sequences, such events could lead to the potentially harmful production of packageable infectious recombinant virus. Since avoidance of any homology with endogenous retroviruses is thus desirable, Anderson suggests using mouse retroviral vectors as a delivery system. However, quite apart from the putative inherent instability of recombinant retroviruses, this proposal is probably insufficient to overcome the recombination problem. This is because sequences with homology to mouse mammary tumor virus (*1*), Moloney murine sarcoma virus (*2*), Abelson murine leukemia virus (*3*), and Moloney murine leukemia virus (*4*) have recently been found in the human genome. Indeed, sequences containing murine retrovirus long terminal repeats (LTR's) have been employed in the screening of human genomic libraries (*5*).

There would appear to be two alternative means of circumventing this problem which would eventually enable murine vectors to be used in human gene therapy. Every such attempt would have to be preceded by a search for vector-homologous sequences in the patient's genome by Southern blotting. If sequences homologous to murine retroviral vectors currently in use are indeed found to be common in human genomes, as suggested by the work of Repaske *et al.* (*4*), alternative vectors derived from more distantly related species would have to be considered. Clearly, considerable attention will have to be directed toward the construction and experimental trial of appropriate retroviral vectors in order to optimize any future gene delivery system for use in humans.

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David Cooper raises a legitimate concern regarding possible recombination between murine leukemia virus (MuLV)–based viral vectors and endogenous retroviral sequences present in the human genome. In fact, recombination between a deletion mutant of Moloney MuLV and homologous sequences in mouse DNA involving a 400-base-pair segment that was 78 percent homologous has recently been demonstrated (*1*). To evaluate possible recombination between MuLV's and human endogenous retroviral sequences, mouse cells have been cotransfected with defined *gag* and *pol* deletion mutants of Moloney MuLV (*2*) and cloned *gag* and *pol* segments of endogenous human retroviral DNA's. In no case could recombination be demonstrated. Although the deduced amino acid sequences comprising the *gag* and *pol* regions of endogenous human retroviral sequences are evolutionarily related to comparable segments of MuLV's (*3*), the extent of polynucleotide sequence identity may be too low for homologous recombination. For example, the *gag* and *pol* regions of human endogenous MuLV sequences are only 35 percent and 44 percent, respectively, related to analogous segments of MuLV. Furthermore, nucleotide sequencing of several different human endogenous retroviral clones (*4*) has indicated the presence of point mutations, inappropriate terminator codons, and deletions of various sizes, any one of which could render recombinants that might be generated replication defective.

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Erratum: The name of M. Wallroth was omitted as the fourth author of the report "A simple and general method for transferring genes into plants" by R. B. Horsch *et al.* (8 Mar., p. 1229).

Erratum: In the legend for figure 2 of the report "*Plasmodium falciparum* malaria: Band 3 as a possible receptor during invasion of human erythrocytes" by V. C. N. Okoye and V. Bennett (11 Jan., p. 169), a reference for the use of metrizamide to purify schizonts was inadvertently omitted after the fifth sentence. It should have read, "Following the method of C. S. Pavia *et al.* [*Am. J. Trop. Med. Hyg.* **32**, 675 (1983)], as modified by Lyons."

Erratum: In figure 1 of the report "How bees remember flower shapes" by J. L. Gould (22 Mar., p. 1492), the results shown for the 24-element patterns (K_1 and K_2) should have been $P > 0.05$.