ginning of this communication. As seen from Table 1, the two sets of observations (1954 and 1956) are well suited for such a comparison. No significant change could be detected, however, in the average γ -radiation.

The investigation is being continued.

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¹ Sievert, R. M., "Peaceful Uses of Atomic Energy", 13, 187 (1955); *Strahlentherapie*, 99, 185 (1956). Sievert, R. M., and Hultqvist, B. (to be published).

² Burch, P. R. J., and Spiers, F. W., *Nature*, 172, 519 (1953).

The Serum Gamma-Globulin-Level in Malignant Disease

VERY little is known about immunity in relation to malignant disease. In animals there is evidence that resistance develops to transplanted tumours. Lumsden¹ and his co-workers were of the opinion that this resistance was due to the appearance in the serum of certain antibodies. They found that the serum of rats bearing the Jensen rat sarcoma contains a cytotoxin which is greatest in amount in those rats in which tumour regression afterwards occurs. In the human subject the development of humoral resistance to malignant tumours has not been proved conclusively. A number of workers have investigated the serum gamma-globulin-level in patients with malignant disease. Their results can be taken as a measure of the antibody-level since most antibodies are found in association with the gamma-globulin fraction of the serum protein. Petermann and Hogness² found that the serum gamma-globulin-level, as estimated by electrophoresis at pH 8.6, was normal in twenty-five patients with carcinoma of the stomach. Miller and Erf³, using a similar method, found that the serum gamma-globulin was raised in twenty-four patients with carcinoma of the gastro-intestinal tract or breast.

In view of the difference between these results it was decided to perform further electrophoretic investigations on the serum gamma-globulin-level of patients with malignant disease. It was hoped by this means to determine with some degree of certainty whether the antibody-level of the serum changes in the presence of malignant neoplasms.

Twenty-two female patients from the Dartford Group of Hospitals, Kent, suffering from malignant disease, which had been verified by histological examination, were used for the present investigation. The patients were chosen at random and their ages varied from thirty-one to seventy years. The majority of the tumours were situated in the genital or intestinal tracts and were at different stages of development, some being localized while others had metastasized widely. Blood samples were taken from each patient on admission, and only the blood from those patients having normal temperatures, total and differential leucocyte counts, erythrocyte counts and haemoglobin-levels were used. Blood samples were also taken from twenty healthy females of the same age-group to serve as controls.

The gamma-globulin-level of each of the sera was estimated by first separating the gamma-globulin

fraction by paper electrophoresis at pH 8.5 using a veronal buffer. The filter-paper strips were then treated with bromphenol blue and the optical density of the protein dye complex was measured by means of a photoelectric cell. In this manner the percentage of gamma-globulin in relation to the total protein in the serum could be assessed.

No significant difference was found between the serum gamma-globulin-levels of the patients with malignant disease and those of the healthy controls. The gamma-globulin-level in each group averaged approximately 20 per cent of the total serum protein. These findings provide further evidence that the development of malignant disease in man is not associated with an increase in the antibody-level of the serum.

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¹ Lumsden, T., and Phelps, H. J., *Amer. J. Cancer*, 29, 517 (1937).

² Petermann, M. L., and Hogness, K. R., *Cancer*, 1, 100 (1948).

³ Miller, B. J., and Erf, L., *Surg. Gynaec. and Obst.*, 102, 487 (1956).

The Expected Blood-Group Antibody, anti-Lu^b

THE Lutheran blood group system was first described by Callender and Race¹ in 1946: an antibody, afterwards named anti-Lu^a, was found to react with the red cells of approximately 8 per cent of the English population. The antigen with which the antibody reacts is called Lu^a and can be recognized in the red cells of heterozygous as well as homozygous individuals. The corresponding gene is called *Lu^a* and an allele *Lu^b* is postulated. The antigen Lu^b has been recognizable so far only as an absence of Lu^a; thus persons of the phenotype Lu(a-) are presumed to be of the genotype *Lu^bLu^b*.

The results of testing large numbers of English blood samples with various anti-Lu^a sera have been brought together by Race and Sanger², and the following gene and genotype frequencies for the Lutheran system have been calculated.

Genes		Genotypes	
<i>Lu^a</i>	0.0390	<i>Lu^aLu^a</i>	0.0015
		<i>Lu^aLu^b</i>	0.0750
<i>Lu^b</i>	0.9610	<i>Lu^bLu^b</i>	0.9235

An antibody which gives the reactions expected of anti-Lu^b has now been found in the serum of a woman, Mrs. R., who has never been transfused and who has given birth to three healthy children.

In a search for compatible blood for Mrs. R. it was found that her serum agglutinated saline suspensions of the red cells of each of fifteen blood samples. The identity of the antibody was suspected when it was found that Mrs. R.'s own cells reacted very strongly with an anti-Lu^a serum and that her serum reacted less well with Lu(a+) cells than with Lu(a-) cells.

Mrs. R.'s phenotype is as follows: O, MNSs, P+, CcDEe, Lu(a+), K+ k+, Le(a- b+), Fy(a-), Jk(a+ b+). The cells react with an anti-H serum. Dr. R. R. Race kindly tested the cells with anti-U, anti-Vel, anti-Yt^a and anti-Tj^a; in each case a positive reaction was obtained.