ABSTRACT

This study was designed to investigate the effect of nicotine and cigarette smoke on insulin release in vivo, and furthermore, to elucidate the mechanism of action of nicotine on insulin release. Nicotine is known to be the most important pharmaco-active agent of cigarette smoke.

One hundred and sixty-five non-diabetic Wistar rats and seventy diabetic Wistar rats of both sexes, weighing between 250 - 350 gms, were investigated. Diabetes mellitus was induced by the application of 25 mg/kg body weight of streptozotocin, intravenously. After anaesthetizing the rats with sodium pentobarbitone, at a dosage of 50 mg/kg body weight, by intraperitoneal injection, blood was sampled at intervals from the portal vein to determine insulin, glucose and catecholamine levels.

In the non-diabetic rats, nicotine in doses up to 20 ug/kg body weight produced a significant change (p < 0.005) in the total output of insulin compared with the non-stimulated insulin output over a 90-minute period. A reduction in the insulin output was observed as the nicotine dose increased above 20 ug/kg body weight.

Although in the non-diabetic animals, pulse and bolus forms
of nicotine application produced an insulin output that was not significantly different from that obtained with the smoke from one cigarette, the result with the pulse form of nicotine application came nearest to that of the cigarette smoke.

After the application of nicotine, the blood glucose and catecholamine levels rose significantly and the hyperglycaemia was more pronounced in the diabetic rats, which showed reduced insulin levels, and therefore were not in a position to counteract the rise in the blood glucose levels. It became apparent that although nicotine utilizes the autonomic nervous system to induce insulin release, there may be another route by which nicotine exerts its influence on the B-cells. This route appeared to be via hexamethonium - and atropine-resistant receptors on the B-cell, which react directly with nicotine to induce insulin release.