(Canada balsam). Structures containing aldehyde are stained bluish-violet.

The results obtained with *p*-phenylenediamine using sections treated in this way are comparable with those obtained with Schiff's reagent, though greater sensitivity and selectivity is obtained.

As a control on the selectivity of the method, the following reagents for blocking aldehydic groups were used before treatment with *p*-phenylenediamine: (1) 0·2 per cent 4-nitrophenylhydrazine solution in 50 per cent v/v ethanol containing 1 per cent sodium acetate, incubation for 2 hr. at 60° C.; (2) saturated hydroxylamine hydrochloride, ethanolic solution, 2 hr. at 60° C.; (3) 1 per cent aniline solution in 75 per cent v/v ethanol, 2 hr. at 60° C. All these reagents completely prevented the peroxidase oxidation of *p*-phenylenediamine.

V. Scarselli

Istituto Biochimico Italiano,
Via Brembo 65,
Milano.

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PATHOLOGY

Growth of Tumours in Rats fed with 4-Dimethylaminoazobenzene injected with Homogenized Tumour mixed with Freund Adjuvant

The enhancing (or *XYZ*) effect on the growth of tumour homografts in animals pre-treated with non-living donor tissue first described by Flexner and Jobling[1] led to many fruitful immunogenetic investigations in oncology. In order to explain the phenomenon, several working hypotheses have been evolved: the 'blockage' hypothesis was advocated by Medawar and co-workers[2] and Snell[3], 'immunoselection' by Hauschka and co-workers[4] and the hypothesis of some 'physiological' alteration in the tumour cells was argued by Kaliss[5]. According to experiments of Casey and co-workers[6], it is possible to enhance the tumour growth in the strain from which the tumour originated. It is difficult to explain these results on the hypotheses mentioned above.

I have tried to enhance the growth of indigenous tumours. Albino rats of mixed breed were subjected to a carcinogenic diet supplied with 4-dimethylaminoazobenzene. The daily dose of 3·5 mgm. of the drug per rat given during a period of 203 days induced liver carcinoma after a latent period of nearly 120 days after stopping the drug. The incidence of hepatoma was 68 per cent.

In the experiment to be described, 120 rats fed 4-dimethylaminoazobenzene for 203 days were divided at random into five groups and injected with normal liver tissue or liver carcinoma of the same origin, both mixed with Freund's adjuvant. The antigen mixture was heated on a water-bath at 60° C. for 45 min. No palpable liver tumour was present in animals at the time of injection, so it was assumed that the animals were in a pre-cancerous stage. The intracutaneous injections, 8 × 0·2 ml. of antigen mixture, were given along both flanks of the rats simultaneously on the fourth day after stopping the carcinogenic diet. The three intraperitoneal injections (1 ml. each of antigen mixture) were given at intervals of 1 week, the first being given on the same day as intracutaneous injections. On the twenty-second day after stopping the carcinogenic diet, the animals were killed, and diagnosis was by gross inspection and histological examinations of the liver.

Table 1. Incidence of Carcinoma Hepatitis in Pre-cancerous Rats treated with Tumour Tissue and Normal Liver Tissue mixed with Adjuvant

Animals	Treatment				
	Intraperitoneal		Intracutaneous		
	Tumour tissue	Liver tissue	Tumour tissue	Liver tissue	None
Pre-cancerous	12/24	0/24	0/24	0/24	0/24
Normal	0/20		0/18		

The results presented in Table 1 show that intraperitoneal application of the antigen mixture containing cancer tissue was in this instance only suitable for inducing growth promotion of the cancer. It is worth mentioning that inflammatory reaction in the abdominal cavity which occurs after intraperitoneal injection of adjuvant alone or mixed with some tissue was negligible in tumour-bearing rats in comparison with other animals injected intraperitoneally.

As already stressed[7], the enhancement of tumour growth and tumour graft may show a specific difference between cancerous and normal tissues. The effect of enhanced tumour growth produced by the stimulating effect of antibodies might be explained simply by assuming that antibodies are complementary bodies, which act on normal tissue by destroying it and on cancerous tissue on the contrary by enhancing its growth. Cachexia and enhanced tumour growth in cancer-bearing animals could thus be ascribed to a manifestation of the same process in tissues of different physiological and functional state. Further experiments are in progress to investigate the problem.

Nikša Allegretti

Department of Physiology,
Medical Faculty,
University of Zagreb,
and
Department of Biology,
Ruđer Bošković Institute,
Zagreb, Yugoslavia.

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Interaction of Enzymes with Normal and Tumour Cells

Wheat germ lipase (Worthington) has been shown[1] to reduce the adhesion of washed mouse ascites cells to glass. This was considered to be due to the adsorption of the enzyme on to the cell surface. The same lipase preparation has also been found[2] to inhibit the growth of hamster kidney tumour cells in tissue culture at certain concentrations (0·01–0·1 per cent), without inhibiting growth *in vitro* of hamster normal kidney epithelial cells from which the tumour was derived.

An investigation has been made into the cytological effects of lipase on these normal and tumour cells growing in tissue culture, using 'Anoptral' contrast microscopy. At an enzyme concentration of 0·1 per cent, the tumour cells showed extensive vacuolation