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Cell Cycle Arrest

We were pleased to see the three reports (1-3) and Research News article by Jean Marx (p. 963) in the 17 February issue that highlight the induction of the protein p21(WAF1) cyclin-dependent kinase (Cdk) inhibitor in myogenesis (1) and the high level of expression of p21(WAF1) in terminally differentiated tissues (2). These findings elegantly extend the findings published last fall in our papers "Induction of p21(WAF1/CIP1) during differentiation" (4) and "Induction of differentiation in human promyelocytic HL-60 leukemia cells activates p21, WAF1/CIP1, expression in the absence of p53" (5). We had reported that multiple differentiation inducers caused immediate-early and sustained up-regulation of p21 in many cell types through a p53-independent pathway. The report by Skapek *et al.* (3) demonstrating

p21(WAF1) reversal of a cyclin D1-mediated differentiation block in muscle raises the hope that in some settings p21(WAF1)-inducing agents may be anti-oncogenic. We would caution, however, that this strategy would be ineffective in settings in which p21(WAF1) induction is uncoupled from growth arrest. An example is our demonstration that deregulated c-myc expression is capable of uncoupling p21(WAF1) induction both from growth arrest and from differentiation (4).

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4. R. A. Steinman *et al.*, *Oncogene* **9**, 3389 (1994).
5. H. Jiang *et al.*, *ibid.*, p. 3397.

"More" Is Not "Different"

I agree with Sheldon Krinsky (Letters, 17 Feb., p. 945) that "[m]odifying an inert chemical structure and modifying an organism are two very different things." Yet, he illogically extends this observation to a comparison of two organisms. Modification of an organism by traditional breeding and by recombinant DNA methods are not very different things. The fact that we can make a greater variety of changes by recombinant DNA is not an inherent reason to place a higher regulatory burden on products of recombinant DNA techniques.

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Sampling Zooplankton: Correction

We have learned that there is an internal inconsistency in the zooplankton dataset used in our report "Climatic warming and the decline of zooplankton in the California Current" (3 Mar., p. 1324) (1). The data

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file we used, written from the archive maintained by the National Marine Fisheries Service, was found to change at the end of 1977, without notation, from values of zooplankton normalized by sample volume to raw values of zooplankton volume. Because

the series was treated uniformly, pre-1978 data were incorrectly normalized a second time, while post-1978 data were handled correctly. This boosted pre-1978 values by up to a factor of 2. Correction of this error does not change the conclusions of our

report. There has been a large decrease in zooplankton biomass during the past 43 years, which is likely related to the concurrent warming of the upper 100 meters. However, the correction does reduce the magnitude of the observed downward trend in zooplankton.

Cruise-by-cruise averages of log-transformed data show the decline of zooplankton volume (Fig. 1A; see figure 2A in our report), which is especially prominent from 1978 to the present. Average values over all cruises in a year were transformed back to natural units by taking the inverse logarithm (Fig. 1B). The average zooplankton volume over the final 7 years of the survey (1987–1993) was 70% lower than the average over 1951–1957. The reduction was approximately uniform with respect to distance from shore, possibly intensifying slightly offshore (Fig. 1C; see figure 3A in our report).

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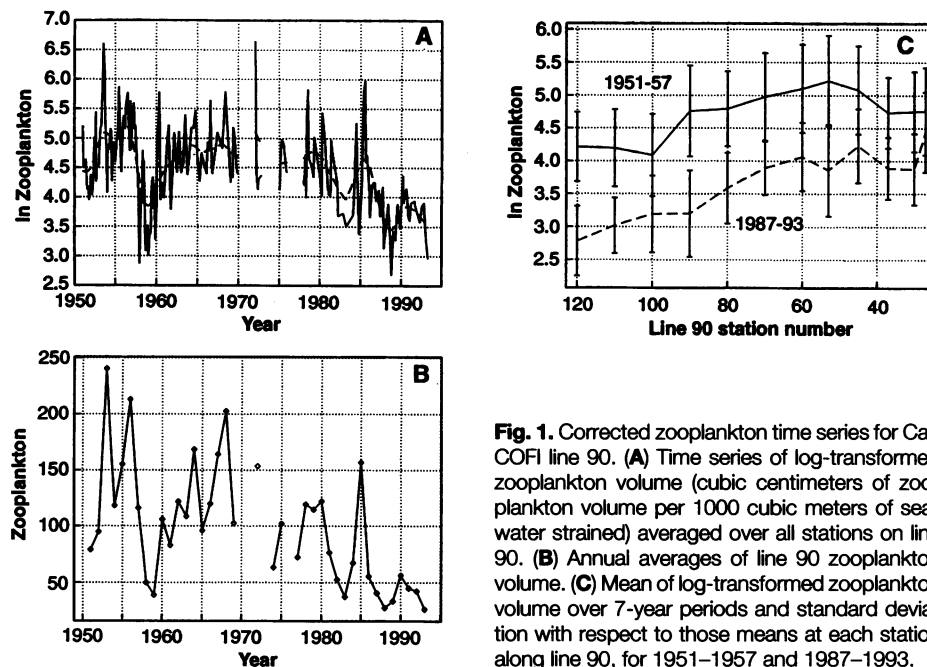


Fig. 1. Corrected zooplankton time series for CalCOFI line 90. (A) Time series of log-transformed zooplankton volume (cubic centimeters of zooplankton volume per 1000 cubic meters of seawater strained) averaged over all stations on line 90. (B) Annual averages of line 90 zooplankton volume. (C) Mean of log-transformed zooplankton volume over 7-year periods and standard deviation with respect to those means at each station along line 90, for 1951–1957 and 1987–1993.

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