aerated water. After 1 wk, the plants were removed and new plants were substituted. Four days later the trifoliate leaves of those growing in water that had previously supported growth of treated plants showed typical MOPA effects. Width of the modified leaves was 88 percent and their length 79 percent less than that of leaves of plants grown in water that had previously supported the growth of the untreated plants.

Snap bean, sunflower, cucumber, buckwheat, cotton, and corn seeds were germinated together in composted soil in 6-in. clay pots to determine whether MOPA would move from one kind of plant to another through their roots systems. After the pots had been divided into six groups with equal numbers of plants, MOPA was applied in the carrier as a band around the stems of all plants of one species in the first group. Plants of another species were treated in the second group, and so on, until each species (except corn) had been treated and was growing with untreated plants of the remaining species. Corn plants in one group were treated by applying a band of the paste, about 1 cm wide, across the base of the first true leaf. On the basis of the formative effects induced, MOPA moved from bean plants to sunflower, cucumber, buckwheat, and cotton; from cucumber to bean, sunflower, buckwheat, and cotton; from sunflower to bean; from buckwheat to bean; and from cotton to bean. There was no evidence that MOPA moved from corn to any other plants.

A wide variety of growth-modifying compounds known to induce formative effects when applied to bean plants were tested to determine whether they moved from one plant to another through the root systems. In these tests, about 150 to 200 μg of each compound tested was applied in the Tween-20-lanolin carrier as a band around the stem of one of two bean plants growing in the same pot. The following compounds (*6*) were tested: 2-naphthoxyacetic acid; 3,4-xylyloxyacetic acid; 2,4-dibromophenoxyacetic acid, 2,3-dichloropropyl ester; para-chlorophenoxyacetic acid, 2-butenyl ester; para-chlorophenoxyacetic acid, 2-methylallyl ester; 2,4-dichlorophenoxyacetamide; 2,3,5-triiodobenzoic acid; and 2,4-dichlorophenoxyacetic acid and 6 of its salts and esters. In addition, 14 other chlorine-substituted phenoxyacetic acids (*7*) were tested, all of which were known to induce growth modifications when applied directly to bean plants. None of the compounds was moved from the treated plants to the untreated ones in sufficient amounts to induce visible growth effects.

MOPA apparently did not accumulate and remain in detectable amounts in soil in which treated plants were grown. Bean plants treated with about 150 μg of MOPA on the first internodes were removed from soil in which they had grown for 2 wk. Bean seeds were immediately planted in the soil, but the resulting plants did not develop growth modifications.

When a treated and an untreated plant were grown in soil contained in a pot, MOPA may have moved from roots of the treated one through the water films into roots of the untreated one and apparently through those roots that were in contact with each other, or at least through those that grew very near to each other. Close proximity of bean roots was repeatedly observed, sometimes as many as five roots growing in contact with each other for a distance of 1 in. or more.

References and Notes

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2. J. W. Mitchell and W. H. Preston, Jr. *Science* **118**, 518 (1953).
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Central Nervous System Gliogenesis in Piromen-Treated Rat Embryos

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In addition to its pyrogenic properties, the bacterial polysaccharide Piromen has been shown by Windle, *et al.* (*1*) to possess the ability to inhibit glial proliferation in traumatic lesions of the spinal cord and brain of mammals. These investigators employed a dose of 0.5 micrograms per kilogram (μg/kg) of body weight, administered intravenously daily for 2 wk.

Because of these results, we have attempted to inhibit the normal maturation of central nervous system glial tissue in the rat embryo and in the developing rat with Piromen. Since intravenous administration was impractical, relatively large doses were given subcutaneously and intraperitoneally. Fischer females were bred with Agouti males (both are Curtis-Dunning lines). The Piromen treatment schedule was varied in three ways.

A-1. Injections were begun on the 10th to 12th days of pregnancy via the intracardiac or intraperitoneal route. Four animals were treated for 1 to 5 days and received total doses of 4 to 10 μg/kg. Embryos were obtained at the 12th and 15th days of pregnancy and immediately after birth.

