Letters

Genome Analysis

I was quite surprised to find myself quoted in Leslie Roberts' 12 May article "New chip may speed genome analysis" (Research News, p. 655) since my contact with Applied Biosystems has consisted of one phone call and one salesperson visit in the last 4 years. The quoted numbers are apparently from a recent article of mine (*1*). Complete details are available (*2*).

Any mention of run times is highly system- and algorithm-dependent. The algorithm I used was similar to that of Wilbur and Lipman (*3*), and the program was a modified version of the Kanehisa IDEAS package (*4*) SEQF. The run time of a complete HIV genome against GenBank was indeed 24 hours, but it was on a Cray-1S, not a Cray-2. Performance improvements in the code, conducted here (*2*) and at the Pittsburgh Supercomputer Center, have cut the Cray run time to an expected 11 hours.

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Attending to Inattention

It is heartening to read Daniel E. Koshland, Jr.'s statement (Editorial, 28 Apr., p. 405) that "Research and procedures directed at counteracting the inevitable lapses of attention that occur in jobs with long periods of high boredom are indicated" to help prevent high-consequence accidents such as that involving the *Exxon Valdez*. Similar concerns for reducing human error due to inattention in accidents involving transportation of hazardous materials were voiced by Philip H. Abelson (Editorial, 10 Oct. 1986, p. 125). Estimates place human error as the source of at least 60% of all transportation accidents, and recent reviews by the Office of Technology Assessment of transportation safety in commercial aviation and trucking have concluded that major safety improvements can come from human factors solutions, including issues associated with hours of service and fatigue (*1*).

The problem of operator inattention due to poorly designed work and rest scheduling is becoming more acute in transportation for a number of reasons. Automation has reduced crew size and has limited operator activity to vigilant monitoring of the system—the *Exxon Valdez* was on automatic pilot during critical minutes leading to its grounding—yet vigilance is most degraded by sleep loss and fatigue. Increases in the size and speed of ships, airplanes, trucks, and trains transporting people and environmentally hazardous materials has made the consequences of a lapse of attention evermore serious. The relentless push to operate transportation and other systems around the clock has meant catastrophe at times when people are least prepared to cope with it—the *Exxon Valdez* hit Blight Reef at 12:04 a.m.; the accident at Three Mile Island (TMI) began at 4:00 a.m.; the explosion at Chernobyl occurred at 1:23 a.m. The safety problem is further exacerbated by regulations that permit prolonged and dangerous work schedules for personal reasons (for example, to earn extended free time) and by competitive forces that pit safety against economic gain or expediency.

Scientists studying human sleep and chronobiology agree that research has much to offer problems of inattention in transportation safety (*2*). The National Transportation Safety Board (NTSB), whose job it is to investigate and determine cause in catastrophic accidents, has seen the cost of driver fatigue and inattention first hand. In a recent Safety Recommendation to the Department of Transportation (DOT), NTSB chairman T. Kolstad noted that (*3*)

> it is time for an aggressive Federal program to address the problems of fatigue and sleep issues in transportation safety. Such a program should include a coordinated research effort, and extensive educational effort directed toward all segments of the transportation industry, and a systematic review and improvement of regulations governing hours of service across all transportation modes.

In order for this to happen, federal agencies that are charged with responsibility for transportation systems must actively promote research on human sleep and inattention. Although DOT has recently summarized its increased activity in this area (*4*), no coordinated program involving the relevant scientific community exists (*2, 3*). Yet techniques in the field of sleep research can now permit answers to important policy questions, which include evaluating economically viable work-rest schedules that are most conducive to safe operations; resolving whether the effects of drugs and alcohol on alertness are potentiated by fatigue; and identifying the most cost-effective ways to enhance operator alertness.

Research can ultimately improve our ability to avoid high-consequence errors of inattention, thereby saving untold numbers of human lives and precious environmental resources. Fatigue-related inattention is first and foremost an issue of safety predicated on farsighted economics and a great public trust. The loss of public trust is the ultimate price paid for high-consequence disasters that involve no human mortality, such as the *Exxon Valdez* and TMI accidents. Surely an ounce of prevention aimed at promoting research on human alertness and sleep-related inattention is worth the billions of dollars now being aimed at rekindling that lost trust.

