Table 1

Treatment	Uterus from Strong-*A* mouse, grafted intraperitoneally five days before, with draining lymph nodes from Strong-*A* mice, bearing Crocker tumour (12 days old)	Normal Strong-*A* mouse uterus, similarly treated with normal mouse lymph nodes
1. *Rd*/3 rat tumour soluble protein	0	0
2. Normal mouse organ (liver, spleen, kidney) soluble protein	++	0
3. Desensitization with normal mouse organ soluble protein		
4. Crocker mouse tumour soluble protein	++++	±
5. Crocker mouse tumour soluble protein (heated)	±	0
6. Desensitization with Crocker mouse tumour soluble protein		
7. Crocker mouse tumour soluble protein	±	0
8. Acetylcholine (10 μgm./ml.)	++++	++++

were minced and injected intraperitoneally into normal female mice of the same strain. Controls were treated similarly with tissues from normal mice. Animals were killed at intervals, the uteri were removed immediately and tested in the Dale bath for sensitization, by exposing them to different test antigens and recording the reaction on a kymograph. Soluble protein, lipoprotein[7] prepared from fresh or lyophilized Crocker mouse tumour, *Rd*/3 rat tumour and normal mouse organs were used as test antigens. After contraction with one antigen, a uterus was washed completely and the same antigen was added repeatedly to test for desensitization. The reactivity of the tissue was tested with acetylcholine at the end of each experiment.

It was found that draining lymph nodes and spleens from tumour-bearing mice are very effective; the uterus so sensitized gives a positive reaction when exposed to Crocker tumour soluble protein, lyophilized Crocker tumour extract and Crocker tumour lipoprotein. After heating, the antigens do not have this effect. Other organ-grafts are not effective. No such contraction is seen in the uterus when exposed to normal mouse organ soluble protein, lyophilized normal mouse organ extract and soluble protein from *Rd*/3 rat tumour as test antigens. Heating of the lymph nodes prior to grafting also abolishes this effect. The reaction appears on the second day after grafting and disappears completely after 3 weeks. This reaction has no strain specificity; lymph nodes from Strong-*A* mice bearing Crocker tumours, grafted into *C*57 black mice and *vice versa*, also give a positive reaction.

A typical result is given in Table 1.

It was shown previously that the host animal (rat and mouse) reacts to a progressively growing transplanted tumour by developing a characteristic plasma-cellular reaction in the draining lymph node and spleen[2,8], which can be correlated with the increase of deoxyribonucleic acid and thus with protein synthesis in these organs[9]. It was suggested that the plasma-cellular response is the morphological manifestation of a systemic immune reaction, due to the antigenic stimulation from the graft, and that plasma cells are involved in the production of antibodies against the tumour[8]. Evidence has been presented by Billingham *et al.*[2] that this heightened resistance in 'adoptive immunity' is not due to passive transference of any preformed antibody from the primary hosts, but of immunologically activated tissue, which continues to function in these secondary hosts after transfer.

Although these experiments do not throw any light on this question, they demonstrate that the presence of antibody in the host is the basic mechanism involved in this passive tumour transplantation immunity. This investigation, which will be published later in detail, also confirms observations[1,3,8] that the regional lymph nodes and spleen are important seats of the host's reaction against tumour homografts, that they form antibodies and that plasma cells play an important part in their production.

This work was carried out in the Department of Experimental Pathology and Cancer Research, School of Medicine, Leeds, and I wish to thank Prof. H. N. Green for valuable advice and helpful discussion.

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Anti-allergic Effect of Cortisone

Investigations on the release of histamine and slow-reacting substance from the perfused anaphylactic lung of the guinea pig reveal that cortisone is not inhibitory to the release of either substance. On the other hand, in as low a concentration as 1 in 140,000 the response of the guinea pig gut to histamine and slow-reacting substance is greatly reduced if the cortisone is left in contact with the gut for 1–3 min. Contact for 10 sec. is without significant effect so that assay of the output of histamine and slow-reacting substance is satisfactory on the isolated gut of the guinea pig. This effect, owing to the delay, may be due to intracellular action of cortisone on the effector organ.

This peripheral depressant action, which is not reported as being significant in man from clinical observations, has probably not been observed because of the concentration required to produce it. More soluble cortisone preparations, now available, should show whether this has clinical significance, since larger rapid intravenous dosage is now possible. Lower dosage of cortisone is known to inhibit antibody production and so produce a more gradual reduction in sensitivity.

These experiments will be described in detail elsewhere.

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Nov. 27.