

EFFECTS OF DIETARY SODIUM PROPIONATE ON VOLATILE
FATTY ACIDS AND LIPID METABOLISM IN RATS AND PIGS

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Human diets high in various dietary plant fibre preparations seem to be of benefit in lowering blood lipids in subjects with hyperlipidaemia. These fibre fractions include guar gum, oat bran and pectin and in each case the active constituent appears to be a polysaccharide which forms a viscous solution in water. Such fibres are also highly susceptible to bacterial fermentation in the large bowel and yield VFA in significant amounts (Topping & Illman, 1986). This latter feature has led Chen et al (1984) to propose that one of these VFA (propionate) mediates the lipid-lowering by inhibiting hepatic cholesterol synthesis. This suggestion stems from observations that dietary propionate opposes diet-induced hypercholesterolaemia in rats while sodium propionate at high (>15mM) concentrations suppresses cholesterol synthesis from ¹⁴C-acetate in isolated hepatocytes. To determine whether such inhibition occurred in vivo, rats were fed diets containing 5% sodium propionate for 10 days and synthesis then measured with tritiated water.

As in previous studies with cholesterol supplemented diets, plasma cholesterol was lowered from 2.92±0.12 (5) umol/ml in controls to 2.36±0.21 (5) umol/ml in rats fed propionate (P<0.01). Although total hepatic portal venous VFA were similar in both groups with a combined mean of 1.64±0.07 (10) umol/ml of plasma, propionate levels in this vessel rose significantly (P<0.001) from 0.22±0.02 (5) umol/ml in controls to 0.38±0.04 (5) umol/ml in the supplemented group. Nevertheless, this was far below the level required for inhibition of cholesterologenesis in vitro and was shown to be the case in vivo also as synthesis was found to be identical in both groups with a combined mean of 310±10 (10) nmol/min/g of liver.

The recovery of propionate appeared low in comparison to the amounts fed so the response to a diet supplemented with 2% propionate was followed in pigs with hepatic portal venous cannulae. Plasma concentrations rose by 0.4-0.6 umol/ml within 45min of feeding but at later times did not differ from controls given chow alone. Post-mortem examination of digesta suggested substantial absorption (and so metabolism) in the upper gut. It appears that in both species dietary propionate does not increase plasma concentrations enough to cause inhibition of hepatic cholesterol synthesis. The present data support the view that while VFA are important metabolic fuels in non-ruminant omnivores, they do not appear to lower plasma cholesterol directly (Topping and Illman, 1986).

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