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Correction: RuGli Cell Line Not of Human Origin

In the report "The human laminin receptor is a member of the integrin family of cell adhesion receptors" [K. R. Gehlsen, L. Dillner, E. Engvall, E. Ruoslahti, *Science* **241**, 1228 (1988)], some of us reported the isolation of an integrin-type receptor for human and mouse laminins from RuGli glioblastoma cells. These cells, which had

been thought to be of human origin (*1*), have now been shown to be rat cells. Having noticed that the immunological reactivities of various proteins did not agree with the assumption that they would be human cells, we submitted the cells in our possession in La Jolla to an isoenzyme analysis. This analysis, performed by the American Type Culture Collection, showed that the isoenzyme pattern of all seven enzymes tested agreed with rat isoenzyme patterns. No contribution from any other species was evident. The RuGli cells in our possession are, therefore, of rat origin. Analyses performed on the original RuGli cells in Germany (*2*) show that these are also rat cells. The species of the RuGli cells does not change the main conclusion of our *Science* paper, that an integrin-type laminin receptor was isolated. Moreover, the receptor from human placental tissue we reported on in our paper is, of course, of human origin. The RuGli laminin receptor, as well as the analogous receptor from human cells, has now been shown to be an $\alpha_3\beta_1$ integrin (*3*).

The RuGli cells have been used by our laboratories in other studies (*4*). Although the species of origin of the cells was not critical, the results obtained with these cells should now be considered as applying to rat cells.

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Erratum: The Research News article by Roger Lewin "Genome planners fear avalanche of red tape" (30 June, p. 1543) incorrectly identified Maynard Olson as being involved in a gene mapping and sequencing project of the nematode *Caenorhabditis elegans* jointly with researchers at the Medical Research Council's Laboratory of Molecular Biology in Cambridge, England. In fact, Olson's Washington University, St. Louis, colleague Robert Waterston is organizing the U.S. end of the project.

Erratum: In the report "Activation of salivary secretion: Coupling of cell volume and $[Ca^{2+}]_i$ in single cells" by J. K. Foskett and J. E. Melvin (30 June, p. 1582), figures 1 and 2 were inadvertently interchanged. The correct figures are printed below.

