No; for one outstanding fact is that two healthy brothers may differ at the same age, one being slender, the other very fleshy. Different human strains differ in build just as Jersey steers differ from Aberdeen Angus steers. Armsby has inquired into the cause of the difference in build of such steers: he concluded that it is partly due to different amounts of food consumed; the Jersey steer is a lighter eater. It is also due to the fact that the Jersey's excess calories are used in building up protein which stores up a relatively great amount of energy, per kilogram, while in the Angus, the excess calories are stored in the form of fat which uses up relatively little energy per kilo. There is a difference in the method of metabolizing. Apparently this difference is found also in families,—so that we have some families in which the members store fat, in others, protein; at least, some fatten easily, others with difficulty. Probably the constitutional difference in human families is that which distinguishes chow dogs and grey hounds, Cochin china pigs and razor backs.

Returning to humans, one finds that the offspring of two very slender parents are practically all slender. But the progeny of two parents of medium build in certain cases range from very slender to very fleshy. Slenderness is recessive; but fleshiness is not differentiated genetically from slenderness by a single factor, but sometimes by at least two independent factors, possibly more. However this may be, the analysis that can be applied to the 500 matings studied, shows that the capacity of fattening easily depends on germinal factors just as truly as stature does.

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The action of salicylates on the isolated heart.

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Most of the experiments were carried out on the frog heart but turtles were also used occasionally. Sodium salicylate in different concentrations in Ringer's solution produced the following results:

A solution of 1:2000 caused no effect or stimulation. When the concentration was 1:1000 and the heart was exposed to the action of the salicylate for ten to fifteen minutes considerable depression occurred. Slight improvement was sometimes noticed when the sodium salicylate was discontinued, but complete recovery was never observed. Stronger solutions produced still greater depression; 1:500 caused cessation of heart action in two minutes, but some improvement also occurred in this case when perfusion with Ringer's solution was resumed. With a concentration of 1:250 depression was still more pronounced.

Marked depression of the heart also occurred when it was perfused with very weak solutions of acetylsalicylic acid (1:4000 to 1:2000). It was found, however, that this was due to the increased hydrogen ion concentration of the solution, for by adding sodium hydroxide or sodium bicarbonate until the P_H was 7.4 or 7.5 (the same as that of Ringer's solution) stimulation was produced when the heart was perfused with the acetyl derivative of the same molecular concentration as that of sodium salicylate. Stronger concentration (1:250), however, caused cardiac depression though the hydrogen ion concentration was corrected as above to correspond with that of the Ringer's solution. The results show, therefore, that sodium salicylate is more toxic than the corresponding acetyl derivative. Similar results were obtained by Dreser[1] who perfused the frog heart with sodium salicylate and aspirin in defibrinated ox blood.

Experiments were also performed with methyl and ethyl salicylates. A saturated solution of the former (0.07 per cent. or less) produced complete heart block within two or three minutes which was promptly removed by perfusion with Ringer's solution alone.

Similar effects were produced by a saturated solution of ethyl salicylate, but the arrest of heart action set in after a period of five to ten minutes or longer. Recovery occurred also as after perfusion with methyl salicylate.

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[1] H. Dreser, *Archiv. fur Ges. Physiol.*, 1899, lxxvi, 306.