easily and quickly lowered or raised (Fig. 2). Thus, the necessity of a time-consuming prehypothermic state, the shock of surface cooling with the subsequent biological catastrophe of the "stress phenomenon," and the arrhythmia of ventricular fibrillation that are encountered in previously used external cooling methods are eliminated by use of this internal method.

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Anaphylactic Shock in Guinea Pigs Sensitized to Polytyrosylgelatin

The lack of antigenicity of gelatin has been explained by the deficiency of aromatic groups in this protein. As a matter of fact, the attachment of O-β-glucosido-N-carbobenzoxytyrosine (*1*) or N-carbobenzoxytyrosine (*2*) to gelatin gave substances which elicited the production of antibodies. The finding of Maurer (*3*) that gelatin shows weak antigenic character in man does not seem to invalidate the assumed role of tyrosine in enhancing antigenicity. In this paper, we want to report the sensitization of guinea pigs by repeated injections of a modified gelatin in which L-tyrosine polypeptides are attached to the free amino groups of gelatin through peptide bonds (*4*).

The polytyrosylgelatin was prepared as follows: O-carbobenzoxy-N-carboxy-L-tyrosine anhydride (*5*) was polymerized in aqueous dioxane solution (1 to 1) at *p*H 7.0 (phosphate buffer) and 5°C in the presence of gelatin (*6*), containing less than 1 percent tyrosine. The carbobenzoxy groups of the polycarbobenzoxy-L-tyrosylgelatin obtained were removed with anhydrous hydrogen bromide in glacial acetic acid, and the product formed was dialyzed against water. The polytyrosylgelatin that was obtained contained 16 percent tyrosine as determined spectrophotometrically. Unlike poly-L-tyrosine, which is soluble in water only in the presence of strong alkali (*5*), polytyrosylgelatin is soluble in water, acids, and bases. Since polytyrosine is insoluble at physiological *p*H, a copolymer of L-aspartic acid and L-tyrosine in a residue molar ratio of 9 to 1 was prepared for comparison.

Guinea pigs weighing 200 to 250 g received intra-abdominally three injections of 0.5, 1.0, and 2.0 ml, respectively, of the substance to be tested for its sensitizing potency. Five-percent solutions of gelatin and polytyrosylgelatin and 1-percent solutions of the copolymer of tyrosine and aspartic acid were used, and the injections were given at 3-day intervals. Fifteen days after the last intra-abdominal injection, all the pretreated animals, as well as an equal number of nontreated controls, received intracardial injections of 0.25 ml of solutions of the substances to be tested for their antigenicity.

Two out of five guinea pigs that were sensitized with polytyrosylgelatin exhibited large drops in body temperature after an intracardial injection of a 0.2-percent solution of the homologous substance, while three showed typical anaphylactic shocks and died (see Table 1). No serious symptoms were observed in nonsensitized animals or in animals that were pretreated with the copolymer, even when 2-percent solutions of polytyrosylgelatin were injected. Gelatin injected as a 5-percent solution, or the copolymer as a 1-percent solution, did not produce in sensitized or in untreated animals any significant symptoms except slight drops in temperature.

The results obtained (Table 1) show clearly that polytyrosylgelatin injected intra-abdominally sensitizes guinea pigs against intracardial injection of the same compound. Since no sensitization was observed by a similar treatment with gelatin, or with a copolymer of tyrosine and aspartic acid, it seems plausible to assume that the attachment of tyrosine peptides enhances the antigenicity of gelatin.

Table 1. Anaphylactic reactions in nonsensitized and sensitized guinea pigs. G, gelatin; PTG, poly-L-tyrosylgelatin, containing 16 percent tyrosine; CAT, copolymer of L-aspartic acid and L-tyrosine in a residue molar ratio of 9 to 1.

