It is evident that the substances *T* 375 and particularly *T* 356 inhibit growth of experimental sarcoma tumour. The relatively high toxicity of the substance should, however, be emphasized.

Detailed description of the experiments, including histological examination, will be reported elsewhere.

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Œdema Formation in Rat's Skin

It is known that histamine liberators such as 48/80, egg white and dextran produce in rat skin œdema and capillary changes which lead to accumulation of circulating colloidal dye. These changes show a characteristic distribution and occur in skin regions, such as those of feet and face, which are particularly rich in histamine[1]. However, the part of histamine in these changes has been questioned. When discussing these changes it is necessary to deal separately with the œdema formation and the dye accumulation. The œdema is a sign of greatly increased capillary permeability. Concerning the accumulation of a circulating colloidal dye such as trypan blue, the present concept is that the dye is bound in the bloodstream to plasma proteins which penetrate the dilated capillary wall, moving through the dilated capillaries into the damaged area. The mechanism for this penetration is the phagocytic activity of the endothelial cells, histiocytes and fibriocytes which take up the proteins. The accumulation of circulating dye thus indicates an increased local phagocytic activity. The present experiments show that release of histamine is responsible for the œdema formation but that the accumulation of circulating dye is independent of histamine release.

According to Feldberg and Talesnik[1] the œdema in the feet of the rat produced by intraperitoneal injection of 48/80 or egg white is due to release of histamine, because it no longer occurred when the skin had been depleted of its histamine by repeated intraperitoneal injections of 48/80. In contrast, Kramer[2] found that the histamine content of the rat's skin did not influence the œdema produced by egg white and that the œdema occurred also when the skin had been depleted of its histamine.

Using the technique of Brocklehurst *et al.*[3], rats were treated with intraperitoneal injections of 48/80 for eight days. After treatment for five days 48/80 no longer produced œdema, and after treatment for eight days egg white (2 ml. intraperitoneally) also failed to cause œdema. These results agree with those of Feldberg and Talesnik and differ from those of Kramer. They show that the œdema formation is dependent on the histamine release.

In rats in which the skin histamine had been depleted by repeated intraperitoneal injections of 48/80 and in which injections of histamine liberators no longer caused œdema, intradermal injections not only of histamine and of 5-hydroxytryptamine, but also of 48/80 and of egg white, retained the property of causing accumulation of circulating dye at the site of the injection. Intradermal injections of saline solution were ineffective. The dye used was trypan blue, which was injected into the tail vein immediately after the intradermal injections. The rats were killed 60 min. later and the inside of the abdominal skin examined for local dye accumulation. Similar results were obtained in two other series of experiments in which either the histamine in the rat's skin was depleted by dextran or egg white instead of by 48/80, or in which the trypan blue was replaced by indian ink. These results show that the changes responsible for the accumulation of dye or indian ink are independent of histamine release and therefore not an effect of histamine. A similar conclusion was reached by Brocklehurst *et al.*[3] for the dye accumulation of passive cutaneous anaphylaxis in rats in which the skin had been depleted of its histamine by 48/80.

Indian ink and trypan blue accumulation was not caused by the liberation of histamine or 5-hydroxytryptamine since these latter two substances were no longer present after the first day during repeated treatments with 48/80, dextran or egg white. Even in the absence of histamine or 5-hydroxytryptamine the reaction of accumulation continues, showing an unchanged functioning of the inflammatory defence mechanism.

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Effect of Blood on the Viability of Dried Cultures of Cholera Vibrios

Between 1949 and 1954 twenty-two strains of *Vibrio cholerae*, grown at 37° C. on 'Lemco' agar and suspended in 'Mist. desiccans'[1], were dried by the spin-freeze method developed by Greaves[2]. Viable counts were made by the Miles and Misra[3] technique on the suspensions used for drying, and on ampoules of dried material reconstituted at intervals after drying. In routine tests reconstitution was in 'Lemco' broth, which was also used as diluting fluid. All counts dropped rapidly, thus confirming the experience of others[4,5] that the vibrios were unduly susceptible to drying by sublimation.

Experiment showed that higher viable counts could be obtained (*a*) by using 5 per cent blood broth as diluent, or (*b*) by plating 'Lemco' broth dilutions on blood agar. This suggested that blood provided some essential factor for the recovery of cholera vibrios