(3) *Transformation of* ΔT *to* ΔV *and* ΔH

Using equations (5), (6), (7) and (8), we deduce the following relations among ΔV, ΔH, ΔT and ΔT_H :

$$\Delta V = (\Delta T \sin I + \Delta T_H \cos I)/b \quad (11)$$

$$\Delta H = \sin \alpha\, (\Delta T \cos I - \Delta T_H \sin I)/b. \quad (12)$$

Hence, from equations (11) and (12), the vertical and the horizontal components can be computed from the total component and its Hilbert transform.

The relations given in equations (8), (9) and (10) are fundamental to all the two-dimensional bodies. Bhattacharyya and Leu[1] have shown that the relations given in equations (5) and (6) are true for all two-dimensional bodies. Therefore, the transformations can be carried out using the above relations even in the case of other two-dimensional bodies.

Computation of Hilbert Transform

The practical approach of obtaining the Hilbert Transform $F_H(x)$ of any function $F(x)$ using the Fourier Transform $F(\omega)$ of $F(x)$ has been suggested by Green[5] which involves the following procedure.

The Fourier Transform $F(\omega)$ can be broken up into a series of sine and cosine terms of known amplitude. Let $a_0, a_1, a_2 \ldots a_n$ be the amplitudes of the cosine terms and $b_1, b_2, b_3 \ldots b_n$ be the amplitudes of the sine terms. Now produce a waveform made of cosine terms with amplitudes $0, b_1, b_2, b_3, \ldots \ldots b_n$ and sine terms of amplitudes $-a_1, -a_2, -a_3, \ldots -a_n$; then, the synthesized waveform is the Hilbert transform of the original waveform. It amounts to an interchange of the amplitude coefficients of the sine and cosine terms of the Fourier transform with a change in sign of the sine terms. The following three steps are involved in the computation.

(1) Calculate the Fourier transform of the profile,
(2) Set the negative frequencies to zero,
(3) Take the inverse Fourier transform of the modified Fourier transform.

The resulting output gives the Hilbert transform.

PRACTICAL EXAMPLE

The relations given in equations (8) and (9) are used to transform a vertical field magnetic anomaly (ΔV) over Pima copper mine in Arizona, taken from figure 10 of Gay's paper[4]. The ΔV anomaly has been transformed into the total (ΔT) and the horizontal (ΔH) anomalies using the above relations and are shown in Fig. 2 along with the original ΔV anomaly. Gay[4] interpreted the anomaly comparing it with the standard curves presented by him. The transformed ΔT and ΔH anomaly curves are found to fit well with the corresponding standard theoretical curves expected over the same body.

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A STUDY ON THE LYSOZYME PATTERN IN MURINE LEUKAEMIA AND LYMPHOMAS

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LYSOZYME, a low molecular weight basic protein, is a non-specific mucolytic enzyme which is widely distributed in nature—both in the plant and in the animal kingdoms. Its presence in the majority of animal tissues and fluids has been demonstrated as early as 1922 by Fleming[1]. Recently, lysozyme determination in blood and urine has proved to be a valuable diagnostic tool in tubular kidney disease[2], in sarcoidosis[3], certain tumors of the central nervous system[4] and specific types of leukaemias[5,6] in man. Similar studies on animals, however, are relatively few.

In an attempt to gain insight into the biological role of lysozyme in malignant neoplasia, determination of serum levels of this enzyme in a variety of tumor bearing mice was undertaken, and the changes that occurred during the development of the tumors noted.

The present communication represents a part of the 'estigation and is concerned with four types of rine leukaemia and lymphomas.

Schwartz and Molony virus induced lymphoblastic lymphatic leukaemias are being maintained in Swiss and Strain 'A' mice respectively by serial intraperitoneal injections of the cell free extracts of organs, infiltrated with leukaemic cells (spleen, lymph nodes, mesenteric gland, thymus, liver) into 24–48 h old mice.

Daltons lymphoma, which originated as a spontaneous tumor of thymus in DBA/2 mouse[8] is now being maintained in the ascitic form in Swiss mice by serial transplantation of tumor cells intraperitoneally in 5-6 week old mice. Well developed ascitic tumor containing large number of neoplastic mono-nuclear cells is produced around 10th to 14th day of tumor transplantation.

L1210, a lymphoid leukaemia, induced initially by 20-methylcholanthrene[9], is now being maintained in the ascitic form by serial passage of ascitic cells in 4–5 week old DBA/2 mice. This is a very fast growing tumor and kills the host in about a week.

