Be that as it may, Duesberg's rejoinders to all objections reminded me of the old story of the man who complained to a psychiatrist that he thought he was dead. The psychiatrist then asked whether dead men bleed, and on receiving a negative answer, took out a lancet and stuck the patient's finger. The man stared at his bleeding finger for a moment and then said, "Well, I guess dead men do bleed."

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Response: I find Peter Duesberg's letter confusing. The mainstream view of AIDS is that HIV debilitates the immune system, allowing pathogens that are usually quite feeble to cause severe disease and even death. I don't see how the finding that a newly discovered herpesvirus may cause KS challenges the mainstream view. The role of HIV is highlighted by the fact that KS, in the absence of immunosuppression, is almost always a benign disease, but in AIDS patients it is often fatal.

On a separate point, Nicholas Birkett correctly notes that the Concorde data, like any scientific data, can be interpreted in more than one way. But Birkett does not emphasize just how slight the differences were in the Concorde trial between the HIV-infected people who were treated early with AZT and those who were untreated or received deferred treatment. As shown in a table accompanying the story, the *P* values—a measure of statistical significance—were 0.13 for total deaths and 0.34 for HIV-related deaths. Those are far from the *P* value 0.05 that most researchers accept as the minimal cutoff point for statistical significance.

Therefore, while it is true to say—as Duesberg does—that there is a 25% difference between the total deaths in the two groups, how much significance can be imputed to that figure remains an open question. In my article, I reported the view of the chief French Concorde researcher, who argues that the Concorde data do not support Duesberg's contention that AZT causes AIDS. Instead, as the Concorde study team concluded, the data simply indicate that treatment with AZT does not benefit healthy, HIV-infected people.—***Jon Cohen***

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Chromosome Behavior in Mutants Defective in DNA Methylation: Correction

In our report describing the isolation of *Neurospora* mutants defective in DNA methylation, "Abnormal chromosome behavior in *Neurospora* mutants defective in DNA methylation" (10 Dec. 1993, p. 1737) (*1*), we reported evidence for aneuploidy in a mutant (*dim-2*) devoid of detectable methylation. We also reported that mutants with defects in the biosynthetic pathway leading to the methyl group donor *S*-adenosylmethionine can be manipulated to show reduced methylation, and we used one such mutant, *met-7*, to follow up the suggestion that reduction in methylation leads to the abnormal chromosome behavior. A large fraction of progeny from crosses between two *met-7* strains showed evidence of aneuploidy, supporting the notion that normal DNA methylation is required for proper chromosome behavior. Results of recent experiments in our laboratory indicate, however, that our source of the *met-7* mutation, FGSC strain 3915, contains a previously unrecognized recessive mutation, unrelated to *met-7*, that is responsible for the unusual chromosome behavior (*2*). In addition, attempts to quantify the effect of *dim-2* on aneuploidy did not show significant increases in aneuploidy attributable to *dim-2* (*3*). Thus, we wish to retract our interpretation that methylation deficiencies cause abnormal chromosome behavior. Finally, we wish to correct an error in the legend to figure 6: the *met-7* strain number should have read "N561 (FGSC 4143)" instead of "N556 (FGSC 3915)."

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1. H. M. Foss, C. J. Roberts, K. M. Claeys, E. U. Selker, *Science* **262**, 1737 (1993).
2. A. Hagemann, J. Irelan, H. Foss, C. Roberts, E. Selker, unpublished data.
3. A. Hagemann and E. Selker, unpublished data.

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Sequence Correction

We wish to note a correction of the *NPM-ALK* sequence (GenBank accession number U04946) described in our report "Fusion of a kinase gene, *ALK*, to a nucleolar protein gene, *NPM*, in non-Hodgkin's lymphoma" (4 March 1994, p. 1281) (*1*). After re-examination of our sequencing data, we identified an erroneous omission of two nucleotides in the codon of NPM-ALK amino acid residue 495 that was made because of a reading error. The frame-shift produced by this error led us to assign a premature termi-

nation codon, resulting in a predicted NPM-ALK protein of 525 amino acids. The correct *NPM-ALK* nucleotide sequence, now entered in GenBank, encodes a protein of 679 amino acids with a predicted molecular weight of 75.3 kilodaltons. A polypeptide with this relative mobility, generated by in vitro transcription/translation of the full-length *NPM-ALK* complementary DNA, co-migrates with the NPM-ALK protein immunoprecipitated from the t(2;5)-containing lymphoma cell lines SU-DHL-1, SUP-M2, and UCONN-L2.

To our knowledge, all other data reported in our paper are correct. We regret any inconvenience or confusion that this sequencing error may have caused our scientific colleagues.

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1. S. W. Morris *et al.*, *Science* **263**, 1281 (1994).

Corrections and Clarifications

In the article "Molecular basis of mammalian sexual determination: Activation of Müllerian inhibiting substance gene expression by SRY" by C. M. Haqq *et al.* (2 Dec., p. 1494), parts B and C of figure 1 were inadvertently interchanged. In figure 3A, the third, fourth, and fifth lanes should have been labeled "$A_7 \rightarrow T$," "$A_7 \rightarrow C$," and "$A_7 \rightarrow G$," respectively. In figure 5, the factor labeled "SRIF's" should have been labeled "SRYIF's."