Letters

Alaskan Oil Spill

Philip H. Abelson's editorial of 12 May (p. 629) presents an excellent summary of the Exxon Valdez oil spill, but contains an error in nomenclature. The current that flows through Prince William Sound is the Alaska Coastal Current (*1, 2*), not the Alaska Current. The Alaska Current is confined to the deep waters of the Gulf of Alaska about 150 kilometers offshore and is analogous to the Gulf Stream, although its flow is only about 10 million cubic meters per second (*3*), about 1/10 that of the Gulf Stream. The Alaska Coastal Current is found within 40 kilometers of the coast and has an average flow of about 200,000 cubic meters per second. As a comparison, the volume of the spill was about 40,000 cubic meters. Over the past several months, the Alaska Coastal Current has been flowing at about half its average rate. This is a result of the normal seasonal fluctuation in its driving forces of freshwater and wind stress. The mean annual rate of freshwater entering this coastal system (23,000 cubic meters per second) (*4*) from precipitation, runoff, and glacial melt is greater than the mean annual discharge of the Mississippi River (18,000 cubic meters per second), making this the largest freshwater system in North America. The flow in the Alaska Coastal Current peaks in the fall with currents greater than 150 centimeters per second (*5*), and this should enhance the flushing of the sound at that time. The coastal freshwater discharge is partially responsible for keeping the oil off the shore. Unfortunately, this same coastal flow extends for several thousand kilometers along the coast of Alaska into the Bering Sea and Arctic Ocean.

Another serious problem concerning the oil spill has arisen. Some factions of the government (both federal and state) and private industry are requesting that the data on the spill be made proprietary. The apparent reason for this is to keep the "other side" from having the advantage in upcoming lawsuits. This is in direct conflict with the accepted policies within the oceanographic community, where data are exchanged readily. I believe the best solution to this problem is the free exchange of all data and timely publication of the results in the open, reviewed literature.

THOMAS C. ROYER
Institute of Marine Science, University of Alaska, Fairbanks, AK 99775

REFERENCES

1. R. K. Reed and J. D. Schumacher, in *The Gulf of Alaska, Physical Environment and Biological Resources*, D. W. Hood and S. T. Zimmerman, Eds. (MMS 86-0095, National Oceanic and Atmospheric Administration, Washington, DC, 1987), pp. 55–57.
2. T. C. Royer, *J. Mar. Res.* **39**, 251 (1981).
3. ———, *ibid.*, p. 239.
4. ———, in *Coastal Oceanography*, H. G. Gade, A. Edwards, H. Svendsen, Eds. (Plenum, London, 1983), pp. 9–30.
5. W. R. Johnson, T. C. Royer, J. L. Luick, *J. Geophys. Res.* **93** (C10), 12423 (1988).

Further Liaisons with Sperm

Our writing of the minireview "Dangerous liaisons: Spermatozoa as natural vectors for foreign DNA?" [*Cell* **57**, 701 (1989)], which reviewed the article by M. Lavitrano *et al.* in the same issue of *Cell* (p. 717), has been commented on recently in a derogatory manner in an article by David Dickson (Research News, 30 June, p. 1539). The *Science* article contains the imputation that we wrote the minireview to promote personal financial gain and the commercial interests of Genentech and Boehringer Ingelheim [who jointly established our institute, the IMP (Institute of Molecular Pathology)]. In particular, the allegation was made that the IMP has applied for patent rights on the initial work as well as on extensions of Corrado Spadafora's work and that this led to a conflict of interest when we wrote a commentary on the paper. Both statements are false. In fact, the only patent applied for is that of the authors of the research article (and is mentioned in the paper). We can state categorically that neither at the time of writing the review nor since have we, the IMP, Genentech, or Boehringer Ingelheim, had any commercial stake in such patents or licenses in the field of Spadafora's work.

We were invited to write the minireview after the research article had been accepted for publication in *Cell*. We accepted the invitation in order to draw attention to the work of a young and unknown research team (pejoratively referred to as "obscure" in Dickson's article), who had come across an apparently astounding new finding, a simple method to make transgenic animals. One lesson that we felt could be learned from these events is that it does not always take large established research groups to produce interesting science. (The review was published in the minireview section of *Cell*—not in the form of an editorial, as described by the article in *Science*—where it is common for reviews to comment on research published in the same issue of the journal.) Neither we nor Spadafora sought publicity for the results in the form of press releases.

Our involvement with the story started last summer when Spadafora asked us to criticize his initial results. We were (and still are today) very impressed with this work, but we suggested additional controls. Our involvement was not secret and is acknowledged in the original paper as "helpful discussions, suggestions, and critical reading of the manuscript." After the submission of the article, further work was carried out at the IMP by none other than Spadafora, who was able to reproduce the DNA binding to sperm without any difficulties. (On a technical point, Spadafora now suspects that the presence of phenol red commonly found in cell culture media may be inhibitory to DNA binding.)

