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Letters to the Editor

Glucose Tolerance in Opiate Addicts

Dear Sir,

The fact that morphine can raise blood glucose levels has been adequately demonstrated only in the diabetic dog, and as an acute effect [1]. In human opiate addicts, metabolic effects induced by heroin [2] and morphine [3] have been described. Reed and Ghodse [2] performed a 50-g oral glucose tolerance test and found a high basal plasma insulin level with a delayed, raised peak value, with an overall high insulin level. These authors explained their results as being due to a delay in gastric emptying and insulin resistance.

Ceriello et al. [3] found blood glucose levels unchanged and glycosylated haemoglobin A_1 increased, thus inferring an impairment of glucose homeostasis in opiate addicts.

Our results are not in agreement with these findings. After a 4-week period of methadone treatment (40 mg/day), a group of in-patient heroin addicts (> 18 months duration) was submitted to a 75-g oral glucose tolerance test. All the patients were within ± 10% of their ideal body weight, eating a weight-maintaining diet and taking no drugs apart from methadone. The results were compared with those from a group of normal subjects, matched for age, sex and body weight.

The results can be summarized as follows: blood glucose levels after a 12-h fast were similar (normal subjects: 4.3 ± 0.2 versus 4.2 ± 0.18 mmol/l), a significant difference was only found at 30 min (normal subjects: 6.5 ± 0.23 versus 5.6 ± 0.21 mmol/l; $p < 0.02$). Basal plasma insulin levels were significantly higher in the addicts (normal group: 11.7 ± 2.1 mU/l versus addicts: 18.4 ± 1.7 mU/l; $p < 0.02$), whereas at 30, 60, 90 and 120 min after glucose, plasma insulin values were significantly lower in the drug addicts (Table 1).

Our data demonstrate that opiate addiction per se does not impair glucose tolerance, as suggested by Ceriello et al. [3], nor does it induce a condition of insulin resistance, as proposed by Reed and Ghodse [2]. Furthermore, we would like to emphasize the significantly lower plasma insulin levels found in our patients. Our results are in apparent contrast with those reported by Reed and Ghodse [2], who observed an increase in insulin response to a glucose challenge in a group of drug addicts.

In order to explain these differences, two considerations must be taken into account: (1) the nutritional state of the subjects studied (all our patients were well nourished), and (2) the size of the glucose load. Reed and Ghodse [2] used a 50-g glucose load which is known not to provide a reliable stimulus for studying glucose tolerance and insulin release, compared with the 75-g load used in our study. Furthermore, the insulin levels obtained by Reed and Ghodse in normal subjects in response to the oral glucose load seem to be low, even for a 50-g glucose load.

Yours sincerely,

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Table 1. Oral glucose tolerance test in ten normal subjects and ten opiate addicts

		Time (min)						
		0	30	60	90	120	180	240
Blood glucose (mmol/l)	normal subjects	4.3 ± 0.2	6.5 ± 0.23	6.1 ± 0.19	5.7 ± 0.21	4.9 ± 0.3	3.9 ± 0.12	3.5 ± 0.12
	addicts	4.2 ± 0.1	5.6 ± 0.21	6.1 ± 0.16	5.46 ± 0.18	4.76 ± 0.26	4.0 ± 0.16	3.8 ± 0.17
	p	NS	0.02	NS	NS	NS	NS	NS
Plasma insulin (mU/l)	normal subjects	9.8 ± 2.1	95 ± 8.2	78 ± 8.0	66 ± 7.1	52 ± 5.3	23 ± 3.1	16 ± 2.0
	addicts	18.4 ± 1.7	58 ± 8.0	59 ± 7.3	46.6 ± 6.8	38 ± 5.6	30 ± 4.2	20 ± 3.1
	p	< 0.02	< 0.01	< 0.01	< 0.02	< 0.05	NS	NS

Results expressed as mean ± SEM; NS = not significant