A-2. Injections were begun on the 16th to 19th days of pregnancy via the intraperitoneal route. Five animals were treated for 1 to 4 days and received total doses of 0.4 to 1.8 μg/kg. The animals were allowed to come to term, and 25 young rats were sacrificed at postpartum days, 0, 1, 2, 3, 7, 12, and 40.

B. Siblings from animals treated under schedule A-2 were given daily injections after birth. The injections were first made subcutaneously and later intraperitoneally as the animals grew. Three animals sacrificed at the age of 2 or 3 days had received total doses of 0.5 to 2 μg/kg. Nine other animals received

progressively increasing doses during the first 23 days of life, the total dose was 150 to 275 μg/kg. The animals were sacrificed at the age of 39 days.

C. The mothers were not treated. The animals were treated as under schedule B, the total dose was 2.5 to 15 μg/kg in 23 days. There were eight animals in this group. They were sacrificed at 23 or 28 days of age.

Approximately an equal number of embryos and young rats from mothers that received no treatment were sacrificed for control studies. Also, litter mates of young rats receiving Piromen in both groups B and C were given injections of 0.9 percent sodium chloride as a further control measure.

Sections from representative levels of the brain and spinal cord of both the injected and control material were stained by the hematoxylin and eosin, Nissl, phosphotungstic-acid hematoxylin, Cajal gold sublimate, and Hortega silver carbonate methods. A comparison between the injected and the control animals showed no differences in the condition of either glial or neuronal tissues.

Even though large doses of Piromen were given at a time when gliogenesis was taking place, we could discern no effect from its administration in the manner described. We are unable to answer the possible questions whether Piromen crosses the placental barrier and reaches the central nervous system of the embryos, or whether an adequate concentration of Piromen is produced in the central nervous system by intraperitoneal or subcutaneous administration.

Reference

1. W. F. Windle, C. D. Clemente, D. Scott, Jr., and W. W. Chambers. *Induction of neuronal regeneration in the central nervous system of animals* (Tr. Amer. Neur. Assoc., 1952), pp. 164–170.

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Development of Lymphoid Tumors in Nonirradiated Thymic Grafts in Thymectomized Irradiated Mice[1]

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Lymphoid tumors and lymphatic leukemias arising either spontaneously or in response to exogenous agents (x-rays, estrogens) tend to originate in the thymus in several mouse strains (*1–3*), and their incidence may be drastically reduced by thymectomy (*1–6*). That the effect of thymectomy is not due simply to removal of potentially malignant cells was demonstrated by Law and Miller (*7*), who found that lymphatic leukemia incidence in methylcholanthrene-treated dba mice could be restored to control levels by autologous or homologus thymic implants, although the implants were seldom involved by the leukemic process. This observation was recently confirmed when Kaplan, *et al.* (*8*) noted a significant increase in lymphoid tumor incidence in thymectomized, irradiated C 57 black mice bearing homologous thymic implants.

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FIG. 1. Arrow points to lymphoid tumor mass replacing thymic implant. All other lymphoid tissues were microscopically normal. (×3)

An experiment was then set up to test the influence of such factors as the age of the thymic implants and the time of their implantation on tumor response. The complete experimental design is not pertinent to the present report, which is concerned only with those groups that were treated essentially as follows: C 57 black mice, segregated by sex, were thymectomized by our usual technique (*5*) at 30 to 40 days of age and were started on whole-body irradiation 2 to 7 days later. They received four doses of 168 r each at 8-day intervals; physical factors were: 120 kv, 9 ma, 0.25 mm Cu + 1.0 mm Al added filter, 30 cm mouse-target distance, 32 r/min. Thymus glands were rapidly excised from intact, nonirradiated 33-day-old C 57 black donors and were immediately implanted subcutaneously in the right axillary region of each recipient mouse. Most grafts were made within 1 hr after the last irradiation, but mice of two groups received implants 1 and 8 days, respectively, after irradiation.

Tumors were observed as early as 4 mo after thymic implantation, but most of these had disseminated by