The imputation of Dickson that our review added legitimacy to the scientific work will be believed only by the most gullible. Informed scientists know that this is nonsense because only further experimentation can legitimize the findings of Spadafora's group. Indeed, our minireview opens by saying that readers will treat the research article with a "healthy dose of skepticism." Before writing the review, we scanned the literature for reports on DNA transfer by sperm, and Spadafora inquired of many specialists in the field about possible previous experiments of this type. None of us discovered such reports, but the appearance of the article elicited a single response citing older work, namely that of Brackett *et al.* [*Proc. Nat. Acad. Sci. U.S.A.* **68**, 353 (1971)], which reported the uptake of SV40 DNA by sperm and its transfer to rabbit egg cells.

Brackett *et al.* could show by autoradiographic means that 30 to 35% of rabbit spermatozoa are capable of incorporating labeled SV40 DNA (as opposed to the entire SV40 virion) into the postacrosomal region. The association of the SV40 DNA with the sperm head was further corroborated by fusing spermatozoa exposed to SV40 DNA with cells of the African green monkey line CV-1, which resulted in the production of infectious SV40 virus. The authors presented the first, although indirect, evidence of sperm-mediated transfer of DNA into egg cells: when rabbit ova were fertilized with sperm that had been treated with SV40 DNA and were then analyzed (after mechanical disruption) on a CV-1 cell monolayer, up to 40% induced a cytopathic effect in the cells as a consequence of SV40 virus production in the rabbit zygote.

We, and apparently also the reviewers of Spadafora's paper, were unaware of this earlier report, which is not commonly quoted in the specialist literature. We should like to apologize to the authors and the readership of *Cell* for this oversight. The

issue of patents on Spadafora's work may well prove to be a storm in a teacup, since the basic idea was recorded nearly 20 years ago but remained unnoticed by cell biologists and animal breeders, possibly because no minireview accompanied it. The belated rediscovery of the work of Brackett *et al.* vindicates the findings of the Spadafora group. We are happy to learn that recently at least one other group has been able to repeat Spadafora's experiments.

MAX L. BIRNSTIEL
MEINRAD BUSSLINGER
Research Institute of Molecular Pathology, Dr. Bohr-Gasse 7, 1030 Vienna, Austria

Structure of *ras* Protein

During the structure refinement at 2.2 Å resolution of the catalytic domain of human c-H-*ras* protein, an alternative tracing (Fig. 1, top left) of the backbone structure was recognized (*1*). The new tracing involves the switching of two β strands, β1 and β3, of the original tracing (Fig. 1, bottom left), which was derived from a 2.7 Å resolution multiple isomorphous replacement (MIR) map (*2*). Residues affected by the switch are 1 to 11 and 46 to 65. All the other residues remain in the same place as in the original model. Most of the biochemical and biological interpretations and conclusions we described previously are not affected by the switch except for those relating to residue 61, which is located in weak electron density. The new model better defines the conformation of residues 10 and 11 (as reflected by lower temperature factors) at the beginning of the phosphate binding loop L1 and brings the conserved Asp^{57} close to the magnesium ion. The overall topology is now more similar to that of the G-binding domain of bacterial elongation factor EFTu (*3*), and the conformation of the phosphate binding loop L1, in particular, is very similar to those of EFTu and adenylate kinase (*4*). The evidence for the correctness of the new tracing is listed below:

1) The residual (*R* factor) of refinement by X-PLOR (*5*) and TNT (*6*) programs for the structure excluding both L4 (which is located in a region of very weak electron density) and bound water molecules was 0.234 for the new structure and 0.268 for the earlier structure. The new model for the complete structure refines to a current *R* factor of 0.19.

2) We have constructed a mutant at residue 59, which could be used to distinguish the two structural models. The difference electron density map between the Thr^{59} mutant and the normal protein (Ala^{59}) shows a positive peak at the position expected in the new structure, but not in the old one.

3) The new structure also fits the original MIR map with the exception of residues 61 to 65, which are located in very weak electron density. Furthermore, the heavy atoms (*2*) are located in the same positions relative to the liganding residues in both structures: Cys^{80} and Met^{111} near heavy-atom site A, Cys^{118} near site B, and Met^{67} and Met^{72} near site C.

4) The difference electron density map between another mutant (Val^{12}) and normal protein (Gly^{12}) shows a strong peak corresponding to the side chain of Val at the location expected in the new as well as the old structures, consistent with both structures having the same L1 location and conformation beginning at residue 12.

Two views of the new backbone structure are shown in Fig. 1, top right and bottom right. A short α helix in L4 revealed during the refinement is also indicated in the figures. The α-carbon coordinates of the new structure have been deposited at the Brookhaven Data Bank. New figures and photographs are available on request.

