TABLE I.

	No. of Animals	Absorption per hr. per 100 gm. rat Average mg.	Glycogen	
			Liver Average %	Entire Body Except Liver Average %
1. Fasting Controls	11		0.105 (0.074-0.170)	0.051 (0.042-0.075)
2. Xylose, 1 hr.	6	29.3 (23-34.1)*	0.157 (0.092-0.262)	0.053 (0.045-0.061)
3. Xylose, 2 hrs.	6	39.9 (24-54)	0.129 (0.094-0.186)	0.061 (0.042-0.080)
4. Xylose, 3 "	12	46.7 (32-74)	0.134 (0.060-0.185)	0.059 (0.039-0.073)
5. Glucose, 3 hrs.	8	163.9 (144.4-205.4)	1.916 (1.149-2.997)	0.153 (0.095-0.251)
6. Glucose, 3 " fed at level of xylose absorption	11	47.4 (41.8-54.1)	0.604 (0.459-0.725)	0.088 (0.075-0.101)

* The figures in parenthesis indicate the ranges observed in the individual experiments.

hour per 100 gm. of rat) over a period of 3 hours was similar to the amount of xylose absorbed in a like period (46.7 mg. per hour per 100 gm. of rat). A significant glycogen formation in the livers of this group was also observed, the average figure being 4 to 5 times greater than that of the control fasting rats or the rats receiving xylose. This would seem to indicate that xylose *in the amounts actually absorbed* should cause a significant deposition of glycogen if glycogen formation from it occurred readily. We must therefore conclude that xylose, under our experimental conditions, is not readily available for the formation of glycogen.

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Nature of the Agent Transmitting Leucosis of the Fowl.*

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The studies of Ellerman and Bang,[1] Furth[2] have shown that the agent transmitting leucosis of fowls passes bacteria-tight Berkefeld

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† With the assistance of Charles Breedis.

[1] Ellermann, V., The Leucosis of Fowls and Leucemia Problems, London, 1921.

[2] Furth, J., *J. Exp. Med.*, 1931, **53**.

filters. In most instances the bulk of the transmissible agent was easily removed from the blood by a brief centrifugalization, suggesting a relation of this agent to cells such as has been observed with some viruses. Moreover the opinion of some that the transmission of Rous sarcoma is bound to cells[3] or cell-fragments that pass silicous filters[4] has induced me to investigate the relation of the principle transmitting leucosis to cells.

When diminishing amounts of whole blood or blood cells of leucemic fowls were injected into healthy fowls leucosis was usually not produced with amounts less than about 0.0002 cc. (in terms of the original whole blood) although this quantity of blood contained from 5,000 to 50,000 leucocytes. Therefore, should transmission depend on the presence of white cells a very large number of them would be required to produce leucosis.

Undiluted plasma, after recentrifugalization for about 8 minutes at about 2000 revolutions per minute, when placed in the counting chamber appeared cell-free. Though the plasma was in all but one experiment less active than the cell suspension in producing leucosis, amounts as small as those actually contained in the counting chambers (0.002 to 0.01 cc.) were in several experiments sufficient to transmit leucosis.

No cells could be shown in Berkefeld V and N filtrates of plasma. Even a very coarse non-bacteria tight filter resisting only an air pressure of about 190 mm. appeared to retain leucocytes completely. Since most of the leucocytes can be rapidly thrown to the bottom of a tube by spinning, absence of cells in the bottom layer of such material is sufficient evidence that the entire filtrate is either cell-free or that it contains cells in small number, insufficient to transmit lesions.

Very recently Jármai[5] observed that Zsigmondy-Bachman membranes with an estimated pore-size of about 20 to 100 millimicrons did not retain the transmissible agent of leucosis of fowls but that finer membranes retained this agent and proteins as well. The observations of Jármai as well as our own permit the conclusion that leucosis of the fowl may be transmitted by cell-free material and that its causative agent is filterable.

Filtrable agents of tumors are apparently distinguishable from those causing infectious diseases. The behavior of the filtrable agent of the leucosis of fowls seems similar to that of filtrable tumors. This separation is suggested by certain features that are

[3] Nakahara, W., *Jap. J. Exp. Med.*, 1928, **7**, 101.

[4] *Cf.* Teutschlaender, O., *Z. f. Krebsf.*, 1923, **20**, 43.

[5] Jármai, K., *Arch. f. dissensch. und prakt. Tierheilkunde*, 1930, **62**, 113.

probably common to the filtrable agents of tumors and of leucosis although they have not been satisfactorily investigated in this connection. These are as follows:

Resistance of certain individual animals to these transmissible agents is not based on immunological principles but appears to be governed chiefly by hereditary factors. Ellermann observed that fowls that resisted one inoculation might occasionally succumb to reinoculation. We have found that the blood of a fowl that had recovered from leucosis did not exert any protective action when inoculated simultaneously with leucemic blood into 5 fowls, for all of them developed leucosis about the same time as the corresponding controls.

Some fowls resist inoculation although they are given several hundred times the amount necessary to cause leucosis in susceptible individuals. In one passage, for example, 4 fowls were inoculated with 0.2 cc., 4 with 0.001 cc. and 4 with 0.00005 cc. of plasma (not cell-free). One of the fowls inoculated with 0.2 cc. resisted the disease although 2 of those receiving 0.001 cc. and one of those receiving 0.00005 cc. developed leucosis.

Filtrable tumors of the fowl unlike other filtrable virus diseases of the fowl could not be transmitted to other species of birds. The transmissible agent of filtrable tumors appears to be in large part attached to cells or to cell fragments.

These properties as well as the character of the disease produced (neoplastic growth) seem to justify a separation of the filtrable agents of tumors within the apparently heterogenous group of filtrable viruses and suggest the possibility that the agent of filtrable tumors is not a virus in the ordinary sense.

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Blood Lactic Acid and the Coronary Circulation.*

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Increasing information on the distribution of lactic acid in the body tissues has suggested that it plays a more complex rôle than has generally been suspected. Evidence was produced[1] to show

* Aided by a grant from the Committee on Scientific Research of the American Medical Association.

[1] McGinty, D. A., *Am. J. Physiol.*, 1929, **88**, 312.