

ABSTRACT

Low insulin release has been observed in PEM; however, structural damage of the endocrine pancreas is not considered a major causative factor. Although PEM can be considered a stress situation, and catecholamines are known to be inhibitors of insulin release, little work has been done on the role of the sympathoadrenal system on insulin release in PEM.

Control rats (132 ± 2.6 g) and malnourished rats (53 ± 0.55 g) fed on lab chow for 21-28 days after weaning were anaesthetized with 50 mg/kg body weight sodium-pentobarbital intraperitoneally, and blood for insulin radioimmunoassay sampled from the portal vein.

Fasting insulin levels in 30 malnourished and 30 control rats were 1.6 ± 0.4 uU/ml and 3.93 ± 0.26 uU/ml respectively ($p < 0.001$). The two groups of animals showed a significant difference in insulin output in response to glucose load.

After collecting fasting blood in malnourished and control rats, an alpha-receptor blocker, phentolamine (0.25 mg/kg body weight), was administered through the inferior vena cava and blood sampled

over a period of 2, 5, 10, 20, 30, 45 and 60 minutes. The area under the curves showed no difference in magnitude of insulin release in the two groups.

Administering α -methyltyrosine (80 mg/kg body weight) for four days before sacrifice, catecholamine synthesis was blocked in 30 control and 30 malnourished rats. The fasting insulin levels in control and malnourished rats were 9.83 ± 0.86 uU/ml and 9.63 ± 0.6 uU/ml respectively. The insulin response to intravenous glucose challenge in the control and malnourished rats treated with α -methyl tyrosine for four days before sacrifice was exaggerated during the first phase of release and was of the same magnitude in both groups as compared with rats without drug treatment.

Fifteen control and 15 malnourished rats were treated with a tranquilizer, diazepam (0.15 mg/kg body weight), over a period of 10-14 days. Fasting insulin levels in the malnourished and control rats were 3.17 ± 0.16 uU/ml and 4.61 ± 0.11 uU/ml respectively, a significant increase

($p < 0.001$) in the malnourished animals compared with rats without drug treatment.

The plasma calcium and potassium levels, which are known to influence insulin release, showed no significant difference between control and malnourished groups.

Finally, the histological examination of the islets by light microscopy showed the presence of "normal" B cells in both the malnourished and control rats.

These results show that the sympathoadrenal system is one of the factors causing low insulin release in PEM. Although in PEM low insulin output is a form of adaptation to life, high insulin levels are required during treatment. The anabolic effects of insulin might, therefore, be enhanced if PEM patients were subjected to less stress during the period of treatment.