

rosettes are higher than those reported after chemotherapy by Catovsky and his colleagues.³ On the other hand, since splenic irradiation induced both decrease of delta determinants and appearance of the μ - γ association, some changes (maturation?) within the cells of the leukæmic clone might have occurred as well.

This study has been supported by C.N.R., Rome, contract no. 74.00411.00.

Blood Research Foundation Centre,
Tortona, Italy.

Institute G. Gaslini, Genova, Italy.

Laboratory of Biological Technology,
National Research Council,
Rome, Italy.

GIOVANNI ASTALDI.

GIULIA C. B. ASTALDI.*

P. PALEAN IVETTORI

ALBERTO ASTALDI.*

CIRCULATING IgE LEVELS IN PATIENTS WITH CANCER

SIR,—We would like to confirm the recent findings of Dr McLaughlan and Dr Stanworth (Jan. 11, p. 64) that patients with advanced malignancies have normal circulating IgE levels as determined by radioimmunoassay. Thus we have determined the IgE levels in serum samples from more than 400 such patients, using both a solid-phase and a double-antibody radioimmunoassay. In no instance have we found significantly abnormal values—other than when an elevated value could be attributed to an undiagnosed coexistent allergic disorder.

These findings are in direct contrast to our previous report⁴ that some 50% of untreated patients with bronchial or other neoplasms apparently had markedly elevated IgE levels as determined by a solid-phase radioimmunoassay but normal or low values as measured by a single radial immunodiffusion technique. At that time we attributed this discrepancy to the presence, in some patients with advanced malignant disease, of a circulating inhibitor which bound IgE and thereby caused spuriously high results using the radioimmunosorbent assay. This explanation was compatible with the finding of antibodies directed against IgE in a substantial number of patients with allergic disease.⁵

We have reported⁶ that the original findings could not be substantiated, and wish to explain that the long delay in publishing a rebuttal of our previous paper has been due to the time taken to develop a more sensitive and specific radioimmunoassay for IgE and establish a normal range⁷; to obtain and assay a large number of further samples from patients with neoplasms; and, in particular, to attempt to explain the previous results.

Fortunately, some of the initial samples were still available, which enabled the presence of an IgE inhibitor to be repeatedly confirmed both by ourselves and by two other internationally recognised groups. These sera were shown, by a variety of procedures, to contain antibodies of the IgG class, directed against IgE, which inhibited the assay of exogenous IgE by a modified Mancini technique and a double-antibody radioimmunoassay and caused excessive histamine release from sensitised leucocytes.⁸

The paradox between our earlier findings and those for all the additional patients studied led to the demonstration,

using specific antisera, that the original samples contained rabbit IgG and that it was rabbit and not human antibodies which bound exogenous IgE. Thus, it can only be concluded that many of the original samples from patients with cancer had been contaminated by rabbit anti-IgE sera.

Benenden Chest Hospital,
Benenden, Kent.

Department of Chemical
Pathology,
St. Bartholomew's Hospital,
London EC1.

M. HOURI.

J. LANDON

T. G. MERRETT.

UNSUITABILITY OF BLOOD-DONORS FOR POPULATION GENETICS

SIR,—Among parents of children with phenylketonuria in Norway there was a significant excess of rhesus-negative individuals, which was especially pronounced among the mothers.¹

The proportion of rhesus-negative individuals among new male and female blood-donors consecutively registered at the Oslo City Hospital Blood Transfusion Centre was therefore investigated, excluding individuals with knowledge of their blood-groups. There was a highly significant ($P < 0.001$) excess of rhesus-negative individuals among the

TABLE I—PROPORTION OF RHESUS-NEGATIVE INDIVIDUALS AMONG NEW OSLO BLOOD-DONORS ACCORDING TO SEX AND YEAR OF REGISTRATION

	Males		Females	
	Rhesus negative	Total	Rhesus negative	Total
1952-1954	14.0%	2132	17.7%	232
1961-1966	13.9%	4724	18.3%	3505
1967-1971	14.8%	2723	18.8%	2607
1972-1974	15.1%	2774	20.9%	2114
1952-1974	14.4%	12353	19.1%	8458

From 1952 to 1974 16.3% of all donors (i.e., 3392 out of 20,811) were rhesus negative.

TABLE II—PROPORTION OF RHESUS-NEGATIVE INDIVIDUALS FROM ALL OVER NORWAY INVOLVED IN PATERNITY CASES (1966-1973)

Male		Female		All	
Total no.	% rhesus negative	Total no.	% rhesus negative	Total no.	% rhesus negative
3007	14.6	3009	14.5	6016	14.6 (i.e., 876)

female compared with the male donors (table I). Over the period investigated the proportion of rhesus-negative females increased considerably as knowledge of the particular need for rhesus donors grew. A similar self-selection among the male donors only became apparent in the past 2 years. The proportion of rhesus-negative individuals among donors exceeds that among paternity cases (table II) ($\chi^2=10.5$, 1 d.f. 0.001, $P < 0.01$). A similar significant heterogeneity is also apparent in Sweden, where the proportion of rhesus-negative individuals was over 16% in 73,581 donors as compared with 14.7% in 8488 paternity cases² ($\chi^2=11$, 1 d.f., $P < 0.001$). The proportion in female donors was not known, but a similar self-selection is probably operating among Swedish and Norwegian female blood-donors. A higher proportion of rhesus-negative individuals was found among new female donors than among new male donors in Britain too.³ Perhaps this "female type of self-selec-

* Present address: Central Laboratory of the Netherlands Red Cross Blood Transfusion Service, Amsterdam, The Netherlands.

3. Catovsky, D., Miliani, E., Okos, A., Galton, D. A. G. *Lancet*, 1974, ii, 751.
4. Jacobs, D., Houri, M., Landon, J., Merrett, T. G. *Lancet*, 1972, ii, 1059.
5. Williams, R. C., Griffiths, R. W., Emmens, J. D., Field, R. C. *J. clin. Invest.* 1972, **51**, 955.
6. Merrett, T. G. LKB Radioimmunoassay Symposium, Magdalen College, Oxford, 1974. Oxford (in the press).
7. Nye, L., Merrett, T. G., Landon, J., White, R. G. *Clin. Allergy*, 1974, **4**, 511.
8. Assem, E. S. K., McAllen, M. K. *Br. med. J.* 1970, ii, 504.

1. Saugstad, L. F. *Lancet*, 1973, i, 1255.
2. Beckman, L. *Hereditas*, 1959, **45**, 1.
3. Kopeck, A. The Distribution of Blood-groups in the United Kingdom, p. 134. London, 1970.