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Effect of Cholesterol Feeding on Growth of Rats.

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In this laboratory, during the past few years, we have fed to a total of several hundred young rats diets containing 1% cholesterol. We have found, almost without exception, that if the basic diet to which the cholesterol was added was adequate to support entirely normal growth on a long-time basis, there was no significant difference between the growth curves of the cholesterol-fed animals and those of the littermate controls which received the same diets without the added cholesterol. Moreover, this was true as well for certain vitamin-deficient diets, notably those lacking A.

This finding is entirely at variance with that reported by Sperry and Stoyanoff¹ for synthetic diets. This would seem to indicate the value of analysis of the diets used in the two laboratories with this point in view.

The composition of our diets has already been reported.² Extracted casein was used as a source of protein in both laboratories and at levels sufficiently nearly alike as to probably rule out differences in behavior due to protein intake. Sperry, *et al.*, have used sucrose, and we have used starch as a source of carbohydrate. While the cornstarch used in our laboratory certainly did not contain enough betaine, as Best and Ridout³ have suggested that certain samples of potato starch may, to largely prevent cholesterol ester deposition in the livers of our rats, nevertheless there is no proof that it did not contain some factor which may be necessary for the growth of cholesterol-fed rats.

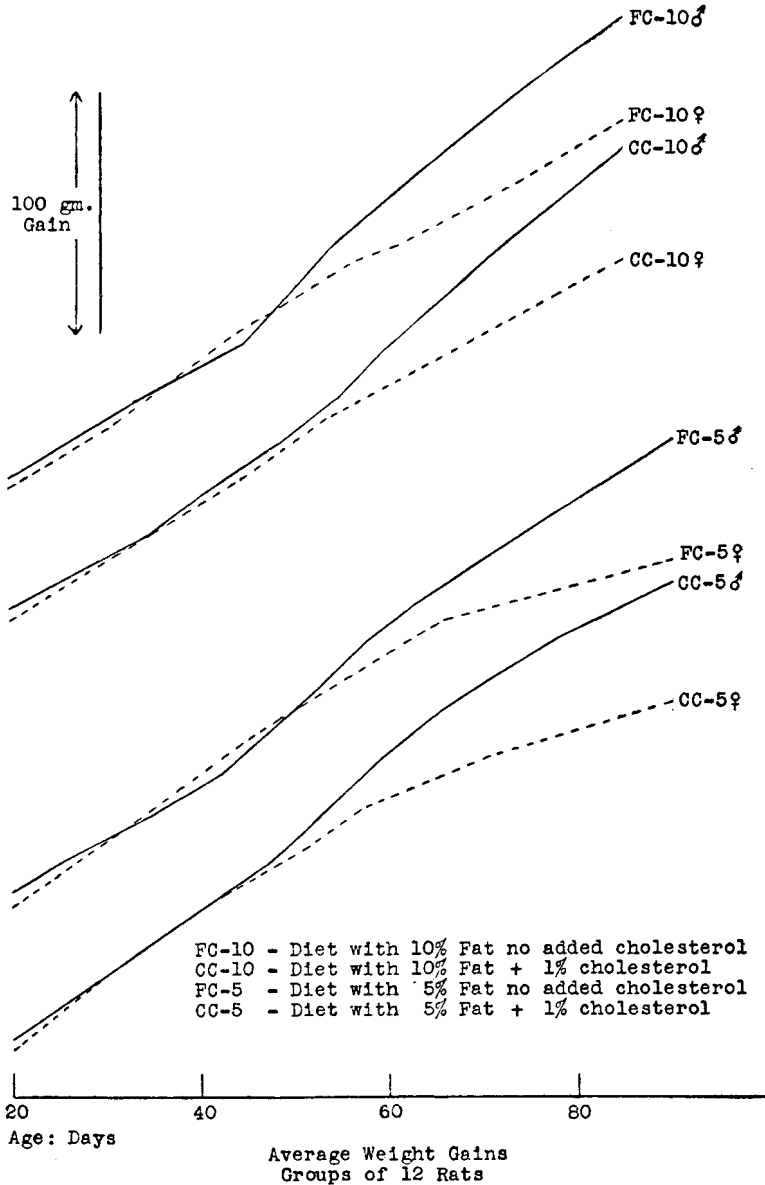
Again, we have used in this laboratory less highly purified sources of vitamins A, D, and, in some case, B and G, than the Columbia investigators. The behavior of vitamin-deficient animals fed cholesterol in this laboratory has been such as to indicate that lack of none of the vitamins just mentioned is in itself responsible for the retarded growth of Sperry's cholesterol-fed rats, but it does

* Deceased, November 16, 1935.

¹ Sperry and Stoyanoff, *J. Nutr.*, 1935, **9**, 131.

² Okey, Gillum and Yokela, *J. Biol. Chem.*, 1934, **107**, 207, and Okey, *Proc. Soc. Exp. Biol. and Med.*, 1933, **30**, 1003.

³ Best and Ridout, *J. Physiol.*, 1935, **84**, 7P.



not eliminate the possibility that some other accessory factor associated with our vitamin sources may have made it possible for our cholesterol-fed rats to grow normally.

Rats fed cholesterol in this laboratory have, when the fat intake was as high as that used by Sperry, shown liver cholesterol ester levels within the same range. The larger proportion of our rats

have, unfortunately for the sake of comparison, been fed cholesterol at lower levels of fat intake and have consequently had a lower percentage of total liver lipid. Analysis of all of our figures at present available shows little or no relationship between level of liver cholesterol ester and growth rate. Data at present available do not justify any conclusion as to the exact nature of the factor responsible for this difference in results in the two laboratories. It seems probable that some accessory substance is necessary to permit normal growth coincident with the storage of high levels of cholesterol ester in the liver, and that lack of this, rather than the cholesterol itself, may have been responsible for the retarded growth reported.

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Prevention of Intranasally Inoculated Poliomyelitis in Monkeys by Previous Intranasal Irrigation with Chemical Agents.*

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That the normal portal of entrance of poliomyelitis virus is the olfactory nerve is now well established.¹ It is also clear from recent studies² that the administration of specific immune serum does not afford significant protection against subsequent intranasal instillation of monkeys with poliomyelitis virus. The relative ineffectiveness of immune serum may be attributed to the fact that the terminals of the olfactory nerve are so situated that they cannot be effectively guarded by immune plasma. Once established in a nerve the virus travels to its destination in the medulla and cord by axonal paths,³ quite safely out of reach of immune substances in the plasma. Recent attempts at active immunization against this disease have also proved disappointing in that the injection of vaccines results merely

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¹ Schultz, E. W., and Gebhardt, L. P., *PROC. SOC. EXP. BIOL. AND MED.*, 1934, **31**, 728; Brodie, M., and Elvidge, A., *Science*, 1934, **79**, 235; Lennette, E. H., and Hudson, P., *PROC. SOC. EXP. BIOL. AND MED.*, 1935, **32**, 1444.

² Schultz, E. W., and Gebhardt, L. P., *J. Ped.*, 1935, **7**, 332.

³ Faber, H. K., *Medicine*, 1933, **12**, 83; Faber, H. K., and Gebhardt, L. P., *J. Exp. Med.*, 1933, **57**, 933.