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Effect of Dietary Galactose vs. Glucose with Corn Oil and with Hydrogenated Coconut Oil on Various Metabolic Functions in Rats

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EFFECT OF DIETARY GALACTOSE VS. GLUCOSE WITH CORN OIL AND WITH HYDROGENATED COCONUT OIL ON VARIOUS METABOLIC FUNCTIONS IN RATS

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It has been shown in several reports that the responses of metabolic systems vary with different dietary combinations. For example, the source of glucose in the diet (that is — glucose, fructose, galactose, etc.) influences the metabolic route which the carbohydrate takes. There have been changes in the extent of lipogenesis, and changes in the amount of glucose converted to glycogen and to other products associated with glycolysis, as a result of the carbohydrate fed. Much comment and controversy has arisen over the variations in cholesterol and phospholipid levels in the body as a result of the fatty acid content of the dietary fat. The interrelationships of carbohydrate and fat metabolism have been shown to be affected by dietary sources also.

This particular study was designed to notice any influence by the type of dietary fat and the use of galactose as an indirect source of glucose on various adaptive metabolic responses in the albino rat. The particular responses which were used to evaluate any adaptations to the diet were the levels of total lipid, cholesterol, phospholipid, nitrogen, and glycogen in the liver, and the cholesterol concentration of the serum.

Male, weanling rats, weighing an average of 59 g at the beginning of the study, were fed a nutritionally adequate diet with the types of carbohydrate and fat as the only variables. The rats were divided into 4 groups, each of which received a 15% fat, 60.55% carbohydrate, 20% protein diet. Group I served as the primary control group, and was fed corn oil (CO) with glucose. Group II was fed the same source of unsaturated fat, CO, with galactose. Group III was fed a saturated fat, hydrogenated coconut oil (HCO), with glucose; and Group IV was fed HCO with galactose. These diets were continued 3 weeks, at the end of which time, the rats were decapitated and the livers and serum saved for analysis.

The overall differences between effects of diets on growth of the rats are shown in this growth curve. (Fig. 1) Galactose depressed the growth rate whenever it was fed, and HCO depressed the growth rate slightly when fed with glucose. The growth rate was extremely low when HCO and galactose were fed in the same diet. This was

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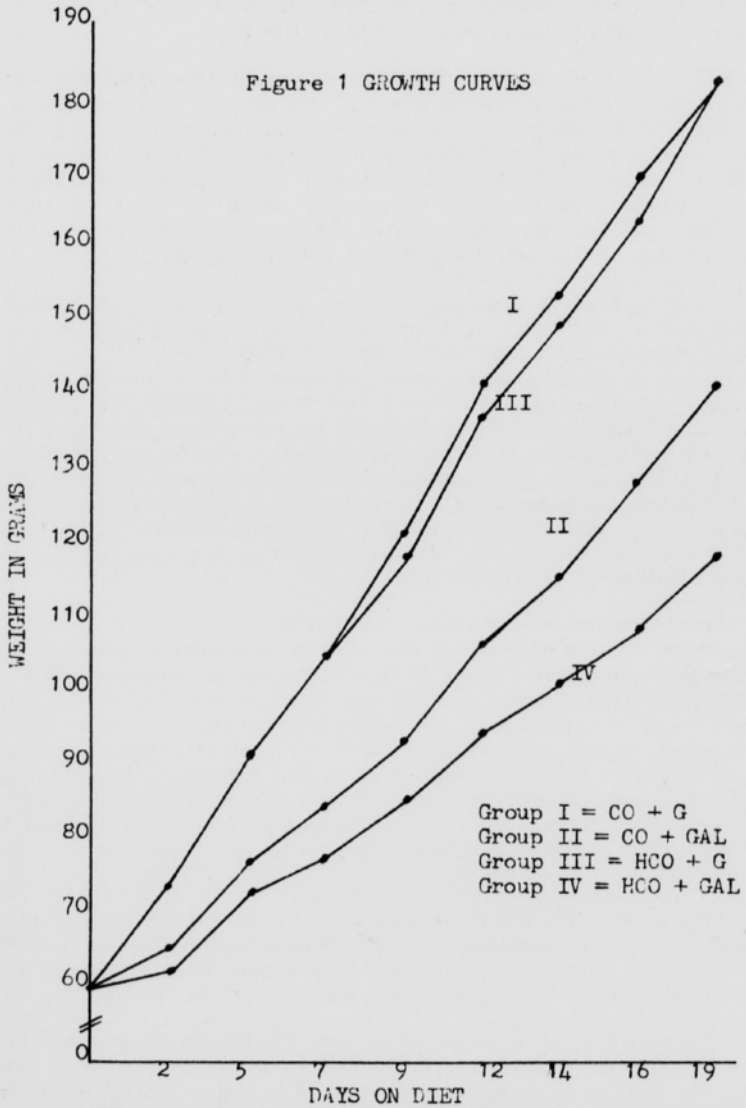


Fig. 1. Growth of rats fed different carbohydrate-fat combinations for 3 weeks.

an effect of an inefficient use of either galactose or HCO, since all groups ate about the same amount of food. (Fig. 2).

The glycogen content of the livers decreased when galactose was fed as compared with feeding glucose. The diet of HCO and glucose caused a slight decrease in glycogen as compared with CO and glucose. This is consistent with reports from other studies in which either HCO or galactose was fed. The effects of galactose and HCO seem to be additive when fed in the same diet, since the group fed HCO and galactose had the most extreme glycogen reduction either as per cent of total liver or per 100 g of body weight. (Fig. 3).