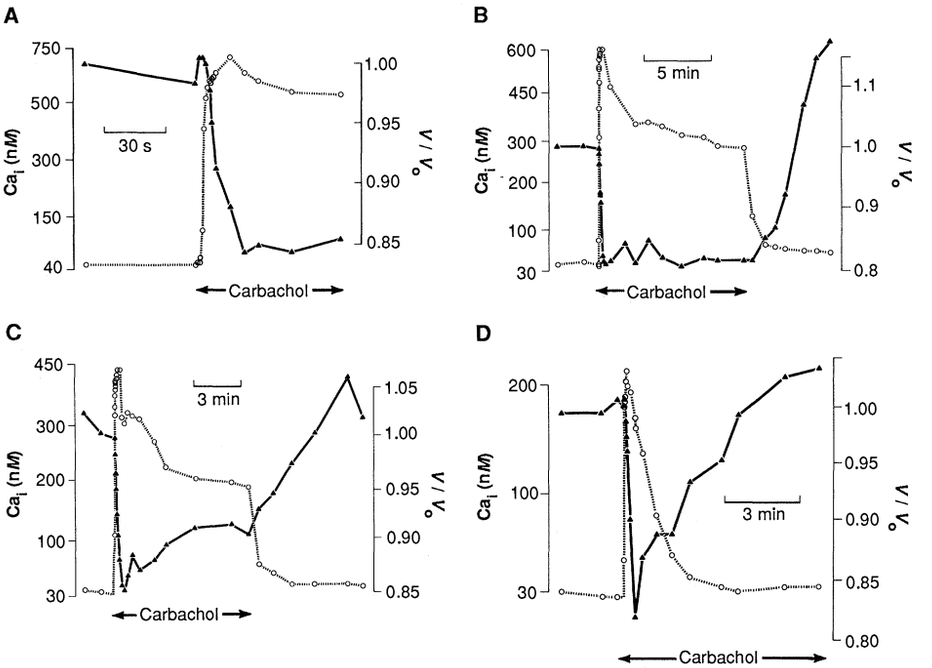

Fig. 1. Time courses of $[Ca^{2+}]_i$ (○) and cell volume (▲). In each experiment, a single acinar cell was stimulated with 10 μ*M* carbachol. Cell volume (*V*) is shown as a fraction of original volume (V_o). (**A**) High-temporal resolution to demonstrate the rates and relations of $[Ca^{2+}]_i$ and cell volume changes. (**B** to **D**) Variability among cells in the responses of $[Ca^{2+}]_i$ and volume to prolonged exposure to carbachol. . . .

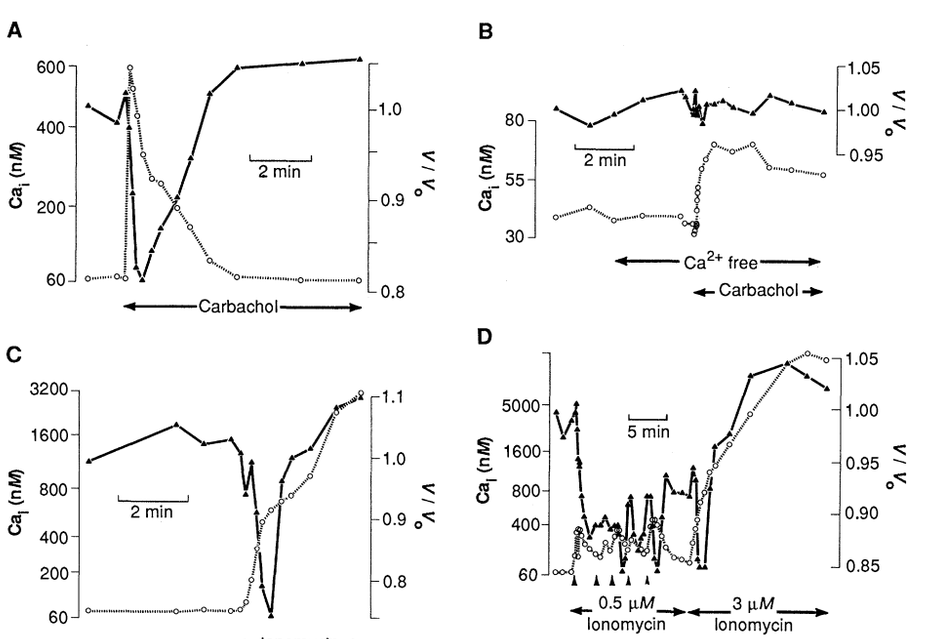

Fig. 2. Time course of single cell $[Ca^{2+}]_i$ (○) and volume (▲) during stimulation with 10 μ*M* carbachol (A and B) or ionomycin (C and D). (**A**) Cell stimulated in the absence of extracellular Ca^{2+}; (**B**) BAPTA-loaded cell; (**C**) 3 μ*M* ionomycin; (**D**) 0.5 μ*M* ionomycin; at arrowheads, 3μ*M* ionomycin was perfused through the chamber for 5 to 15 s. Resultant fluctuations of $[Ca^{2+}]_i$ within the physiological range cause similar fluctuations of cell volume. EGTA (1 m*M*) replaced the Ca^{2+} in Ca^{2+}-free medium. BAPTA loading of cells was accomplished by first loading the cells with fura-2, as described, then incubating the cells for an additional 60 min in 15 μ*M* of the permeant acetoxymethyl ester of dimethyl-BAPTA (K_d = 40 n*M*) (*21*) (Molecular Probes) under identical conditions (*12*).