

2 by 2 by 2 mm, in a moist chamber under 220 ft-ca of fluorescent illumination. Diffusion of auxin into the agar blocks took place during a period of 2 hours after which the auxin content was assayed by the usual *Avena* curvature test. Treatment with gibberellin doubles the diffusible auxin in the normal variety and triples the amount in the dwarf variety (Table 1). Corresponding to the increase in diffusible auxin there is an increase in plant height. These data are typical for plants grown under sunny weather. If cloudy days predominated during the growing period the plants were not responsive to treatment. Such dependence on light intensity may be the explanation of other findings in which gibberellin treatment had little effect on the height of the Alaska variety of pea (3).

The relationship of gibberellin treatment and diffusible auxin content has been examined also in the sunflower plant, variety Mammoth Russian (*Helianthus annuus* L.). One hundred and twelve embryos were sown in soil in a wooden flat and grown under the same conditions as those used for the peas. Ten days after sowing the first foliage leaves appeared and were treated with 0.1 ml of gibberellin solution in the evening. Two days later a second treatment was given to the same leaves. When the second internode was 0.5 to 2.0 cm long the apical portion with leaves was excised, and diffusible auxin was obtained in the same way as before during a period of 100 minutes under 95 ft-ca. The curvature of *Avena* coleoptiles induced by diffusible auxin from plants treated with gibberellin is shown in Table 1, together with the height of the plant at the time of sampling. Again the increase in plant height brought about by treatment with gibberellin is associated with an increase in the diffusible auxin obtained from the apical portion of the plant. For plants treated with $3 \times 10^{-3}M$ gibberellin the diffusible auxin was 10 times that from untreated plants. From this investigation it must be concluded that as Shibaoka and Yamaki suggested (6): "the growth of the stem of the sunflower seedling depends closely on the quantity of auxin supplied from the leaf."

These results correspond to the finding of Nitsch (7) that in the shoot tips of sumac treated with gibberellic acid the amount of extractable auxin is greater than it is in untreated plants.

Although Nitsch was unable to conclude that a decline in growth rate under short days was the result of a lowering of auxin level since there was also an increase in the level of endogenous inhibitors, his data for plants under long days clearly indicate that as a result of treatment with gibberellin there is a larger increase in endogenous auxin than there is in endogenous inhibitors; this increase is associated with increased growth rate.

In the indoleacetic acid oxidase-inhibitor theory of growth regulation (2) the increase in diffusible auxin resulting from gibberellin treatment would be explained by an increase in the inhibitor content which prevents enzymatic destruction. It is just as plausible that gibberellin treatment may directly increase the formation of auxin. Investigation of this mechanism as a possibility is in progress. Studies of the growth effects of gibberellin treatment which include the examination of auxin production may relate all responses to auxin levels (8).

SUSUMU KURAISHI

ROBERT M. MUIR

Department of Botany,
State University of Iowa, Iowa City

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Salmonella Species in Turtles

Abstract. *Salmonella* spp. occur abundantly in the feces of the turtles *Testudo graeca* and *T. hermanni* but rarely in other testudines. These organisms do not seem to produce any kind of infection in the turtles. One possible explanation for the infestation is the coprophagic habit of *T. graeca* and *T. hermanni*.

In the course of inquiries into the origin of a paratyphoid infection in a child in 1952, a chance observation showed that *Testudo graeca* that were imported as household pets contained

Table 1. Species of turtles of the genus *Testudo* in which *Salmonella* serotypes were found.

Species	Number examined	Number infested
<i>T. graeca</i> (captive)	45	41
<i>T. hermanni</i> (wild)	50	33
<i>T. angulata</i>	1	0
<i>T. chilensis</i>	10	1
<i>T. denticulata</i>	5	0
<i>T. elegans</i>	8	0
<i>T. gigantea</i>	7	0
<i>T. nigrita</i>	3	0
<i>T. pardalis</i>	3	1
<i>T. radiata</i>	4	2
<i>T. sentoria</i>	1	0
<i>T. vicina</i>	1	1
<i>T. sp.</i> (Isla Santa Cruz)	10	0

Salmonella spp. in their bowel in large numbers (1). This finding has been confirmed in other countries in northern Europe. The conditions under which these reptiles are imported make cross infection likely; however, Hirsch reported similar infestations in wild turtles caught near Haifa, Israel (2). Vincent, Neel, and Le Minor found that 96 percent of *Testudo graeca* in the countryside around Tangiers contained one or more serotypes of *Salmonella* (3); at several sites in Dalmatia (Yugoslavia) I found infestation of 70 to 80 percent in the closely related *Testudo hermanni* which I examined.

Other *Testudo* spp. have been less productive (Table 1). Some of the turtles whose feces were examined have been in zoological collections, but the majority of them were taken in the wild. The numbers of each species examined were small, but the general picture suggests that only *T. graeca* and *T. hermanni* (and their subspecies) harbor large numbers of *Salmonella*. I have found that turtles in captivity may continue to harbor these organisms for at least 9 years without any evidence of illness but the number of *Salmonella* in any individual and the number of infected individuals both decrease slowly.

One possible explanation for the infestation of *Salmonella* is that both *Testudo graeca* and *T. hermanni* are coprophagic. Where they occur wild this fact seems to be well known. I have found wild *T. hermanni* chewing horse dung, and in captivity they will eat human, bovine, or their own feces with avidity even when fresh lettuce leaves are available. After eating human feces which were naturally infected with *Salmonella typhimurium*, one tur-

tle in my collection continued to excrete this organism for 3 years while remaining in apparent good health. An observer in the south of France has informed me that *Testudo hermanni* is also fond of the more highly flavored scraps which spill from the garbage bin. Though the evidence is scanty I have not discovered any mention of coprophagy as a habit of the other members of the genus *Testudo*.

