latory central mechanism the receptive sites of which belong to the α class.

E. Fischer
M. López Amalfará *

Laboratorio de Investigaciones
Psicofarmacológicas,
Hospital Nacional de
Neuropsiquiatría,
Vieytes 451, Buenos Aires.

* Graduate Fellow of the Universidad Nacional de Buenos Aires.

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PATHOLOGY

Cancer Subjects and Abnormal Cell Division

We reported recently that the proportions of aneuploid cells in cultures of human blood cells increased approximately in proportion to the age of the subject[1]. Some of the cells observed to have chromosome numbers less than normal are likely to have been artefacts. There was, however, evidence that others were genuinely abnormal, and it was concluded that the increase of abnormal cells with age indicates either a real increase in the number of aneuploid cells in the body or an increased liability to abnormal cell division. In either case it was possible that the finding was related to the increase in incidence that occurs with age in most types of cancer.

We have, therefore, examined cells cultured from the blood of cancer patients, exclusive of those with primary reticulo-endothelial tumours, to see if the proportion of aneuploid cells differed from that observed in persons without cancer.

Data obtained from 38 cancer patients, between 26 and 87 years of age, are summarized in Table 1. Between 29 and 50 cells were examined for each patient and altogether chromosome counts were made on 1,281 cells. From the regression formulæ previously reported for persons with a normal karyotype and the age of the patients, it was possible to calculate the numbers of cells which would have been expected to show abnormal chromosome counts. The observed and expected numbers are shown in Table 1 for four age-groups (25–44 years, 45–59 years, 60–69 years and 70 years and over). From these results it is clear that there is no significant difference between persons with and without cancer, and the closeness of the observed and expected numbers at all ages combined makes it extremely unlikely that any important difference would be discovered by examining more patients.

The results do not, in our opinion, weigh against the suggestion that aneuploidy predisposes to the development of cancer. They show, however, that cancer subjects do not have an unusual tendency to abnormal cell division in all tissues.

Karin E. Buckton
Patricia A. Jacobs
W. M. Court Brown
R. Doll

Medical Research Council
Clinical Effects of Radiation Research Unit,
Western General Hospital,
Edinburgh 6.

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Sub-cellular Transmissible Agent from Ehrlich Carcinoma Cells producing Ascites Tumours in Mice

For the past several years, Ehrlich ascites tumour cells have been used in this Laboratory in work on the anti-tumour activity of fatty acids[1–3]. Serial transplantation of the stock tumour in Swiss mice was carried out at weekly intervals using an inoculum of 15×10^6 washed ascites cells, a number which effected a maximum rate of cell proliferation[4]. Under these conditions, the proportion of tumour cells in the ascitic fluid reached 60–70 per cent within 5–6 days after inoculation. Afterwards, however, the percentage of tumour cells declined steadily and dropped to only 10–20 per cent shortly before death of the mice at 10–14 days. This observation suggested that the ascitic fluid might be exerting an oncolytic effect on the tumour cells[5,6], and experiments were carried out to test the activity of the fluid after removal of the tumour cells by high-speed centrifugation. These experiments, rather than revealing an oncolytic factor, indicated the presence of a transmissible agent producing ascites tumours.

Ascites was collected by intraperitoneal puncture from Swiss mice bearing the Ehrlich ascites tumour. The ascitic fluid was centrifuged in an international clinical centrifuge at 2,500 r.p.m. for 10 min. to sediment the majority of the tumour cells. The resulting supernatant was recentrifuged for 1 hr. at the same speed. This second supernatant was carefully removed and centrifuged for a third time in a Spinco ultracentrifuge, model *L*, at various speeds, using rotor No. 40. After removal of the top white layer, the underlying pale yellow fluid was collected and injected intraperitoneally in 0·5 ml. amounts into

Table 1. Chromosome Count Distributions in Patients with Cancer (excluding Primary Reticulo-Endothelial Tumours)*

Age (years)	No. of subjects	Mean age weighted for number of cells examined	No. of modal cells	No. of hypermodal cells		No. of hypomodal cells	
				Expected	Observed	Expected	Observed
25–44	9	33·05	311	4·64	3	17·31	17
45–59	10	52·69	338	7·30	5	23·19	17
60–69	10	64·50	269	6·89	9	20·55	23
70+	9	77·00	258	7·71	9	21·84	22
All ages	38	55·88	1,176	26·54	26	82·89	79

* With the exception of two cases of papillomatosis of the bladder, no case had been subjected to any therapeutic procedure before examination. The two exceptions had been treated by fulguration.
The primary sites were: breast (10), testis (7), bronchus (6), bladder (5), salivary gland (2), cervix uteri (1), larynx (1), colon (1), œsophagus (1), skin (1), prostate (1), ischium (1), soft tissue of leg (1).