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.

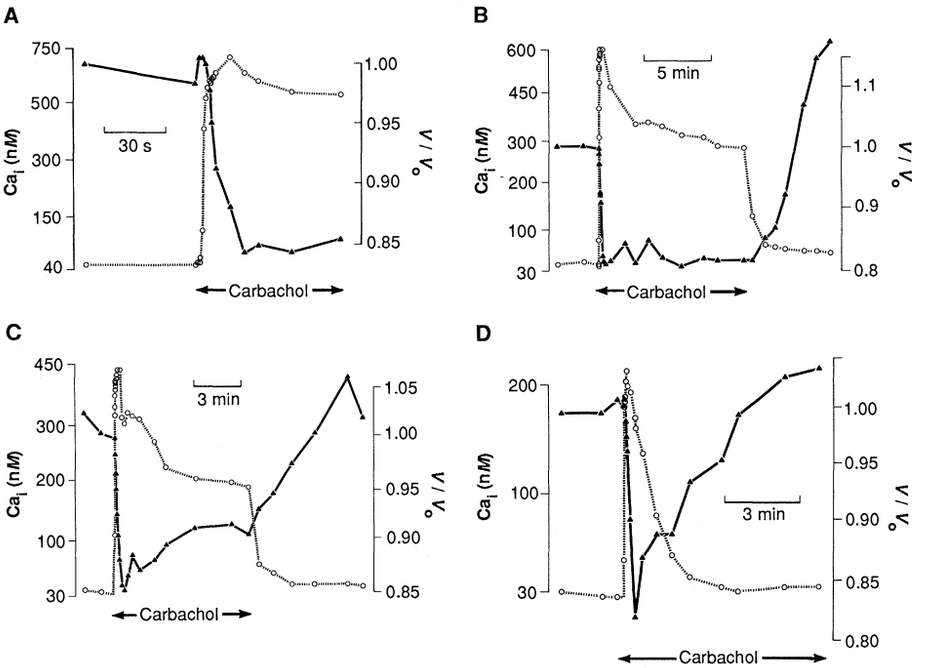

Fig. 1. Time courses of $[Ca^{2+}]_i$ (○) and cell volume (▲). In each experiment, a single acinar cell was stimulated with 10 μ*M* carbachol. Cell volume (*V*) is shown as a fraction of original volume (V_o). (**A**) High-temporal resolution to demonstrate the rates and relations of $[Ca^{2+}]_i$ and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

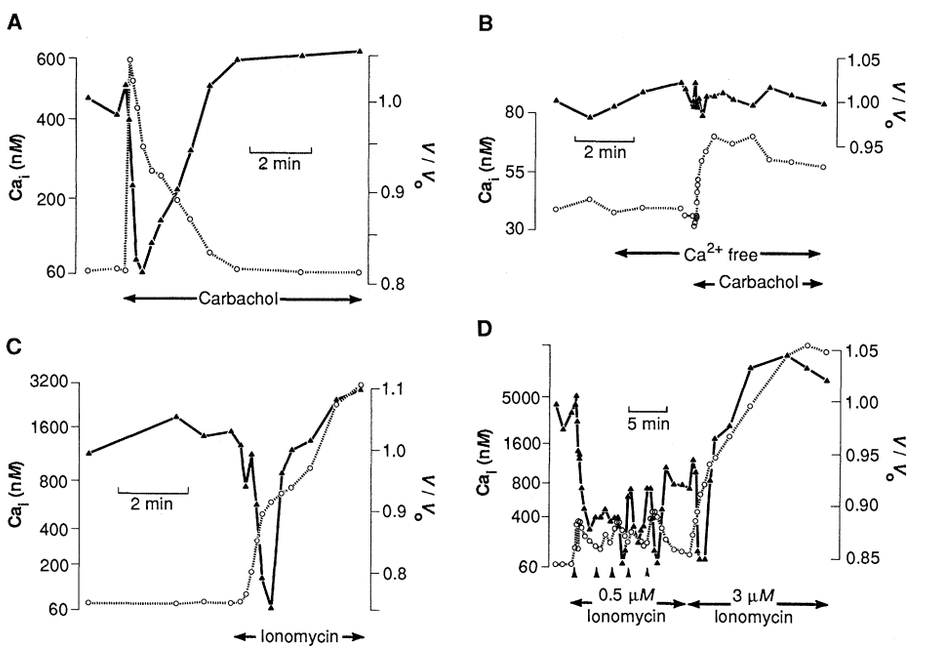

Fig. 2. Time course of single cell $[Ca^{2+}]_i$ (○) and volume (▲) during stimulation with 10 μ*M* carbachol (A and B) or ionomycin (C and D). (**A**) Cell stimulated in the absence of extracellular Ca^{2+}; (**B**) BAPTA-loaded cell; (**C**) 3 μ*M* ionomycin; (**D**) 0.5 μ*M* ionomycin; at arrowheads, 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*).