genetic mechanisms may be involved, such as presence of a specific antigen common to F_1 hybrid tumour and sarcoma-89. In this connexion, it should be noted that while the *CAF* tumour was derived from a methylcholanthrene-induced sarcoma as was also sarcoma-89, the *CAF* hybrid tumour originated from a mammary carcinoma of a host injected with methylcholanthrene. Study of possible immunizing ability present in normal tissues of F_1 has been initiated by injecting *C57BL* mice with splenic cells of ($C57BL \times AKR$) F_1 mice prior to inoculation with sarcoma-89. (2) Another possibility is suggested by previous studies in which we found *C57BL* mice to be much more potent antibody producers than *C3H* and *AKR* mice, as judged from levels of antibodies after injection of heterologous red cells[7]. On this basis one might suspect that only *C57BL* mice, but not *AKR* and *C3H* mice, produced sufficient antibody to F_1 hybrid tumours.

This work is now being expanded, with inclusion of additional systems testing immunizing ability of F_1 tumours in other inbred strains in order to elucidate the phenomenon described.

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Presence of 3-Methoxy-4-hydroxyphenylglycol and Metanephrine in Phæochromocytoma Tissue

THE ethereal sulphate of 3-methoxy-4-hydroxyphenylglycol, a metabolite of epinephrine and norepinephrine, has recently been demonstrated in the urine of normal individuals and was noted to be markedly increased in the urine of patients having phæochromocytoma[1]. The free compound has not been previously demonstrated and the possibility that conjugated (nor)metanephrine gives rise to this compound could not be ruled out. We have now been able to demonstrate the presence of free 3-methoxy-4-hydroxyphenylglycol in phæochromocytoma tissue from four different patients, as well as metanephrine in three of these tumours.

About 5 gm. of phæochromocytoma tissue was homogenized with 2 vol. of 0·5 *M* acetate buffer at *p*H 3, saturated with sodium chloride and extracted into 10 vol. of ethyl acetate. The ethyl acetate extract was then washed with salt-saturated acetate buffer at *p*H 7 and evaporated to dryness *in vacuo*. The residue was taken up in 10 ml. acetone, transferred to a 15-ml. centrifuge tube, and the solvent evaporated in a current of nitrogen until the volume was about 4 c.c. After centrifugation, 0·5 ml. of the supernatant fluid was applied to Whatman No. 1 paper and subjected to two-dimensional chromatography using *iso*propanol/5 per cent ammonia (8 : 2) and *n*-butanol/acetic acid/water (8 : 8 : 2) as the solvent systems. A spot was present having the same R_F and colour reactions as authentic 3-methoxy-4-hydroxyphenylglycol[1]. Addition of the authentic substance to the chromatogram increased the intensity of this spot. From the intensity of the colour of the spot, it was estimated that 5–10 μgm. of the glycol per gm. of tissue was present in the tumours.

Another spot, metanephrine[2], was identified in a similar manner and was shown to be different from normetanephrine by separation during chromatography. This substance was demonstrated in three of the four tumours.

The demonstration of unconjugated 3-methoxy-4-hydroxyphenylglycol of biological origin has not been previously reported. This supports the pathway for formation of the sulphate of this metabolite from the free compound, rather than from (nor)metanephrine sulphate. Although normetanephrine has been reported in phæochromocytoma[3], this is the first demonstration of the presence of metanephrine in these tumours.

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A Soluble Protective Antigen of *Trypanosoma brucei*

Trypanosoma brucei in quantities sufficient for antigenic analysis was obtained by harvesting the blood of rats on the third day of infection by the protozoon, when the fulminating parasitæmia is at its height. The trypanosomes were separated from the blood by centrifugation and the resulting mash was preserved by freeze drying. Several batches made in this way were pooled.

Rabbits inoculated with this material produced not only agglutinins for live trypanosomes but also antibodies precipitating with rat serum. These precipitins, a response to traces of serum proteins not removed during the separation of the trypanosomes from the blood, were removed by absorption with normal rat serum. The absorbed antiserum, no longer reacting with normal rat serum, nevertheless precipitated with some component in the serum of infected rats, from which the trypanosomes had been removed by filtration ; suggesting that infected rat serum contained an antigen peculiar to the infected state. This antigen was designated the 'exoantigen' of *T. brucei*.

Antisera to the exoantigen, free of antibodies to normal rat serum, were prepared by inoculating normal rats with trypanosome-free infected rat serum. These antisera not only precipitated exoantigens, but also agglutinated living trypanosomes *in vitro*, suggesting strongly that the exoantigens were liberated from trypanosomes and were not of rat origin. About a million living trypanosomes, which had been

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