Previous treatment	Intracardial injection	Animals (No.)	Death (No.)	Avg. temperature decreases (°C)
Nonsensitized	G	5	0	0.68
G	G	6	0	1.35
Nonsensitized	PTG	5	0	0.80
PTG	PTG	5	3	3.60
CAT	PTG	5	0	0.50
Nonsensitized	CAT	5	0	0.80
PTG	CAT	5	0	1.20
CAT	CAT	5	0	0.40

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Histological Changes Induced in Soybean Roots by 2,4-Dichlorophenoxyacetic Acid

The anomalous structure caused in the hypocotyl of soybean seedlings by treatment with 2,4-dichlorophenoxyacetic acid (2,4-D) has been reported in an earlier paper (*1*). The present investigation (*2*) is concerned with the histological changes in response to treatment with 2,4-D in the primary roots of soybean seedlings. The method used was essentially the same as that described in the earlier paper (*1*).

Retardation of elongation in the root became apparent on the third day after treatment. The tips of the treated roots were slightly larger in diameter than those of the untreated ones. Histological preparations were made of normal roots and of roots treated with 2,4-D.

Before describing the treatment with 2,4-D, it may be well to review briefly the anatomical structure of the soybean root. The root has a unistratose epidermis. The cortex consists of eight to 11 layers of parenchyma limited on the inside by the endodermis. Immediately within the endodermis lies the pericycle, which is one or two cell layers in thickness adjacent to the primary phloem, and two or three cell layers in thickness opposite the protoxylem ridges. The primary phloem consists of four strands of tissue alternating in position with the protoxylem ridges of the tetrarch structure (Fig. 1).

In the 2,4-D-treated roots, the epider-

mis exhibited no specific response. By the end of 72 hours, however, the cells of the pericycle, endodermis, and inner cortex, in many cases, showed divisions in the radial as well as in the tangential plane (Fig. 2, top). Pericycle cells were especially active, producing a narrow zone of meristematic tissue, which extended well back from the apical region. Active cell division within the pericycle continued until, eventually, the boundary between this tissue and the adjacent tissues became indistinguishable. Because of this active cell division, the pericycle produced a conspicuous mass of dense meristematic tissue, which extended outward toward the periphery (Fig. 2, bottom). The cells of the outer cortex at the basal portion of the root began to collapse during the third day.

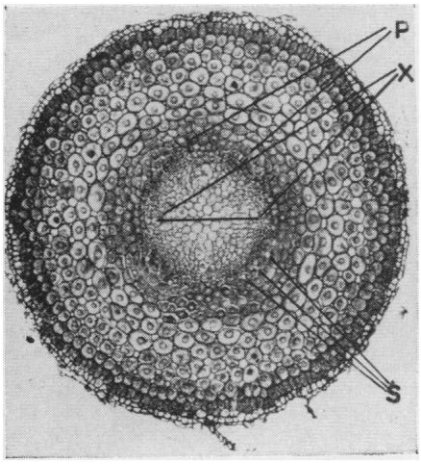

Fig. 1. Transverse section of soybean primary root taken 996 μ behind the stelar initials. *P*, pericycle; *S*, sieve tube; *X*, primary xylem (× 79).

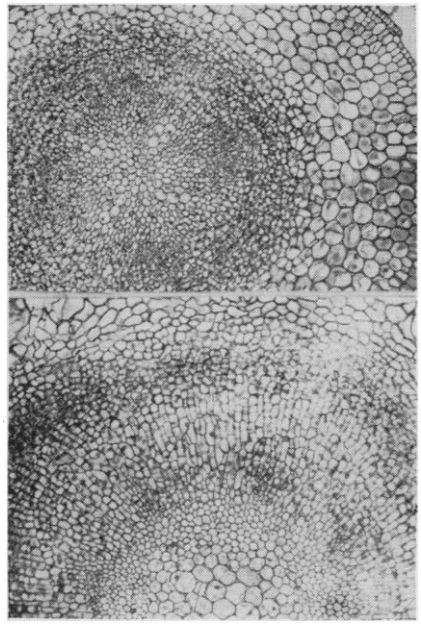

Fig. 2. Transverse sections of soybean primary root, 3 days after treatment with 2,4-D. (Top) Transverse section taken 924 μ behind the stelar initials, showing proliferation of inner cortex, endodermis, and pericycle. (Bottom) Transverse section at the basal region of the primary root showing a conspicuous mass of proliferated tissue produced by the pericycle. The boundaries of pericycle, endodermis, and cortical parenchyma have become indistinguishable. The indented outline suggests the formation of numerous fasciated lateral roots (× 68).

