

that in the combustion of glucose lactic acid may arise from the cleavage of the sugar molecule. On account of this significant place held by lactic acid it appears of interest to investigate the relation it may hold to sugar production in human diabetes. Only cases of diabetes of considerable severity are suitable for the experiment, since in the milder degrees of the human disorder relatively large amounts of carbohydrate may be burned or stored and the sugar output is apt to fluctuate. In a patient it was ascertained that with a uniform diet for three days a fairly constant glucose excretion could be depended upon and lactic acid as the sodium salt was given on the second day of the experimental period with the result that there was a rise in sugar excretion shown by the following abstract from the protocol.

	Amount urine.	Sugar, grams.	Total N.	NH ₃ N.	Acetone and diacetic.	B-oxo butyric.
Vegetable diet. . . .	1,810	2.89	3.62	1.37	.78	0.79
Oatmeal diet.	1,450	2.80	4.80	1.22	.59	1.41
Meat diet.	2,300	6.56	11.46	1.42	.76	1.77
15 gm. Na lactate.	2,010	19.90	14.06	1.56	.82	1.49
	2,050	2.91	14.92	0.71	.86	1.97

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On some vaccinia blood pressor substances in rabbits.

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These notes constitute a continuation of our observations on some blood pressor substances in experimental immunity, reported at the last meeting of this society, and apparently tend to confirm the same. We have endeavored to ascertain when the pressor and depressor substances appeared in the blood serum of living rabbits after they had been successfully infected with vaccinia virus. The height of the production of the vesicles from which the virus was collected was reached on the fifth day. They then rapidly healed and the rabbits appeared outwardly to be in good health.

Observations were also made to ascertain the effect of aging on the presence of the depressor substance.

TABLE I.
Showing when the pressor and depressor substances appear.

Date of experiment.	Time after inoculation.	Drawn from clot.	Amount of injection.	Reaction.
March 8, 1913.	48 hours. No vesicles (bled 3-7-13).	Within 12 hours of bleeding.	7 c.c.	A good depression.
March 8, 1913.	48 hours. No vesicles (bled 3-7-13).	Within 12 hours of bleeding.	8 c.c.	A very marked depression.
March 28, 1913.	5 days ¹ (bled 3-10-13 quite red).	Within 48 hours of bleeding.	2½ c.c.	Decided rise in pressure, followed by clotting.
March 28, 1913.	5 days ¹ (bled 3-10-13 quite red).	Within 48 hours of bleeding.	5 c.c.	A good rise in pressure.
March 28, 1913.	5 days ¹ (bled 3-10-13 quite red).	Within 48 hours of bleeding.	5 c.c.	A good rise in pressure.
March 28, 1913.	8 days (bled 3-22-13).	Within 4 days of bleeding.	8 c.c.	Fine marked double depression.
March 28, 1913.	10 days (bled 3-15-13), quite red.	Within 4 days of bleeding.	2½ c.c.	Slight rise followed by a slight fall.
March 28, 1913.	12 days (bled 3-17-13).	Within 4 days of bleeding.	4 c.c.	Slight rise followed by a slight fall.
March 28, 1913.	17 days (bled 3-22-13).	Within 4 days of bleeding.	5 c.c.	Marked depression.
March 28, 1913.	30 days (bled 3-22-13).	Within 4 days of bleeding.	5 c.c.	Slight rise followed by a slight fall in pressure.

¹ This 5 days result may also have changed to pressor, during the 16 days the serum was kept in the ice-box.

TABLE II.
Showing the effect of aging on the presence of the pressor substances.

Date of experiment.	Time after inoculation.	Drawn from clot.	Amount of injection.	Reaction.
March 8, 1913.	48 hours. No vesicles. Bled 3-7-13.	Within 12 hours of bleeding.	7 c.c.	Good depression.
March 8, 1913.	48 hours. No vesicles. Bled 3-7-13.	Within 12 hours of bleeding.	8 c.c.	Very marked depression.
March 28, 1913.	48 hours. No vesicles. Bled 3-7-13.	Within 12 hours of bleeding.	1 c.c.	No reaction.
March 28, 1913.	48 hours. No vesicles. Bled 3-7-13.	Within 12 hours of bleeding.	6 c.c.	Decided rise. ¹
March 28, 1913.	8 days. Bled 3-22-13.	Within 4 days of bleeding.	8 c.c.	Fine, marked double depression.
April 4, 1913.	8 days. Bled 3-22-13.	Within 4 days of bleeding.	8 c.c.	Slight rise followed by slight fall in pressure. ²
March 28, 1913.	17 days. Bled 3-22-13.	Within 4 days of bleeding.	5 c.c.	Marked depression.
April 4, 1913.	17 days. Bled 3-22-13.	Within 4 days of bleeding.	8 c.c.	Quite a decided rise, followed by an equal fall in pressure. ³
March 28, 1913.	30 days. Bled 3-22-13.	Within 4 days of bleeding.	5 c.c.	Slight rise followed by slight fall in pressure.
April 4, 1913.	30 days. Bled 3-22-13.	Within 4 days of bleeding.	8 c.c.	Marked rise in pressure.

¹ *I. e.*, after standing in the ice-box 20 days the depressor disappeared and pressor appeared.

² *I. e.*, after standing in the ice-box 13 days the depressor was partially eliminated.

³ *I. e.*, same result as the preceding.

Summary.—1. Depressor and pressor substances arise after vaccinia infection in the blood-serum of rabbits. 2. Aging tends to eliminate the depressor substance and a pressor substance then comes in evidence.

Note.—A serum obtained from a rabbit after streptococcus infection, which had 12 months previously given a profound depressor reaction was also tested (3-18-13) and was found to give no reaction. 8 c.c. of a saline extraction of the adrenals of a 30 day vaccinia rabbit, gave (3-28-13) no reaction. This extraction was made in 20 c.c. of physiological saline solution and was kept 6 days in the ice-box. 7 c.c. of a saline extraction of the adrenals of a two day vaccinia rabbit gave (3-8-13) a fine rise followed by a marked fall. This extraction was made in 20 c.c. and was kept 24 hrs. in the ice-box, *i. e.*, since immediately after removal.

ABSTRACTS OF THE COMMUNICATIONS, PACIFIC COAST BRANCH.

Third meeting.

San Francisco, California, April 2, 1913.

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Preliminary communication on the part played by cholesterol in determining the incidence of carcinoma.

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We have elsewhere shown¹ that cholesterol, when injected directly into rat carcinomas, causes a marked acceleration both of the primary and of the metastatic growth of the tumors.

This led us to form the opinion that cholesterol is probably a factor of importance in determining the incidence of carcinoma.

It has been shown by Dorée and Gardner, Ellis and Gardner, and others² that cholesterol is not synthesized by animals, the

¹ T. Brailsford Robertson and Theodore C. Burnett, *Proc. Soc. Exper. Biol. and Med.*, 10 (1912), p. 59; *Journal of Exper. Med.*, 17 (1913), p. 344.

² C. Dorée and J. A. Gardner, *Proc. Roy. Soc. London*, 80 B (1908), p. 227; 81 B (1909), G. W. Ellis and J. A. Gardner, *ibid.*, 81 B (1909), p. 129.