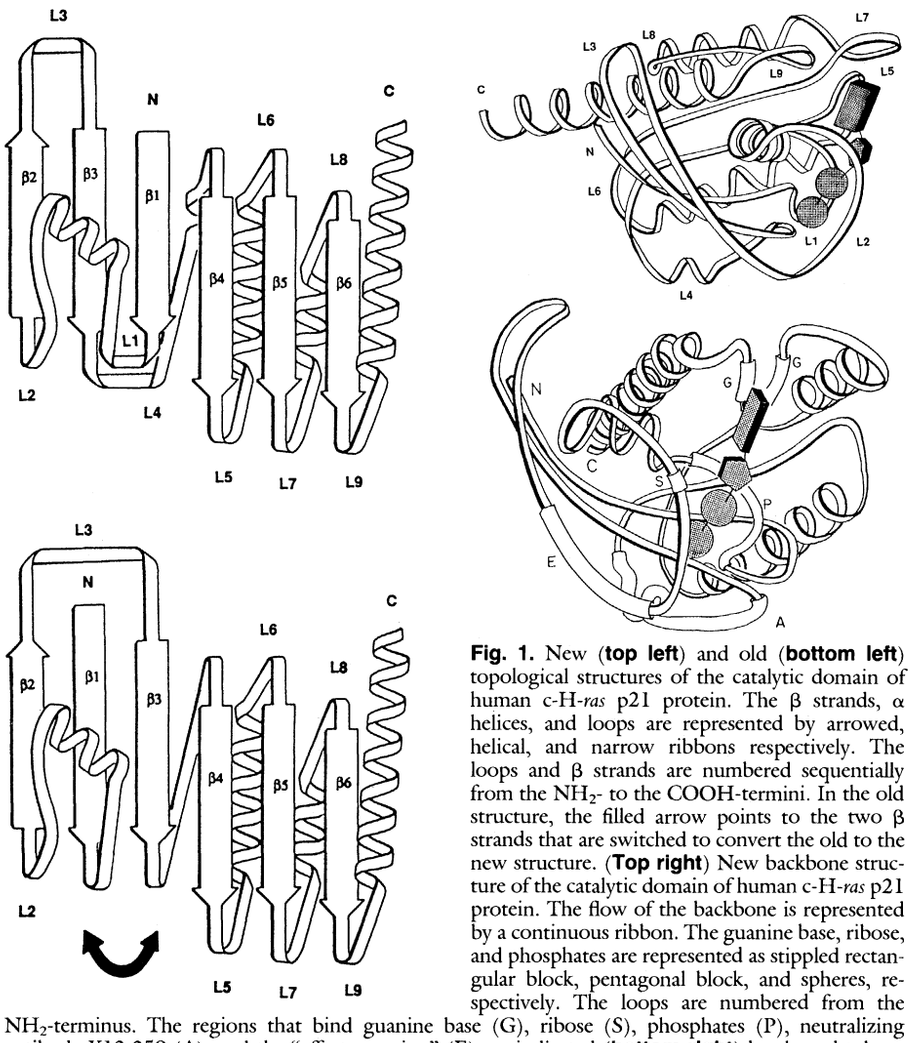

Fig. 1. New (**top left**) and old (**bottom left**) topological structures of the catalytic domain of human c-H-*ras* p21 protein. The β strands, α helices, and loops are represented by arrowed, helical, and narrow ribbons respectively. The loops and β strands are numbered sequentially from the NH_2- to the COOH-termini. In the old structure, the filled arrow points to the two β strands that are switched to convert the old to the new structure. (**Top right**) New backbone structure of the catalytic domain of human c-H-*ras* p21 protein. The flow of the backbone is represented by a continuous ribbon. The guanine base, ribose, and phosphates are represented as stippled rectangular block, pentagonal block, and spheres, respectively. The loops are numbered from the NH_2-terminus. The regions that bind guanine base (G), ribose (S), phosphates (P), neutralizing antibody Y13-259 (A), and the "effector region" (E) are indicated (**bottom right**) by sleeved tubes.

LIANG TONG
MICHAEL V. MILBURN
ABRAHAM M. DE VOS
SUNG-HOU KIM
Department of Chemistry, University of California, Berkeley, CA 94720

REFERENCES AND NOTES

1. We thank E. Pai and A. Wittinghofer of the Max-Planck Institute–Heidelberg and F. Jurnak of the University of California, Riverside, for alerting us to the possibility of this alternative tracing.
2. A. M. de Vos *et al.*, *Science* **239**, 888 (1988).
3. F. Jurnak, *ibid.* **230**, 32 (1985); T. F. M. la Cour, J. Nyborg, S. Thirup, B. F. C. Clark, *EMBO J.* **4**, 2385 (1985).
4. D. Dreusicke, P. A. Karplus, G. E. Schulz, *J. Mol. Biol.* **199**, 359 (1988).
5. A. T. Brünger *et al.*, *Science* **235**, 458 (1987).
6. D. E. Tronrud, L. F. Ten Eyck, B. W. Matthews, *Acta Crystallogr.* **A43**, 486 (1987).

Erratum: In the Table of Contents of the 24 March 1989 issue, the title of the report "Histamine is an intracellular messenger mediating platelet aggregation" by S. P. Saxena *et al.* appearing on page 1596 was incorrectly printed.