In summary, the several mouse strains can be arranged in the following order of decreasing sensitivity to somatotrophin: *C3H* ≃ *DBA* > *C3Hf* > *BALB/c* ≃ *C57BL* ≫ *A*. These results follow the general pattern of responsiveness observed *in vivo*: *C3H* ≃ *DBA* > *C3Hf* ≃ *BALB/c* ≃ *C57BL* ≫ *A* (taken from Nandi and Bern[1], Table 3), and in both situations, mammary tissues from strain *A* mice may be considered refractory to somatotrophin.

It may be noted that although the strain differences in hormonal sensitivity *in vitro* in general compare well with observations *in vivo*, the patterns are not absolutely identical. For example, strains *C3Hf*, *BALB/c*, and *C57BL* mice respond to the same degree to somatotrophin *in vivo*; however, in organ culture *C3Hf* tissues were more responsive to somatotrophin than were tissues from *BALB/c* and *C57BL* mice.

The possibility that contamination with prolactin can account for the lactogenic activity of somatotrophin has already been discussed[4], and the question remains open until absolutely purified somatotrophin preparations are available. However, several other preparations of bovine somatotrophin have since been examined in organ culture and found capable of initiating mammary secretion in *C3H* mice but not in *A* mice[6]. Pigeon-crop and immunological assays of these preparations indicate less than 1 per cent prolactin contamination. The present data, therefore, continue to support the view that bovine somatotrophin possesses lactogenic activity independently of its contamination with prolactin but that this inherent activity is apparently limited to certain strains of mice. Barnawell[7] has found no stimulation of secretion ascribable to bovine somatotrophin itself in cultured mammary tissues from two rat strains, hamster, guinea-pig, rabbit and dog.

Further investigations are now in progress to determine whether somatotrophins from various non-bovine species are also lactogenic in mice.

I thank Prof. H. A. Bern, Dr. S. Nandi and Dr. J. J. Elias for advice. I also thank Miss L. Pissott and Miss D. Brown for their assistance and Prof. C. H. Li for the prolactin and somatotrophin. This work was supported by U.S. Public Health Service grants *CA*–05388 and 5–*T*1–5045.

Evelyn M. Rivera

Department of Zoology and its
Cancer Research Genetics Laboratory,
University of California,
Berkeley, California.

[1] Nandi, S., and Bern, H. A., *J. Nat. Cancer Inst.*, **24**, 907 (1960).
[2] Bern, H. A., and Nandi, S., *Prog. Exp. Tumor Res.*, **2**, 90 (1961).
[3] Nandi, S., *Proc. Soc. Exp. Biol. and Med.*, **108**, 1 (1961).
[4] Rivera, E. M., *Endocrinol.*, **74**, 853 (1964).
[5] Elias, J. J., and Rivera, E. M., *Cancer Research*, **19**, 505 (1959).
[6] Rivera, E. M., and Folley, S. J. (unpublished work).
[7] Barnawell, E. B., thesis, Univ. of Calif., Berkeley (1964); *Amer. Zool.*, **4**, abst., 117 (1964).

Interspecific Spore Inhibition in the Cellular Slime Moulds

Experiments were designed by one of us (H. M. S.) to determine whether or not there was any kind of competition between different species of slime mould when they were inoculated in the same culture dish. If *Escherichia coli* (as a food source) was spread with an inoculation loop over the surface of non-nutrient agar[1] in a Petri dish and approximately equal numbers of spores from two species of slime mould were mixed in one small spot, then invariably only one of the species developed (either in the light or in the dark); the second species appeared to be completely inhibited. Using this method, the following hierarchy of inhibition was established: *Dictyostelium purpureum* (No. 2) > *Polysphondylium violaceum* (No. 6) > *D. discoideum* (No. 1) > *D. mucoroides* (No. 2). This method was also attempted using the wild type of strain No. 6 of *D. mucoroides* and a small mutant derived from the same strain. In this case also, one form (the wild type) completely inhibited the development of the mutant.

In an attempt to make the tests more quantitative, quite conflicting results were obtained and the difference in the techniques provided a clue to the mechanism of competitive inhibitions. With this second method, the spores were suspended in distilled water, counted, centrifuged down and again placed on a small spot as in the previous method. Here, no matter what combination of species was used, equal development was evident in the first few days; there was no inhibition.

The explanation of this apparent conflict came from some experiments that were in progress independently in the same laboratory (C. C.). A few years ago Russell and Bonner[2] showed that densely plated spores had a far lower percentage germination than sparsely plated spores, suggesting the presence of a spore germination inhibitor. This has now been shown to be the case. If spores of *D. discoideum* are washed four or five times with distilled water they will show more than 95 per cent germination at very high densities. It is even possible, after removing the inhibitor from the spores, to concentrate it by boiling, and a very low percentage of spores placed in such a concentrate germinates, even when they are sparsely plated.

From this evidence we may conclude that the germination inhibitor is the effective agent in preventing growth of one species when the spore mixtures are made in dry and concentrated conditions. The inhibition can be removed either in a single species or in mixtures of species by washing or dilution in water. We must presume that the reason for the hierarchy of inhibition is that there is a differential susceptibility to a single inhibitor or possibly different inhibitors.

Howard M. Snyder, III
Costante Ceccarini

Department of Biology,
Princeton University,
Princeton, New Jersey.

[1] Shaffer, B. M., *Nature*, **157**, 133 (1946).
[2] Russell, G. K., and Bonner, J. T., *Bull. Torrey Bot. Club*, **87**, 187 (1960).

Temperature-controlled Meristic Variation in the Salamander *Ambystoma gracile*

The number of meristic parts in fishes (vertebrae, fin rays, scales, etc.) is often susceptible to modification by temperature or other environmental factors to which the developing embryo is exposed. This has been demonstrated experimentally on at least a dozen fish species[1,2]. In reptiles, one experiment[3] showed that the number of scales on a snake could be raised by higher incubation temperature. Serially repeated parts are less variable in amphibians than in fishes, but among the urodeles the number of vertebrae (or of somites) is known to vary intraspecifically among wild populations, and is sometimes used taxonomically. A hereditary basis to the number of trunk segments in *Plethodon cinereus* has been demonstrated by rearing young from different populations under constant conditions[4]. This communication describes the converse experiment, in which urodele eggs from one locality were reared at various temperatures, to determine whether different environments could produce different meristic counts in young having similar genetic background.

Egg masses of the north-western salamander, *Ambystoma gracile* (Baird), were collected by A. L. Hamilton on April 17, 1964, fron Jacobs (Marion) Lake, 7 miles north-north-east of Haney in the lower Fraser River valley of British Columbia. Each egg mass consisted of from 60 to 140 eggs distributed in a clear stiff jelly matrix. Egg masses had been attached to plant stalks a few inches above the mud bottom in 2–5 ft. of standing water, at 3°–4° C. Each batch was probably laid by a single female within the preceding 2 days.