The primary phloem also exhibited a similar response to this treatment. At a distance of about 380 μ from the tip, the nonconducting cells of the primary phloem became meristematic. At a greater distance (about 1640 μ from the tip), even greater meristematic activity was apparent. Here the primary phloem, with the exception of the sieve-tube elements, became indistinguishable from the adjoining areas.

Close to the apex there was no response in the primary xylem. Some dividing cells, however, were observed in the metaxylem in the basal regions.

The striking effect of 2,4-D on the root of soybean is a stimulation of cells in certain tissues to high meristematic activity. The resulting mass of cells is nonpolarized and does not seem to be made up of numerous, closely placed lateral-root primordia as has been suggested by Wilde (*3*). Apparently 2,4-D retards root elongation and disrupts the orderly biochemical and physiological processes of normal development, particularly within the stele and inner cortex, resulting in a change to high meristematic activity within these regions.

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Disease in the Giant African Snail *Achatina fulica* Bowdich

Between March and December 1954, an investigation was conducted to determine the causes for the recent sharp decline in Ceylon populations of the giant African snail (*Achatina fulica* Bowdich) (*1*). One of the most striking aspects of the field research was that in some areas, even after hours of collecting and examining individuals, the oldest live specimens were found to be no more than 1.5 years old.

In many populations, the oldest live specimens were between 2 and 3 years of age, although dead specimens in excess of 5 to 6 years of age were almost countless. In other areas, long hours of diligent hunting failed to bring to light a single live specimen in spite of the fact that 2 to 3 years earlier the place had been overrun with snails.

The last sudden decline in the snail populations in Ceylon took place in 1952, a year during which the large stores of metaldehyde that had been purchased as a result of numerous outbreaks of snails in 1951 were scarcely touched. This reflects a cataclysmic decimation, and perhaps even a near or actual local extermination, of the population. The uniformity of age and the conspicuous absence of older individuals suggest that the present populations are the surviving offspring of those subjected to this catastrophic force. As had earlier been anticipated (*2*), the picture in general was found to be not that of predation but of parasitism or pathogenesis with variable, localized predation.

Of the several predators, only the firefly *Lamprophorus tenebrosus* appeared to offer any possibility as an agent in the biological control of the giant snail. In one area near Pallekelle in the Central Province, however, the fireflies and achatinas had been together for a minimum of 25 years, and the snails could still be classed as "common to abundant" in spite of reasonably common fireflies. Further, it was established both in the laboratory and in the field that the larger the giant snail, the less likely it was to be attacked and killed by the glowworm larva. Therefore, predation by glowworms cannot explain the complete absence of older individuals in some of the erstwhile large, vigorous populations.

In seeking a cause for the manifest sharp decline in the populations of the giant African snail in Ceylon, I found that the greatest amount of evidence pointed toward the existence of a nonspecific, chronic disease of uncertain etiology. In the many different environments examined, the only discernible common factor that was unfavorable to the survival of the snails was a fairly constant syndrome. The most conspicuous symptom of this syndrome was the presence of leukodermic lesions on the fore part of the body (Fig. 1).

The first sign of pathogenesis is the presence of vague, patchy, granular areas on the tentacles, face, and neck. Microscopic examination reveals that the melanophores in the dermis of these areas are undergoing complete distintegration and disappearance. Concomitantly, there is a proliferation of dermal connective tissue cells. The epidermis, however, remains intact. Hence, there is no appearance of frank ulceration. But the epidermis reflects the dermal disturbances by