





When the mast cell counts of the guinea-pigs were plotted against body weight, a rectilinear relationship was found (Fig. 1). The correlation coefficient for the female animals was 0.89 and for the males 0.77. Selve⁶, in his intensive bibliography, presents no constant picture of the mast cell count in the ageing process, and makes no mention of it in guinea-pigs. Because weight gives a reasonable estimate of the age of these animals, my results suggest that there is a significant fall in the omental mast cell count with age within the range studied. R. ST. J. BUXTON

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¹ Shelley, W. B., and Florence, R., Nature, 191, 719 (1962).

² Cairns, A., and Constantinides, P., Science, 120, 31 (1954).
³ Koltai, M., Minker, E., and Deak, G., Die Naturwiss., 52, 15 (1965).

⁴ Harvey, E. B., Anat. Record, 148, 407 (1964).

⁵ Constantinides, P., and Rutherdale, J., J. Geront., 12, 264 (1957).

Selye, H., The Mast Cells (Butterworths, Washington, 1965).

Metabolic Differences between Dietary Liquid Glucose and Sucrose

In the fifty years since Higgins¹ obtained evidence that fructose has a greater tendency to change into fat in the body than has dextrose, more evidence has accumulated, much of it in the past few years, to show that lipid metabolism in man and experimental animals is affected differently by different carbohydrates²⁻⁵. Recently, it was found that substitution of sucrose for starch in the diet of weanling and three month old rats caused metabolic changes⁶, and that diets containing sucrose, and more especially fructose, increased plasma cholesterol levels and carcass and liver fat of adult rats compared with diets containing dextrose or liquid glucose or a standard laboratory diet?.

Long-term feeding trials in man under strict experimental conditions are difficult, but studies of primates are desirable to investigate further the possible connexion between dietary sucrose and ischaemic heart disease⁸⁻¹⁰. The susceptibility of baboons to develop atherosclerosis has been demonstrated¹¹, and at present we are using mature baboons (Papio anubis = P. doguera) for longterm feeding trials in which high carbohydrate diets are being compared. In preliminary studies with a small group of twelve animals, differences have been found between baboons fed liquid glucose and those fed sucrose; the present findings support the accumulating evidence that dietary carbohydrates are not all metabolically equivalent.

Sexually mature animals weighing about 9.5 kg were fed for 26 weeks on diets containing 74 per cent available carbohydrate, either as spray-dried liquid glucose BPC

Table 1	MEASUREMENTS OF	N	BABOONS	FED	LIQUID	GLUCOSE,	SUCROSE
	OR CONTRO	OL	DIETS FO	DR 26	WEEKS'	•	

	Liquid glucose	Sucrose	Control
Fotal serum lipid, mean value	347 ± 29	458 ± 27	341 ± 22
3-lipoprotein fraction (precipita- tion index (ref. 13)), mean	1.3 ± 0.1	$1{\cdot}8\pm0{\cdot}1$	1.5 ± 0.1
value over 20 weeks Results :	at end of exper	iment	
Energy value of food consumed $(\text{kcal} \times 10^3)$	$122 \cdot 1 \pm 4 \cdot 2$	$139{\cdot}1\pm8{\cdot}5$	$103{\cdot}5\pm3{\cdot}1$
Weight gained (kg)	1.0 ± 0.57	$2 \cdot 2 \pm 0 \cdot 51$	2.6 ± 0.16
Increase in thoracic girth (cm)	0.3 ± 1.41	3.5 ± 1.70	2.6 ± 1.33
Abdominal depot fat + epididy- mal fat (g)	33.6 ± 7.0	81.5 ± 5.2	$38 \cdot 6 \pm 19 \cdot 7$

Lipid content of abdominal 60.5 ± 10.3 depot fat (g/100 g) 80.2 ± 1.7 $64 \cdot 3 + 5 \cdot 3$

* The values are means and standard errors of means for groups of four mature baboons, two male and two female. Differences between sexes were not significant.

(ref. 12) (an aqueous preparation of partially hydrolysed starch) or as sucrose, or a control diet containing 47 per cent available carbohydrate mainly as starch but with some sucrose (4.5 per cent) and liquid glucose (4.5 per cent). The results are summarized in Table 1.

The total serum lipid of the baboons fed the three diets increased during the first 2 weeks, and the increase was greatest in the animals fed sucrose; at the end of the experiment, the total serum lipid in these was significantly higher (P < 0.01) than in the animals fed liquid glucose or the control diet. The serum β -lipoprotein was also significantly higher (P < 0.01) in the baboons fed sucrose than in those fed liquid glucose, while the corresponding value in the control group was intermediate.

Although the animals fed sucrose showed trends towards higher food intake, greater weight gain and larger increase in thoracic girth than those fed liquid glucose, the differences were not statistically significant.

In the animals fed sucrose, the combined weight of abdominal and epididymal fat was significantly greater (P < 0.01) than in those fed either of the other diets. The abdominal depot fat from the animals fed sucrose also contained significantly more lipid (P < 0.01) than that from the other animals.

Small atherosclerotic plaques were found in some animals, but their frequency was not related to the diet used and the lesions were apparently of long standing.

Our initial results indicate that, compared with a high liquid glucose diet, a high sucrose diet increases the level of serum lipid, and that this increase is associated with an increase in the amount of abdominal fat deposited. If these results with primates are confirmed in tests with larger numbers they could have implications for human diets.

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- ¹ Higgins, H. L., Amer. J. Physiol., 41, 258 (1916).
- ² Antar, M. A., and Ohlson, M. A., J. Nutrit., 85, 329 (1965).
- ³ Cohen, A. M., and Teitelbaum, A., Amer. J. Physiol., 206, 105 (1964).
- ⁴ Lopez, A., Hodges, R. E., and Krehl, W. A., Amer. J. Clin. Nutrit., 18, 149 (1966).
- ⁵ Macdonald, I., Proc. Nutrit. Soc., 23, 119 (1964).
- ⁶ Al-Nagdy, S., Miller, D. S., Qureshi, R. U., and Yudkin, J., Nature, 209, 81 (1966).
- 7 Allen, R. J. L., and Leahy, J. S., Brit. J. Nutrit., 20, 339 (1966).
- ⁸ Cohen, A. M., Bavly, S., and Poznanski, R., Lancet, ii, 1399 (1961).
- Ošancová, K., Hejda, S., and Zvolánková, K., Lancet, i, 494 (1965).
- 10 Yudkin, J., Lancet, ii, 4 (1964).
- ¹¹ Gresham, G. A., Howard, A. N., McQueen; J., and Bowyer, D. E., Brit. J. Exp. Path., 46, 94 (1965). ¹² British Pharmaceutical Codex 1963, 338 (The Pharmaceutical Press, London,
- 1963).
- ¹³ Heiskell, C. L., Fisk, R. T., Florsheim, W. H., Tachi, A., Goodman, J. R., and Carpenter, C. M., Amer. J. Clin. Path., 35, 222 (1961).