J. A. BOYCOTT

Public Health Laboratory,
Taunton, England

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Mechanism of Tissue Reconstruction by Dissociated Cells, II: Time-Course of Events

Abstract. The details of the process by which cells sort out and reconstruct tissues within aggregates containing two kinds of tissue cells have been correctly predicted from considerations of the kinetic and adhesive properties of such cells. The requisite properties are discreteness, motility, and differential mutual adhesiveness among the types of cells present.

Organs or regions of the body of vertebrate embryos may be dissociated into their component cells, which are then capable of reaggregating and sorting out to reconstruct semblances of the original structure (1, 2). In these autotrophic structures the reconstituted tissues are deployed in their normal mutual histological relationships. Such organization usually involves the formation of discrete inner and outer tissues.

In a previous paper (3) it was shown that individual cells of the prospective internal tissue do not migrate in a directed fashion toward the center of an aggregate. There did appear to be selection against the residence of such cells at the very surface, however. It was concluded that sorting out must proceed in a manner analogous to that in which a dispersion of mutually immiscible liquids "breaks." In such a dispersion the liquid of lower surface tension (that is, lower molecular co-

hesiveness or mutual attraction) quickly occupies the surface of the liquid body, during and after which the droplets of the liquid of higher surface tension progressively coalesce to produce a decreasing number of increasingly large islands in the interior. Thus external (continuous) and internal (discontinuous) phases are established. The behavior of such a system is due to its possession of three properties: (i) the two phases are composed of units which are discrete; (ii) the units are mobile; (iii) the different kinds of units are differentially cohesive and adhesive. The first two of these properties are of course known to be characteristic of most cells; but differential mutual adhesiveness, while known for certain kinds of cells, is not established as of general applicability. If sorting-out indeed depends upon differential mutual adhesiveness among the cells in a mixed population, the time-course of events which characterize the process must conform with that given above with reference to dispersions.

Figure 1 shows the sequence of events in the sorting out of chick embryonic heart cells from chick embryonic retinal cells. By virtue of a staining reaction for glycogen (4), which they alone contain, the heart cells, derived from 5-day embryos, are distinguishable from the retinal cells, derived from 7-day embryos. Techniques are described elsewhere (3). The first event in sorting out is the withdrawal of heart cells from the surfaces of the aggregates. Accompanying this is an initial clustering of heart cells in innumerable foci throughout the interior of each aggregate. These heart foci continue to encounter and fuse with one another, progressively building up one or more coherent, internal masses of heart tissue, the number of which reflects the proportion of heart cells in the population. Townes and Holtfreter have previously described the same sequence of events with amphibian neurula chordamesoderm and endoderm (2).

An alternative explanation of the sorting-out phenomenon has been advanced by Curtis (5), who suggests that cells of different types undergo certain surface changes at different times after their dissociation. These changes would be such that cells which had experienced them would be trapped by contact either with the surface of an aggregate or with other cells already

so trapped. Thus cells of the first type to experience the change would be trapped initially at the surface and then in sequential layers beneath it, leaving those of the type which experiences the change later to be trapped in the

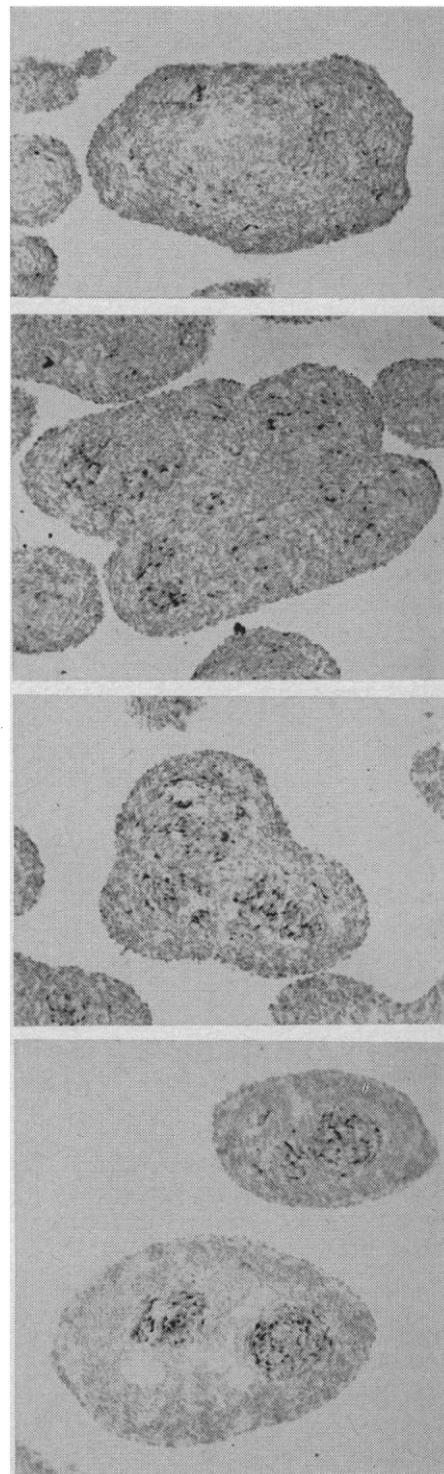


Fig. 1. Sections through aggregates containing chick embryonic heart (darkly stained) and retinal cells fixed at (top to bottom) 17, 24, 31, and 66 hours of incubation at 37°C, showing the process of sorting out ($\times 127$).