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CORRESPONDENCE

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Spin science

To the editor:

Editorials are most valuable when they are grounded in fact and are well reasoned. Unfortunately, your editorial accusing Geron of using "spin science" and damaging the credibility of the biotechnology industry with its November 6, 1998 announcement of the derivation of human embryonic stem cells ignores simple facts and relies on hyperbole in attempting to make a point.

The facts are that the work announced by Geron, like all the work announced by Geron, was published in a highly respected peerreview journal (*Science*). Three independent editorials highlighting the significance and implications of the discovery accompanied the paper in *Science*. Finally the scientific and medical communities have independently heralded the potential of the work. In fact, on December 2, 1998, in a prepared televised statement to the Senate Appropriations Subcommittee on Labor, Health and Human Services, Education and Related Agencies, Dr. Harold Varmus, Director of the National Institutes of Health, said the following:

The development of cell lines that may produce almost every tissue in the human body is an unprecedented scientific breakthrough. It is not too unrealistic to say that this research has the potential to revolutionize the practice of medicine and improve the quality and length of life.

It is *Nature Biotechnology*, not Geron, who does a disservice to our industry by failing to recognize and support important, peer-reviewed discoveries when they are made. Fortunately, in this instance, your journal was among the distinct minority in this failing.

> Ronald W. Eastman Geron Corporation Menlo Park, CA 94025

Nature Biotechnology responds:

Our editorial had no quarrel with peerreviewed ES cell research, which incidentally we concur has a "not too unrealistic" therapeutic potential. What we did object to was the way in which ES cell research was hyped in the media by the companies involved to achieve political and financial agendas.

Shorter is better

To the editor:

As the final structure–function of proteins depends on the folding environment, protein primary structure does not necessarily guarantee a unique tertiary structure, functionality, or even solubility. The paper by Matsuura et al.¹ demonstrated that protein thermal stability could be altered by engineered random peptides (tags) at the C terminus. It provides a great potential for protein improvements and stabilization. One of the limitations of this approach is that the active peptide is covalently linked to the target protein limiting the sites in the protein that it can interact with.

A year ago we demonstrated that "unattached peptides" could modulate the function of a model protein, tryptophan repressor, in vivo and in vitro. One tripeptide, for example, mimicked the inducer and resulted in loss of repression (Fenton et al. 1998)². One obvious application of our approach is "protein therapy," in which the coding sequence of target protein would not be altered, but its activity could be modulated by a short peptide. Early genetic research demonstrated that mutants within the same cistron could complement each other. This restoration of function of dead proteins, as for example in mutant/mutant interaction (Storbakk et al 1996)³, also questions the uniqueness of tertiary/quaternary structure for functionality of the protein. Together with the chaperones and foldases, the current findings open doors to unlimited applications in biotechnology. If therapeutic proteins are reduced to short peptides⁴ and proteins can be modulated by short transportable peptides, perhaps for therapeutics shorter is better.

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Erratum

October 1997's Business and Regulatory News Brief "Microarrays map matings" (*Nat. Biotechnol.* **16**, 893, 1998) incorrectly attributed a report in *Science* (**281**, 1194–1197, 1998) concerning rapid mapping of yeast genes to researchers from the University of California, Berkeley. The majority of researchers are from Stanford University, with other contributors from Duke University and Affymetrix, Inc.

Matsuura, T. et al. Nat. Biotechnol. 17, 17–61 (1999).
Fenton, C. et al. Biochem. Biophys. Res. Commun.

 ^{242, 71–78 (1998).}Storbakk, N. et al. J. Mol. Biol. 256, 889–896

^{(1996).} 4. al-Obeidi F. et al. Mol. Biotechnol. 9, 205-223

^{(1998).}