Rabbit Control in New Zealand

In view of the conflicting scientific views relating to rabbit viral hemorrhagic disease (RHD) [or rabbit calicivirus disease (RCD), as it is called in Australia and New Zealand] and the general lack of sound data relating to the virus (it has not been successfully grown in cell culture), the so-called "scientific testing" program conducted by the Australians for the purpose of detecting cross species transmission (D. Drollette, News & Comment, 10 Jan., p. 154) is highly suspect.

Fortunately, the New Zealand Department of Agriculture has instituted a further discussion period and is requiring interested parties to put forward their views. It is important that discussions are based on sound science, with the least possible bias. It is hoped that wise counsel in New Zealand will decide to not introduce the disease, but to watch the Australian experiment with interest for the next 10 years. At the end of that time, perhaps claims can be made with some degree of certainty, on the basis of scientific fact.

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Drolette states that "most scientists do not see any drawbacks to the use of RHD." This ignores the concerns expressed by international experts and the strong opposition expressed by many New Zealand scientists, including the New Zealand Association of Scientists. In his submission to the decision-making process, the president of the latter association, C. H. Sissons, states

> The reason for Association concern is that we believe that the application to import RCD virus preparations and use them as a biocontrol agent promotes premature action . . . where scientific knowledge for necessary safety and effectiveness is lacking and the controversial and compromising process of evaluating it threatens the credibility of all scientific input into crucial issues.

He warns

> The liability which [the New Zealand] Government faces from possible catastrophic consequences of unknown risk level if it authorises an ill-defined, exotic lethal virus in the face of strong opposition and warnings, may be extremely high. . . .

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Curing Rat Glioblastoma: Immunotherapy or Graft Rejection?

It has been reported by J. Trojan *et al.* (1 Jan. 1993, p. 94) (*1*) that antisense insulin-like growth factor I (IGF-I) messenger RNA induces curative immunotherapy of glioblastoma (*1*). We have determined the haplotypes of the glioma cell line and of the recipient rat strain used in this study (*2*). We found that they were not syngeneic, as stated by Trojan *et al.* Instead, all major histocompatibility (MHC) antigens that we tested (MHC I and II) were mismatched. Because MHC expression is up-regulated by antisense IGF-I in C6 cells (*3*), this mismatch may be a key to understanding the unknown mechanism that underlies this unexpected case of tumor rejection.

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References and Notes

1. J. Trojan, T. R. Johnson, S. D. Rudin, J. Ilan, M. L. Tykocinski, J. Ilan, *Science* **259**, 94 (1993).
2. The haplotype of the C6 glioma cell line was found to be different at all tested loci from BDX, the rat strain reported in the original publication, as well as from BDIX, the rat strain that was in fact used by Trojan *et al.* for the reported experiments (A. S. Beutler *et al.*, in preparation).
3. J. Trojan *et al.*, *Neurosci. Lett.* **212**, 9 (1996).

Response: Beutler *et al.* are correct that the actual strain used in our experiments was BDIX and that neither BDX nor BDIX (*1*) rat strains share the MHC haplotype of C6 cells. In light of this, the C6 cell/BDIX rat system must be regarded as allogeneic. These data were unavailable at the time of our experiments and do not fundamentally change the main conclusion of our paper, namely, that antisense suppression of IGF-I in C6 cells increases their immunogenicity.

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References

1. K. Bender *et al.*, *J. Exp. Anim. Sci.* **36**, 151 (1994).

Corrections and Clarifications

The Brookhaven Protein Data Bank accession number 1FT1 was inadvertently omitted from note 45 (p. 1804) of the report "Crystal structure of protein farnesyltransferase at 2.25 angstrom resolution" by H.-W. Park *et al.* (21 Mar., p. 1800).

In the Table of Contents for the issue of 28 February (p. 1234), the title of the letter by N. N. Yang *et al.* should have read "Correction: Raloxifene Response Needs More Than an Element."

In figure 3 on page 379 of the report "Kinetic measurement of the step size of DNA unwinding by *Escherichia coli* UvrD helicase" by J. A. Ali and T. M. Lohman (17 Jan., p. 377), the label of the abscissa was removed. It should have read, "Time (s)".

The response by S. J. Clemett and R. N. Zare (20 Dec., p. 2122) to the technical comments under the title "Evaluating the evidence for past life on Mars" (20 Dec., p. 2119) should not have included (in the last paragraph, p. 2123) reference to unpublished correspondence by Simoneit and Hites and by Requejo and Sassen.

In figure 4 on page 1119 of the article "Diversity and pattern in the developing spinal cord" by Y. Tanabe and T. M. Jessell (15 Nov., p. 1115), the label "MCC_l" in blue (far left) was incorrect due to an editing error. It should have read, "MCC_m".

In note 25 (p. 1173) of the report "Regulation of T cell receptor signaling by tryosine phosphatase SYP association with CTLA-4" by L. E. M. Marengere *et al.* (24 May 1996, p. 1170), thanks should have been given to T. Pawson for contributing key reagents, developed in his laboratory.

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.