Studies in which activities of various enzyme systems were measured showed that when HCO was fed, there was an increase in glucose-6-phosphatase activity over that exhibited when CO was fed. There was a similar increase when galactose was the dietary carbohydrate instead of glucose. Since this enzyme catalizes the reaction from G-6-P to glucose, the decrease in glycogen could be due to increased hydrolysis of G-6-P to glucose with a resultant decrease in G-6-P available for other pathways. Since galactose is converted to G-1-P before going to G-6-P, it might be expected to be a better precursor of glycogen than glucose, but the interconversion of G-1-P to G-6-P has been shown to be sufficiently rapid so that the 2 compounds serve metabolically as a single pool.

Some researchers have found that in the conversion of galactose to glucose there is an accumulation of large quantities of galactose or galactose-1-phosphate. They think this excess of galactose-1-P inhibits an enzyme necessary in the normal utilization of glucose. It is known that in galactosemia, the inherited metabolic disorder in which the enzyme which converts Gal-1-P to G-1-P is missing, there is another enzyme system which increases in activity with age. Possibly this enzyme system could increase in activity when diets containing large amounts of galactose are fed, and help eliminate the accumulation of galactose-1-P. The study would have to be continued over a longer period of time to prove this theory.

There is also a possibility that relatively low levels of liver glycogen in rats fed galactose were an effect of mobilization of glucose for fatty acid synthesis, since the total lipid expressed as per cent of the liver was higher in rats fed galactose than in those fed glucose (Fig. 3).

As shown in other studies with rats, the dietary unsaturated fat caused more total cholesterol in the liver than did the saturated fat. Also the diets containing galactose resulted in higher cholesterol levels in both liver and serum than did diets containing glucose. When CO and gal were fed together, serum and liver cholesterol values were higher than in any of the other groups.

Phospholipid content of the liver was higher when saturated fat was fed than when CO was fed. There were no significant differences

Figure 2 FOOD EFFICIENCY RATIO

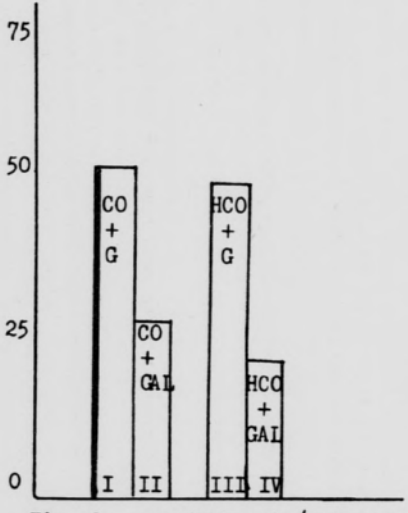
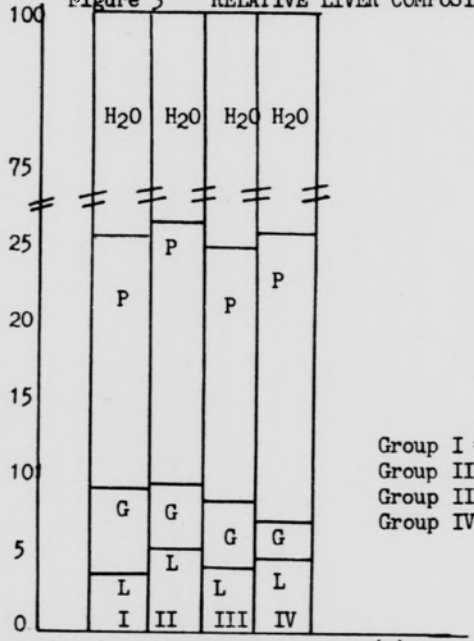


Fig. 2. g weight gain/g food intake for groups of rats fed different carbohydrate-fat combinations for 3 weeks.

Figure 3 RELATIVE LIVER COMPOSITION



Group I = CO + G
Group II = CO + GAL
Group III = HCO + G
Group IV = HCO + GAL

Fig. 3. Percent of protein (P), glycogen (G), lipid (L) and water in livers of rats fed different carbohydrate and fat combinations for 3 weeks.

in phospholipid values between the groups fed the two sources of glucose.

Per cent hepatic nitrogen (and hence, hepatic protein) was found to be slightly greater in rats fed diets containing galactose instead of glucose.

There were interesting physiological effects when galactose was fed with either fat. After two days on the diets both groups developed polyurea, and after 13 or 14 days developed a dense lenticular opacity. These effects lasted from the time of first appearance throughout the experimental period.

These effects, as well as the striking reduction in weight gain of the galactose-fed rats, indicate that they were in a state of relative protein deficiency in comparison to the glucose-fed controls, due to increased excretion of nitrogen-containing compounds. A test with phlorogulcinol indicated the presence of galactose in the urine. Possibly then, the renal tubules were so saturated with galactose they were unable to reabsorb many of the amino acids and peptides back into the blood stream, causing this protein deficiency.

As yet the cause for cataracts is not certain, but they are associated with both galactosemia and diabetes. It has been suggested that the excessive amounts of galactose in the urine prevent re-entry of other essential substances which require the same pathway in the body, and this deficiency somehow results in cataract formation. Also the inhibition of aerobic glycolysis by galactose has been linked with marked derangements of metabolic processes in the lens, causing cataract formation.

This leads to a little more insight into the intricacies of metabolic interrelationships which, as we learn more about them, seem to become even more complex.