For those of us who have dedicated our careers to finding treatments for children with cancer that not only cure their disease but also avoid long-term toxicity and debilitation, we can only respond that when we enter our patients' rooms, we leave our hats and boots at the door.

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Seismic Hazard at the New Madrid Seismic Zone

In their report "Slow deformation and lower seismic hazard at the New Madrid seismic zone" (23 Apr., p. 619), Andrew Newman *et al.* analyze a regional network of Global Positioning System (GPS) velocity vectors in terms of a model developed for "infinitely long" strike-slip faults like the San Andreas, in the central United States (*1*). The apertures of the geodetic networks along the San Andreas are small with respect to the length of the fault, and far-field velocities approach the rate of relative plate motion. The exact opposite is the case in the study by Newman *et al.* The segmented fault system in New Madrid seismic zone is smaller than the scale of their regional geodetic network, and because the fault system they are studying is located within a stable continental interior, far-field velocities must approach zero (or extremely small values).

For these reasons, my colleagues and I made a detailed study in 1991 (*2*) of crustal strain with the use of a dense concentration of geodetic stations located astride a single major fault. Our repeated GPS measurements of this network in 1993 and 1997 appear to indicate lower rates of strain accumulation than we originally reported (*2*) on the basis of combined GPS and triangulation measurements. Lower rates of strain, however, do not necessarily imply lower seismic hazard for the region. It is quite possible that the strain energy released in the "storm" of large earthquakes that have been occurring in this area for the past few thousand years took hundreds of thousands, or even millions, of years to accumulate. If this is the case, a slow rate of strain accumulation over the past 6 years does not imply low seismic hazard.

The persistently high rate of seismic activity in the New Madrid Seismic Zone over the past few thousand years implies high seismic hazard in the foreseeable future.

To communicate any other message to the public would seem to be a mistake.

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1 J. Savage and R. Burford, *J. Geophys. Res.*, **78**, 832, (1973).
2. L. Liu, M. Zoback, P. Segall, *Science* **257**, 1666 (1992).

Response

Our report examined two arguments widely cited to support assertions of high seismic hazard in the New Madrid zone, as illustrated by the National Seismic Hazard maps showing a higher hazard there than that shown for California. We found that both arguments seem incorrect.

First, our GPS measurements showed little or no far-field motion across the seismic zone, both near the fault and at distant sites. In contrast, Liu *et al.* (*1*) studied a network within ours, reported rapid strain accumulation comparable to that for the San Andreas fault, and interpreted this as consistent with an earthquake of magnitude 8 on the Richter scale occurring about once every 1000 years. Our observation of little or no resolvable motion, which Zoback and others now also find in their network, is independent of assumptions about fault mechanics. Both we and Liu *et al.* relate the inferred slip to earthquake recurrence through the standard steady-state assumptions criticized by Zoback. Although one might postulate alternatives, including time-dependent effects, the present data seem inadequate to require any explanation beyond that of little present motion.

Second, we revaluated an analysis by Johnston and Nava (*2*), which yielded a 550- to 1100-year recurrence for earthquakes with a magnitude greater than 8.3. We found that these data in fact correspond to a 14,000 +/– 7000 year recurrence for such earthquakes, or a 1,400 +/– 600 recurrence for magnitude 7 earthquakes. It thus appears that the largest New Madrid earthquakes are either smaller or less frequent than previously assumed. In our preferred model, these earthquakes are magnitude 7 (10 times smaller than one of magnitude 8). Similar proposals are being advanced by others based on fault lengths and geologic estimates of fault slip, both of which appear too small for magnitude 8 earthquakes. These observations have implications for seismic hazard estimates in the area. The predicted hazard depends on assumptions, many of which have considerable uncertainty because we have little seismological data from any but small earthquakes. For example, treating a magnitude 7 earthquake as one of magnitude 8 overpredicts the peak ground acceleration by a factor of two or more. Other factors contributing to the high values in the hazard maps include a model predicting higher ground motions than those estimated by alternative models, and parametrization of the largest earthquakes as occuring on widely separated faults, which increases the area of highest predicted hazard.

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Thus, given what we are now learning, to avoid investigating and reassessing the assumption of high seismic hazard at the New Madrid seismic zone would seem a mistake.

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1. L. Liu, M. Zoback, P. Segall, *Science* **257**, 1666 (1992).
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Asking the Right Questions

Don C. Rockey, in a recent letter (*Science*'s Compass, 7 May, p. 915), suggests that "'professional scientists'...constitute a distinct minority of those performing disease-orientated research" and goes on to say that "without a sound understanding of clinical issues (that is, clinical training), how does one ask the appropriate questions?" We suspect that more disease-orientated research is actually conducted by Ph.D. scientists than this statement would imply. However, do such scientists ask the appropriate questions? Clearly so, unless many of the most dramatic pharmacological and behavioral advances made over the last century were only made by clinicians, which is not the case.

Physicians, physician-scientists, and basic scientists all have contributed to biomedical research through questioning and formatting hypotheses and rigorous experimentation, and then re-questioning the original hypotheses.

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CORRECTIONS AND CLARIFICATIONS

In the table of contents of 16 July, on page 293, the URL was incorrect for the technical comments "Functional approaches to gene isolation in mammalian cells" by A. V. Gudkov *et al.* and by A. Kimchi *et al.* The URL should have read, www.sciencemag.org/cgi/content/full/285/5426/299a

In the report "Multilineage potential of adult human mesenchymal stem cells" by M. F. Pittenger *et al.* (2 Apr., p. 143), in the legend for figure 1 (p. 144), reference 12 should have been cited (not reference 11).

In the letter "Large animals in the fast lane" by R. Miller and S. Austad (*Science*'s Compass, 9 July, p. 199), in the first column under item 1, the word "smaller" was omitted. That sentence should have read, in part, "1) Within species, superior longevity is associated with smaller body size...."

In the article "NIH urged to fund centers to merge computing and biology" by David Malakoff (News of the Week, 11 June, p. 1742), Larry Hunter should have been identified as "president of the International Society for Computational Biology (www.iscb.org)."

The crystal structure coordinates for the report "Crystal structure of the human papillomavirus type 18 E2 activation domain" by S. F. Harris and M. R. Botchan (4 June, p. 1673) have been deposited in the Protein Data Bank (accession code 1QQH).