electrophoresis[16]. There were two components, of which the first was active with a specific activity of about 2,000 units for AH109A cells. After further disc electrophoresis only one component with a specific activity of about 2,500 units for AH109A cells was obtained. In this state the material behaved as a homogeneous substance on analytical disc electrophoresis[17,18]. Approximately 3 mg of this substance was obtained from 11 g powdered tumour tissue. The substance was a protein, free of nucleic acid with a molecular weight of about 70,000 (estimated by gel filtration on 'Sephadex *G*-200'[19]). It was thermolabile. The substance was active not only for AH109A cells but also MH134 cells and C-1498 cells. The factor was isolated in negligible amounts from normal skin and muscle.

MH134 cells were transplanted in male C3H mice and the growing tumour was excised at 18 days. Following the method described above, the chemotactic factor was isolated; it was active not only for MH134 cells but also AH109A cells and C-1498 cells. A similar factor was isolated from certain human tumour tissues; these were gastric cancer, hepatoma and a metastasis of myeloid leukaemia in kidney. None of the substances isolated from animal or man was active on PMNs *in vivo* or *in vitro*. Conversely leucocyte chemotactic factor (refs. 7, 8, and M. Yoshinaga, K. Y., A. Tashiro and H. H., manuscript in preparation) was ineffective on cancer cells *in vivo* or *in vitro*.

Intradermal injection of the tumour cell chemotactic factor (50–70 units) in rats induced an extravascular emigration of AH109A cells, which had been previously injected intravenously and allowed to proliferate. The emigration of circulating cancer cells from the venules was detected within 24 h and their proliferation in about 72 h. In 7–10 days, cancer cells actively invaded the surrounding tissues such as panniculus carnosus and underlying muscles. The results suggest that some tumours may produce substances that facilitate malignant invasion.

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Yttrium-90 in Gonads of Monkeys containing Strontium-90

THE distribution of ^{91}Y and ^{90}Y in tissues after intravenous injection of ^{91}Y or of $^{90}Y + ^{90}Sr$ has been reviewed[1,2]. This information is not very helpful in understanding the transfer of ^{90}Y from bone into soft tissues because of the overwhelming contribution of the directly injected Y. But if a mixture of ^{90}Sr and ^{90}Y is injected intramuscularly, ^{90}Sr is free to diffuse into the blood stream but the injected ^{90}Y is almost immobilized locally. The ^{90}Sr is largely taken up into the bones and in these circumstances soft tissue content of ^{90}Y may be an index of transport of ^{90}Y formed in bone out of bone and into the blood stream.

A number of young adult monkeys *Cercopithecus* spp. received about 1 mCi/kg of an equilibrium mixture of ^{90}Sr and ^{90}Y by a single intramuscular injection[3]. The radioactivity of the ovary of one animal and of the testis of another was measured by standard methods after they died, 24 and 25 days respectively after injection. Activity measurements were made at a number of time intervals thereafter to allow calculation of the excess or deficiency of ^{90}Y above or below the equilibrium value at the time of death. There was clearly no unexpected concentration of ^{90}Y in the gonads (Table 1).

Table 1. ^{90}SR AND ^{90}Y IN MONKEY GONADS

	Gonad ^{90}Sr μCi/kg fresh weight	Gonad $^{90}Y/^{90}Sr$ (μCi)	Bone ^{90}Sr μCi/kg fresh weight (average)
M 65 Ovary	4	1·0	4,500
M 66 Testis	3	0·9	4,000

In the normal course these results would not have been submitted for publication, but in the present climate of speculation about possible genetic effects of ^{90}Sr a fragment of observed fact may possibly be useful.

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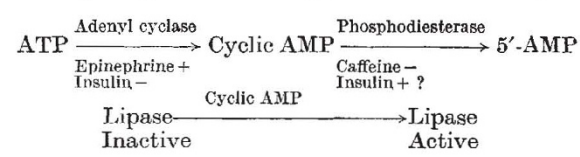
Fatty Acid Mobilization in Obese Mice

PREPARATIONS of adipose tissue from obese hyperglycaemic mice of the C57Bl/6J-ob strain have a reduced sensitivity to the fatty acid mobilizing activity of epinephrine[1]. Epinephrine is believed to exert its lipolytic effect by increasing the production of cyclic AMP[2]. This activates



the "hormone sensitive" lipase[3] resulting in triglyceride breakdown. The cyclic AMP is then destroyed by 3′,5′-nucleotide phosphodiesterase[4] which can be inhibited by caffeine or theophylline. The site at which this pathway is altered in obese mice is not known. Although the concentration of lipase is reduced in adipose tissue from obese