blood glycerides was ten times higher in livers from fed rats (0.69% of the dose/min) compared with livers from rats starved for 24h (0.065%). Production of ^{14}C -labelled ketone bodies in the steady state was presumably a result of lipolysis of liver ^{14}C -labelled glycerides. It was higher in the livers of starved rats ($0.0278 \pm 0.008\%$ of the dose/min) compared with those of fed rats ($0.0013 \pm 0.0002\%$ of the dose/min). Production of total ketone bodies was also higher in livers from starved rats. Addition of glucagon to perfused livers from fed rats depressed the rate of formation of ^{14}C -labelled very low density lipoproteins and increased the rate of formation of ^{14}C -labelled ketone bodies; it was without effect on livers from starved rats. Addition of insulin to livers from starved rats resulted in an increased output of ^{14}C -labelled very low density lipoproteins and a reduction in formation of ^{14}C -labelled ketone bodies.

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Direct Stimulation by Insulin and Fructose of Very-Low-Density Lipoprotein Secretion by the Perfused Liver

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Perfused livers from fed male rats (340–360g) were infused with [$1\text{-}^{14}\text{C}$]oleate, to maintain a concentration of $0.9\text{ }\mu\text{mol/ml}$ of serum. A trace quantity of [$1\text{-}^3\text{H}$]acetate was also infused continuously. In some experiments fructose was infused to maintain a concentration of $25\text{--}30\text{ mg/100ml}$ of blood. Where appropriate, bovine insulin was added to maintain a minimum concentration of 56 ng/ml . High physiological concentrations of rat insulin were obtained in some experiments by pre-treating the blood-donor rats with intraperitoneally administered glucose. The perfusions were initiated with defibrinated rat blood that had been dialysed against Krebs–Henseleit bicarbonate buffer. An equal volume of similar but undialysed blood was added subsequently. The blood glucose concentration was stabilized at $200\text{--}300\text{ mg/100ml}$. Other experimental details were as described by Mayes & Felts (1966).

In control perfusions, production of very-low-density lipoprotein (density <1.006) averaged $27\text{ }\mu\text{mol}$ of triglyceride fatty acids/h per liver. In the presence of higher concentrations of rat insulin or fructose the average rate of secretion rose to $39\text{ }\mu\text{mol/h}$ per liver. However, when bovine insulin or an increased concentration of rat insulin was present in addition to fructose there was a marked augmentation of the rate of secretion to 56 and $53\text{ }\mu\text{mol/h}$ per liver respectively. Incorporation of ^{14}C -labelled long-chain free fatty acids into very-low-density lipoprotein showed similar trends. There was, however, a stimulation of incorporation in the presence of higher concentrations of rat insulin. Oxidation of ^{14}C -labelled free fatty acids to $^{14}\text{CO}_2$ was decreased by insulin and fructose added separately and was decreased further when both were present together. Lipogenesis, as indicated by the incorporation of [^3H]acetate into triglyceride of very-low-density lipoprotein, was stimulated by the presence of fructose and insulin and was inversely related to the oxidation of ^{14}C -labelled free fatty acids.

The following hypothesis is suggested to explain the combined effect of insulin and fructose in stimulating very-low-density lipoprotein secretion. The decreased oxidation of ^{14}C -labelled free fatty acids may indicate that insulin enhances net esterification of free fatty acids (acyl-CoA), thus removing inhibition of lipogenesis by acyl-CoA. The pathway would then be open for rapid lipogenesis from fructose, followed by enhanced secretion of very-low-density lipoprotein. Present results may help to explain the correlation between atherosclerosis, sucrose intake and the concentration of serum insulin (Yudkin, Szanto & Kakkar, 1969).

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The Site of Fatty Acid Biosynthesis in the Avocado Pear

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Barron, Squire & Stumpf (1961) have described the incorporation of [$1\text{-}^{14}\text{C}$]acetate into fatty acids by the mitochondrial fraction from homogenates of the mesocarp of the avocado pear. Subsequently Yang & Stumpf (1965) reported the incorporation