Detailed reports on the chemical and biological properties of alanosine will be published elsewhere.

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PATHOLOGY

Trauma and Melanoma Production

It has long been held that malignant melanoma of man can arise as a result of trauma to a mole (naevus or benign melanoma) or even normal skin¹⁻⁴. Other workers⁵⁻⁸, however, do not accept this thesis. They believe that as yet there is no authenticated case of a mole having become malignant as a result of trauma and that the local appearance of a malignant melanoma after incomplete removal of a supposed benign naevus indicates that the lesion was already malignant before removal.

Hitherto, only one attempt has been made to investigate this problem in the experimental animal. My colleagues and I⁹ traumatized about eighty carcinogen-induced benign melanotic tumours in the Syrian hamster but failed to induce malignant change or even a modest growth stimulation in any of these tumours. In fact many of the lesions regressed as a result of trauma.

This problem has now been investigated in another experimental animal, and forms the subject of this report.

It is now well established^{10,11} that deeply infiltrating and destructive melanomata can be produced in various viviparous fishes by hybridization and that these tumours arise as a result of the genetic imbalance produced, which permits the proliferation of macromelanophores and melanocytes. In most instances this proliferation affects large areas of the skin which are thickened (a state referred to by Reed and Gordon¹⁰ as melanosis) and it is from such genetically predisposed sites that the malignant melanoma ultimately arises.

In this respect the piscine model is similar to the human, for most human melanomata arise from pre-existing moles which are considered to be areas of melanocyte proliferation produced as the result of a developmental defect⁴. Therefore, it appeared worth while to investigate the effect of trauma to the skin of *Xiphophorus maculatus* × *X. helleri* hybrids showing various grades of melanosis to see if frank melanomata could be precipitated by this procedure.

With this in view 112 hybrids of both sexes, aged between 3 and 5 months, were divided into two equal groups. The skin of each experimental animal was traumatized in one of the three following ways: (1) Pricking with a needle over a selected area once a week for 3 months. (2) Excising a strip of skin measuring about 5 mm × 1 mm. (3) Excising portions of dorsal or caudal fin through hyperpigmented areas. The experimental and control animals were observed until they died, or for a period of 12 months. During this period three melanomata developed in the control group and four in the experimental group, but in no case did a melanoma arise from, or near, a traumatized site.

These observations indicate that single or repeated mechanical trauma does not induce melanomata to develop in the skin of these hybrid fishes genetically predisposed to melanoma formation. These observations are therefore in keeping with the observation that trauma does not induce malignant change in the benign melanomata of hamsters and also with current opinion that trauma does not precipitate malignant change in human moles.

It is extremely difficult to evaluate the role of trauma in tumour production or in the transformation of benign tumours into malignant ones. There is, however, fairly conclusive evidence that mechanical trauma can produce epithelial tumours from skin that has been pretreated with carcinogens. This has been shown to occur both in the rabbit and man¹². The situation regarding melanocytes and melanomata, however, seems to be different, for neither recent clinical opinion nor experimental investigation supports the idea that melanomata arise as a result of trauma.

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IMMUNOLOGY

Transfer of Experimental Nephritis in Rats by means of Lymphatic Duct Lymphocytes

THE successful transfer of nephrotoxic nephritis (Masugi's method) in rats to essentially healthy animals of the same inbred strain by parabiosis was described earlier^{1,2}. In these experiments, transfer of rabbit antibody from the first into the second rat was excluded by the fluorescent antibody technique³. Recently, experiments were reported in which the so-called auto-immune nephrosis was also induced by parabiotic transfer⁴. Furthermore, this disease has been transferred by means of lymph node cells⁵. Nephrotoxic nephritis was "transferred" by peripheral white blood cells of nephritic donors⁶. It was assumed that blood lymphocytes were the immunologically responsible cells and caused the renal lesions in the recipient. It was considered that further investigations of the transfer of circulating lymphocytes obtained from a lymphatic vessel would prove whether or not this assumption was correct.

Sprague-Dawley rats of an inbred strain were injected with 0.5 ml. of nephrotoxic rabbit serum per 100 g body weight. Between twelve and sixteen days later, when the nephritic lesions were fully established, the lymphatic vessel accompanying the superior mesenteric artery was cannulated with a PE 50 polyethylene catheter using the Bollmann technique⁷. The animals so treated were kept in a special cage throughout the experiment which allowed forward and backward movement, but prevented the animals from turning around. The rats were fed an ordinary diet and received water *ad libitum*. The outflowing lymph was collected in tubes containing Hanks's solution with 100 units of heparin. Up to 15 ml. of lymph fluid was obtained from a single donor. The recipients of lymphatic material were divided into three groups. The first group of twenty-seven animals received the whole lymph containing 100–500 × 10⁶ lymphocytes in 10–12 ml. lymphatic fluid. The material was injected intravenously in from four to thirteen portions. In the second group, twenty-two rats were given isolated lymphocytes in a total of 70–300 × 10⁶ cells injected in between two and nine portions. When preparing the cell suspension, the whole lymph was centrifuged for 20 min at 1,000 r.p.m. The supernatant was discarded and the cells were resuspended in Hanks's solution. This procedure was