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16. Lens epithelia were cultured as described in (*10*) except that the explants were oriented with the lens capsule upward and the epithelial cell monolayer positioned between the capsule and the surface of the culture dish.
17. Cell length was determined on living epithelia by focusing on the apical and basal cell surface and measuring the distance between these focal planes. Measurements were made with a micrometer attached to the stage of a Zeiss inverted microscope equipped with Nomarski interference contrast optics (Baltimore Instrument Co.). Measurements were made at the center and at four equally spaced points 125 μm from the center of each epithelial explant. Cell lengths were confirmed by direct measurement with an ocular micrometer after the explants were fixed, embedded in glycol methacrylate (JB-4 embedding kit, Polysciences) and sectioned at 2 μm with a Dupont Sorvall JB-4 microtome.
18. Mean cell area was determined on fixed epithelia after staining with Gill's hematoxylin 2 (Polysciences). By use of an ocular reticule, a 2768-μm^2 area was superimposed on the cells at the center of the epithelia and the number of nuclei lying within this area was counted. Mean cell area was taken as 2768 μm^2 divided by the number of nuclei. Average cell areas for control epithelia were: zero time, 37.2 $\pm$ 0.7 μm^2; 5 hours, 37.6 $\pm$ 0.9 μm^2; and 24 hours, 35.7 $\pm$ 0.9 μm^2 (mean $\pm$ S.E.).
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8 March 1979; revised 13 April 1979

Triglyceride Concentrations: The Disaccharide Effect

Abstract. *The mean 24-hour or integrated concentration of triglyceride is significantly higher when dietary sucrose is provided rather than an equivalent amount of its component monosaccharides, glucose and fructose. In contrast, the plasma triglyceride concentration after a 12-hour fast is not significantly different.*

Plasma concentrations of triglyceride are influenced by a variety of factors, including the amount and composition of dietary carbohydrate. Sucrose has repeatedly been reported to result in elevated concentrations of triglyceride in humans and animals (*1*), an effect usually ascribed to the fructose component of sucrose. Michaelis and co-workers (*2*) found that lipogenic enzymes in rat liver were induced to a greater extent when the animals were fed sucrose than when they were fed equivalent amounts of glucose and fructose, the monosaccharide components of the disaccharide sucrose. This phenomenon has been termed the disaccharide effect. The metabolic scope of the disaccharide effect has been expanded to include differences in serum triglyceride and free fatty-acid concentrations after fasting (*3*); differences in serum insulin concentrations, food efficiency, and relative fat pad size (*4*); and differences in hepatic microsomal enzyme activities (*5*). Documentation of the disaccharide effect has heretofore been limited to studies with various strains of rats (*2–5*). This report indicates that when normal human subjects consume diets containing sucrose, the mean 24-hour (or integrated) triglyceride concentrations are significantly higher than when the subjects are fed diets that are identical except that fructose and glucose are provided as monosaccharides.

The design, sample collection, assay methods, and statistical evaluation used in this study are analogous to those described in detail earlier (*6*). Significant features include the use of a diet sequence that results in all subjects ingesting both test diets in a balanced crossover design, and continuous collection of blood over an entire 24-hour period as a series of 48 half-hour integrated collections. The withdrawal system allowed normal activity and consumption of the test diets while the blood samples were collected. Eight normal males aged 24 to 27 were studied on the tenth day of ingestion of the test diets. The study protocol was approved by the University Committee on Research Involving Human Beings. Informed consent was ob-

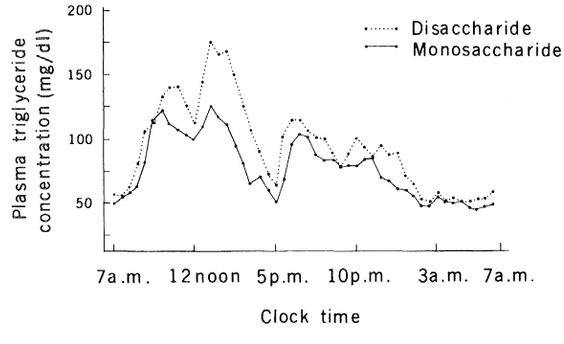

Fig. 1. Mean diurnal triglyceride patterns for two diets that varied in the form of fructose. The disaccharide diet (sucrose) and the monosaccharide diet (glucose and fructose) each provided 11.3 percent of total calories as fructose.

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tained from each subject after an explanation of the purpose, methods, and potential benefits and risks of the study.

Liquid formula diets provided 45 percent energy from carbohydrate, 40 percent from fat, 15 percent from protein, and 300 mg of cholesterol daily. Casec (Mead Johnson Laboratories), a lactose-free, defatted casein preparation, supplied 95 percent of dietary protein; the remainder was provided in an egg yolk mixture. Dietary fat was provided as a mixture of peanut oil, cocoa butter, and egg yolk. The proportions of these components were adjusted to maintain a constant iodine number of 80 to 85 and a ratio of polyunsaturated to saturated fat of 0.7 for each dietary period.

