Chloroplast membranes retard fat digestion and induce satiety: effect of biological membranes on pancreatic lipase/co-lipase

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Biochem. J. (2007) 401 (727–733) (Printed in Great Britain)

Human obesity is a global epidemic, which causes a rapidly increased frequency of diabetes and cardiovascular disease. One reason for obesity is the ready availability of refined food products with high caloric density, an evolutionarily new event, which makes over-consumption of food inevitable. Fat is a food product with high caloric density. The mechanism for regulation of fat intake has therefore been studied to a great extent. Such studies have shown that, as long as fat stays in the intestine, satiety is promoted. This occurs through the fat-released peptide hormones, the best known being CCK (cholecystokinin), which is released by fatty acids. Hence, retarded fat digestion with prolonged time for delivery of fatty acids promotes satiety. Pancreatic lipase, together with its protein cofactor, co-lipase, is the main enzymatic system responsible for intestinal fat digestion. We found that biological membranes, isolated from plants, animals or bacteria, inhibit the lipase/co-lipase-catalysed hydrolysis of triacylglycerols even in the presence of bile salt. We propose that the inhibition is due to binding of lipase/co-lipase to the membranes and adsorption of the membranes to the aqueous/triacylglycerol interface, thereby hindering lipase/co-lipase from acting on its lipid substrate. We also found that chloroplast membranes (thylakoids), when added to refined food, suppressed food intake in rats, lowered blood lipids and raised the satiety hormones, CCK and enterostatin. Consequently, the mechanism for satiety seems to be retardation of fat digestion allowing the fat products to stay longer in the intestine.