In the present study, in all cases, animals bearing well developed tumors were selected. Normal mice of the same strain and age group were taken as control.

The lysozyme concentration in the serum was determined by the lysoplate method[5], using human lysozyme, as standard (Figs. 1 and 2). Micrococcus lysodeikticus uniformly suspended in 1% agar in phosphate buffer (pH 6·3) was poured in petridishes and allowed to solidify. Several circular wells were cut in the agar and filled with serum samples and standard lysozyme solutions. The diameter of the lytic zones of standard solutions were measured and plotted against the log of lysozyme concentration and test values determined from the resultant graphs.

In all the four different tumor strains studied, there was a general enhancement of lysozyme concentrartion, as compared to the controls. The highest serum lysozyme level was observed in case of Schwartz virus induced lymphoblastic lymphatic leukaemia. Next in order of concentration was Dalton's lymphoma (ascitic). However, in Molony's leukaemia and Dalton's lymphoma a wide range of lysozyme concentration was observed.

Our observation on the increased serum lysozyme levels, in four types of leukaemic tumors of different origin, seems interesting in the light of prevailing reports in literature, most of which deal with monocytic or granulocytic leukaemias[5,6,10,11]. The present study reveals that, at least in mice, the presence of other malignant tumors may also be reflected in the serum in the form of increased lysozyme concentration.

Failure to demonstrate any significant lysozyme activity in neoplastic cells indicated[12] that the phenomenon of elevated lysozyme level was related to a stimulation of the reticuloendothelial system of the host in response to the growth and multiplication of injected leukaemic cells. Other investigators have also reported the increased serum lysozyme concentration due to chronic infection[5] and due to tumor cell transplantation[13]. According to Bordin and Young[14] macrophages are the primary sources of the released lysozyme in tumor bearing host. It has been further demonstrated that the number and functional activity of granulocytes and macrophages increased during the host's reaction to neoplasia resulting in the realease of lysosomal enzymes[12].

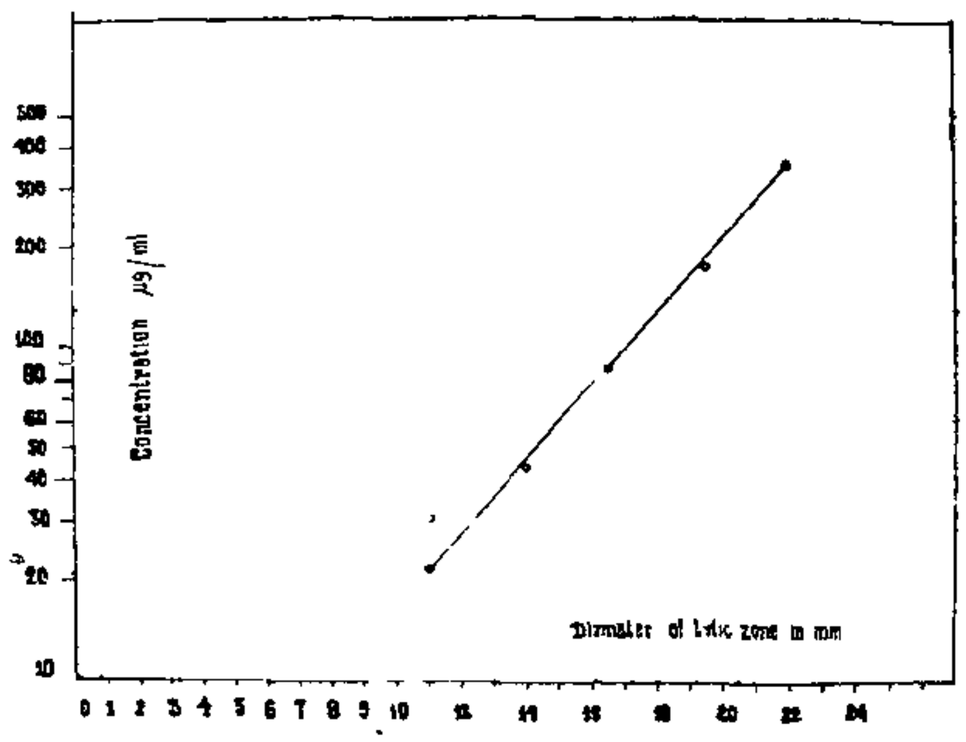

FIG. 1. Graph illustrating relationship between diameter of lytic zone and concentration of human lysozyme (given in logarithmic scale) used as a reference standard for determining test values.