Only the carbohydrate composition of the diets varied. Both diets provided 50 percent of the carbohydrate from cornstarch. The remaining 50 percent was provided as sucrose in one diet and as an equimolar mixture of fructose and glucose in the other.

The timing of ingestion of the test diet and the proportion of calories consumed at each meal varied between subjects on the basis of personal preferences, but remained consistent for each subject during all study periods. The mean pattern provided 28 percent of calories at 7:30 a.m., 4 percent at 10:00 a.m., 29 percent at noon, 29 percent at 5:00 p.m., and 10 percent at 9:00 p.m. The evening snack varied between subjects more than any other meal, providing 0 to 28 percent of total calories and being taken between 8:00 and 10:00 p.m.

The mean plasma concentrations of triglyceride obtained after a 12-hour fast were 64 ± 9.9 mg/dl (mean ± standard error) after the sucrose diet and 58 ± 9.9 mg/dl after the ingestion of glucose and fructose as monosaccharides; the difference is not statistically significant. In contrast, the mean 24-hour triglyceride concentration of 97 ± 13.8 mg/dl during ingestion of the sucrose diet was significantly higher ($P < .02$) than the mean of 77 ± 9.6 mg/dl during ingestion of the glucose and fructose diet.

The integrated concentration represents the arithmetic mean of the 48 individual integrated samples collected over the 24-hour period. The higher triglyceride integrated concentration observed during ingestion of sucrose resulted from a marked difference in tryglyceride concentration between approximately 10:00 a.m. and 5:00 p.m., as seen in Fig. 1.

These results indicate that the disaccharide effect originally described by Michaelis and co-workers (*2*) occurs in human subjects. Under the conditions of the study, consumption of a formula diet containing sucrose by normal adult males results in significantly higher integrated concentrations of triglyceride than does consumption of equivalent amounts of fructose and glucose provided as monosaccharides. The mechanism for the difference in the integrated concentration and the diurnal pattern of triglyceride remains undefined. The possibility that a difference in insulin response to the two diets could explain the observed difference in triglyceride concentrations was suggested in earlier animal studies (*4*). However, the mean insulin concentrations after an overnight fast, the mean integrated concentrations of insulin, and the diurnal pattern of insulin concentrations did not vary between the two diets evaluated in this study. Thus, insulin does not explain the disaccharide effect in humans.

These results indicate that dietary carbohydrate may influence prandial and postprandial triglyceride concentrations without resulting in similar changes in plasma triglyceride concentrations after an overnight fast. The higher triglyceride concentrations observed between 10:00 a.m. and 5:00 p.m. can only be ascribed to the different sources of dietary carbohydrate, because the amount of carbohydrate, fat, and protein and the source of fat and protein were identical in the two diets. This discrepancy between the triglyceride integrated concentrations and the triglyceride concentrations in the fasting state (Fig. 1) suggests that future studies of dietary influences on triglyceride concentrations should include evaluation of the concentrations after ingestion of the test diets as well as after an overnight fast.

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12 March 1979; revised 18 May 1979

Pierce's Disease Bacterium: Mechanism of Transmission by Leafhopper Vectors

Abstract. *The bacterium that causes Pierce's disease of grapevines is isolated most consistently from the foregut of its leafhopper vector* Graphocephala atropunctata. *As seen in light and scanning electron microscopy of infective leafhoppers, the bacteria are attached to the cibarial pump and the lining of the esophagus in the foregut where they appear to multiply. These findings suggest that the bacterium is transmitted from the foregut by egestion during feeding by infective leafhoppers.*

The bacterium that causes Pierce's disease (PD) of grape can be transmitted to grapevines and other plants by a large number of xylem-feeding leafhopper and spittlebug species (*1, 2*). In addition to its wide vector range, this as yet unclassified bacterium (*3*) can infect a large diversity of plant species (*4*) and cause disease in almond, alfalfa (*3, 5*), and perhaps citrus (*6*). Another characteristic that distinguishes the transmission of the PD bacterium from other leafhopper-transmitted prokaryotic plant pathogens is that adult leafhoppers can transmit immediately after acquiring the bacterium (*7*), and that they continue to transmit efficiently for the remainder of their lives, which may be several months (*1, 2, 7*). The finding that infective nymphs do not transmit the PD bacterium after molting suggested that the PD bacteria attach to some portion of the foregut (the foregut lining is shed in molting) (*8*).

We have identified the location of the PD bacterium in the foreguts of infective leafhoppers by examining tissues in both infective and noninfective blue-green sharpshooters [*Graphocephala atropunctata* (Signoret)] (*9*) with conventional light microscopy and scanning electron microscopy. In addition, we isolated the bacterium from aseptically

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