Lipolysis induced by coffee and tobacco: its modification by insulin

Smoking or drinking coffee produces lipolysis as shown by the increase in plasma free fatty acids (PFFA). It has already been shown (Portugal-Alvarez, 1972; Kershbaum, Bellet & others, 1961; Bellet & Kershbaum, 1965; Portugal-Alvarez, Perezagua & Velasco-Martin, 1972) that habitual smokers and coffee drinkers possess higher basal PFFA levels than those who are not habituated to these substances. In the present study we have confirmed the lipolytic action of tobacco and coffee and we point out the different enzymatic mechanisms by which one or the other of these agents produces lipolysis.

In the study of lipolysis produced by tobacco the PFFA levels were determined (Dole & Meinertz, 1960) in 15 subjects under basal conditions. The subjects stayed resting and smoked and inhaled 2 cigarettes of black tobacco in 10 min. Blood samples were taken 10, 30, and 60 min after starting the cigarettes. The experiment was repeated, except that an injection of insulin (10 I.U.) was given before smoking. In the study of lipolysis produced by coffee the PFFA levels were determined in 18 subjects under basal conditions and then again 30, 60, 120 min after drinking 200 ml of coffee prepared by adding 15 g of Colombian coffee to 250 ml of boiling water. Analytical tests showed that each subject received 170 mg of caffeine in 200 ml of coffee. The experiment was repeated and each subject was injected with 10 I.U. of insulin as they drank the coffee.

Each experiment was carried out after 12 h of rest and with no intake of food at any time during this period. Blood samples were heparinized and analysed immediately after collection. All medication was suppressed for a week before the experiment to eliminate the possibility of drugs or their metabolites influencing the metabolism of lipids. Statistical significance of the difference between means was determined by the t-test for paired data (Snedecor & Cochran, 1967).

The increment of PFFA, with respect to the basal values was statistically significant at 10 but not 30 and 60 min after smoking 2 cigarettes. There was no significant increase in the PFFA relative to the basal values, 10, 30, or 60 min after smoking in subjects given insulin (Fig. 1). The change in PFFA, relative to the basal values, was significant 30, 60 and 120 min after coffee in subjects not given insulin and in those who were, (Fig. 2) but in the presence of insulin the increase was less marked than with coffee alone.

The fundamental difference in the lipolysis produced by tobacco and the produced by coffee is that the first is inhibited by insulin whereas the second is not. Tobacco produces lipolysis by means of ganglionic stimulation as well as suprarenal

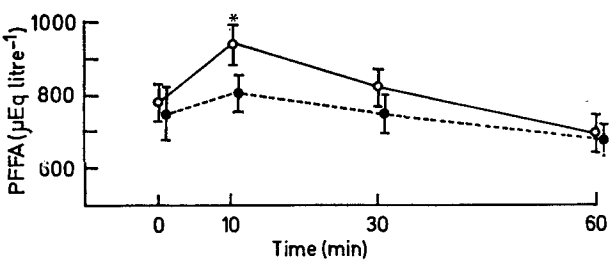

FIG. 1. PFFA after 2 cigarettes had been smoked (mean ± s.e., n = 15) before (○) and after (●) administration of 10 I.U. of insulin. The zero point on the abscissa represents the time at which the cigarettes were started. * represents a significant increase from basal levels ($P<0{\cdot}01$).

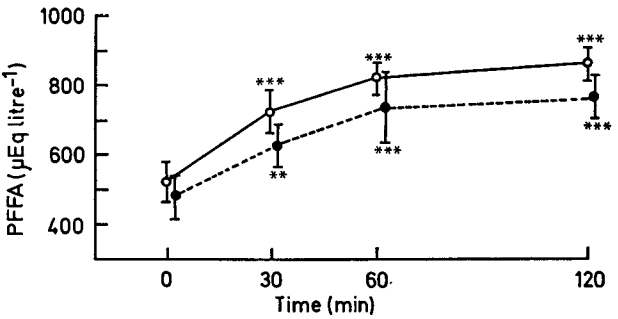

FIG. 2. PFFA after 200 ml of coffee containing 168·7 mg caffeine (mean ± s.e., n = 18) before (○) and after (●) administration of 10 I.U. of insulin. The zero point on the abscissa represents the time which the coffee was started. Asterisks represents a significant increase from basal levels; **, $P<0.02$; ***, $P<0.001$.

stimulation. The liberated catecholamines activate adenylcyclase in the plasma membrane of the adipose cells, catalysing the formation of cyclic 3′-5′-AMP which in turn activates the triglyceride-lipase. However coffee produces lipolysis by inhibiting the nucleotide 3′-5′-phosphodiesterase enyzme that catalyses the formation of 5′-AMP from cyclic 3′-5′-AMP. This inhibition of the degradation of cyclic 3′-5′ AMP results in increased activation of the triglyceride-lipase.

Insulin inhibits the lipolysis produced by tobacco because in some way it impedes the activation of adenylcyclase by catecholamines. On the other hand, it is inefficient or less efficient in inhibiting lipolysis produced by coffee because insulin possibly does not affect the nucleotide 3′-5′-phosphodiesterase enzyme. However, caffeine sensitizes tissues to catecholamines, producing on some occasions the cardiovascular effect of the catecholamines (Rall & West, 1963). This together with the inhibiting effect of the phosphodiesterase contributes to increased lipolysis. A discrete liberation of catecholamines, induced by methylxanthines may also be involved (Darby, Sprouse & Walton, 1958), which could explain why the lipolysis, after coffee and insulin, even though significantly elevated, is less than that produced only by coffee alone.

This suggests therefore, that there is an interaction of some kind in the lipolysis induced by coffee and that this has two components: (1) induced tissue sensitivity to catecholamines by the caffeine; and (2) direct lipolysis through the phosphodiesterase.

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