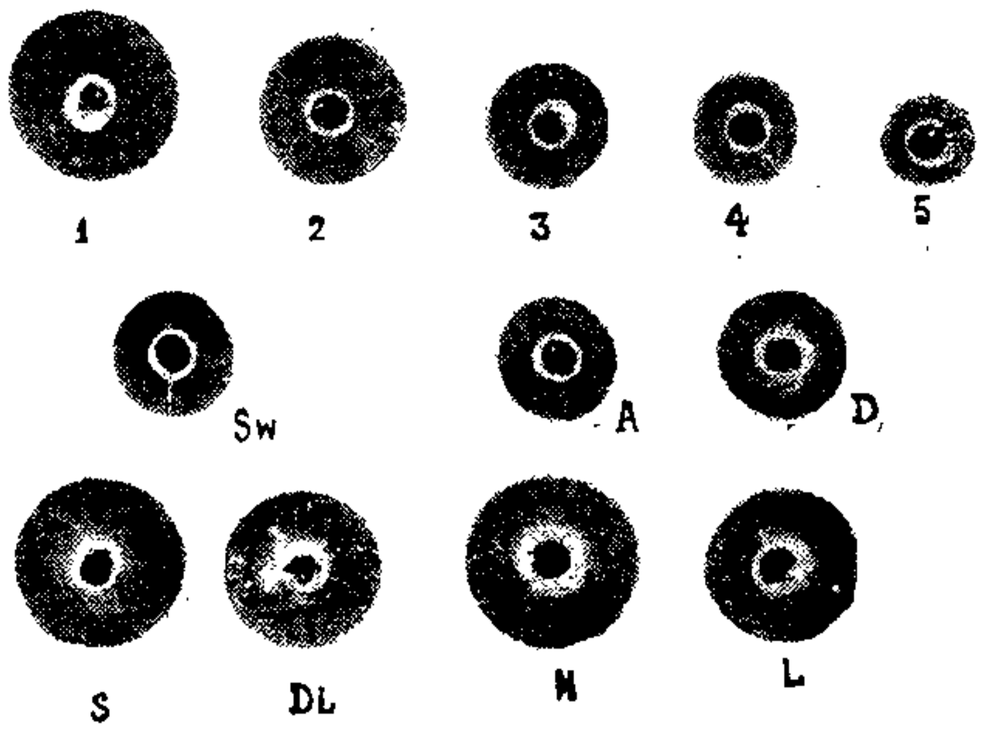

FIG. 2. Lysoplate analysis of serum samples using human lysozyme standard. 1–5: Lytic zones produced by human lysozyme at different concentrations. Sw., A and D.: Lytic zones produced by serum samples of normal Swiss, Strain A and DBA mice. S, DL, M and L: Same as above for Schwartz, Dalton, Molony and L1210 leukaemic mice.

The increased levels of lysozyme in serum, observed in the present study, perhaps reflects some host-reaction towards the neoplastic cell-proliferation or towards bacterial or virus like agents that may be associated with the tumor cells. Since lymphocytes, besides red cells and platelets, do not contain any lysozyme, the elevation of serum lysozyme level may perhaps be a reflection of the breakdown of a large number of granulocytes with high intracellular lysozyme content.

TABLE I

Lysozyme concentrations in serums of normal and tumor bearing mice determined by Lysoplate assay using human Lysozyme standard

Diagnosis	No. of Mice	Concentration μg/ml Mean ± S.D.	Minimal-Maximal values
Normal strain A mice	10	80·6± 7·8	68–93
Normal swiss mice	10	82·6±12·6	66–100
Normal DBA mice	5	89·6±12·0	84–100
Molony lympho-blastic leukaemia	10	153·5±67·7	110–338
Schwartz lympho-blastic leukaemia	8	278·5±49·8	210–349
Daltons lymphoma (Ascitic)	7	263·1±99·3	132–420
L1210–Lymphoid leukaemia (Ascitic)	5	198 ±55·2	120–270

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OBITUARY

We regret to announce with deep sorrow, the sad demise of Lady Lokasundari Raman, wife of the Nobel Laureate late Sir C. V. Raman, at the age of eighty-six on Thursday, the 22nd May 1980, at her residence 'Panchavati' in Malleswaram, Bangalore (India). Lady Raman was a very popular social worker who endeared herself with all those that came into contact with her. She was a 'friend, philosopher and guide' not only to Sir C. V. Raman but also to a host of individuals and organisations in this country.

MAY HER NOBLE SOUL REST IN PEACE

EDITOR