change from the 0 min values. Student's t-test[7] was used to assess the statistical significance between the control and experimental groups at each time period.

Results. 10 min after anesthesia, the plasma prolactin levels in the chloroform and halothane anesthetized groups were significantly higher ($p < 0.01$) than the unanesthetized control groups (figure). In the subsequent time periods the prolactin levels were not significantly different from that of the control group. With both chloroform and halothane, anesthesia was induced within 1–2 min after exposure to the anesthetic.

Discussion. The pattern of plasma prolactin response induced by chloroform and halothane in ovariectomized, estrogen-treated rats was similar, although the magnitude of rise and sharpness of decline was more pronounced with chloroform than with halothane. Such an acute rise and fall in the plasma prolactin levels was observed by us for 2 other inhalation anesthetics, ether and MF[4,5]. From the similarity of the responses observed, it is possible that all 4 inhalation anesthetics may act through a common neuroendocrine mechanism in releasing prolactin. Although Krulich[8] has reported that a central serotonergic mechanism is involved in ether-induced prolactin release in rats, recent reports[5,9] from our laboratory indicated the possible involvement of other biogenic amine systems as well.

In our experience, all 4 inhalation anesthetics release prolactin in estrogenized female rats, however, MF seems to be safer, easier to manage, and causes minimum respiratory difficulties.

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Nicotinamide and streptozotocin diabetes in the rat. Factors influencing the effectiveness of the protection

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Summary. The effectiveness of nicotinamide in protecting against streptozotocin diabetes does not only depend on streptozotocin dosage but also on the age of the rats. Also, the extent of the protection suddenly decreases when the interval between streptozotocin and nicotinamide injections exceeds 1 h.

Many drugs can protect against the diabetogenic effect of alloxan but only nicotinamide is effective in the case of streptozotocin (SZ)[1]. It is known that this long-lived precursor of nicotinamide-adenine dinucleotides[2] prevents the destruction of NAD[3] and the subsequent decrease of the NAD levels in beta cells after SZ[4]. Also, pyridine nucleotides may be involved in insulin release induced by glucose[5,6]. The specificity of this interaction invited further research. It was found that protection depends on the dosage of both drugs[7,8] and that nicotinamide can even reverse the effects of SZ[7,9,10], although its efficacy decreases with the time elapsed after SZ administration[10]. However, quantitative information is still lacking which could explain the early damage of the beta cells and lead to a more accurate timing of the onset of no-return changes in this endocrine tissue.

In our first experiment, we administered nicotinamide (70, 140, 210 and 240 mg/kg b.wt, i.p.) 15 min before the i.v. injection of 50, 60, 70 or 80 mg/kg b.wt SZ to 130 g rats and we evaluated plasma glucose levels by a glucose-oxidase/peroxidase method (Glucosio-Test, Sclavo I.S.V.T., Siena) 24 h later. Results indicated that nicotinamide could let us detect differences among the effects of the slightly different doses of SZ, in spite of the similar

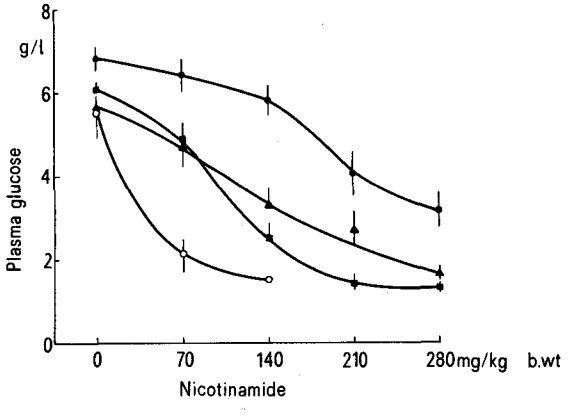

Fig. 1. Plasma glucose concentrations in 130 g male Wistar albino rats 1 day after the injection of different doses of streptozotocin (○, 50 mg/kg; ■, 60 mg/kg; ▲, 70 mg/kg; ●, 80 mg/kg b.wt) which followed the i. p. administration of the amounts of nicotinamide indicated on the abscissa. The interval between the 2 injections was 15 min. Means of 6 cases are given. Vertical bars represent 1 or 2 × SEM.

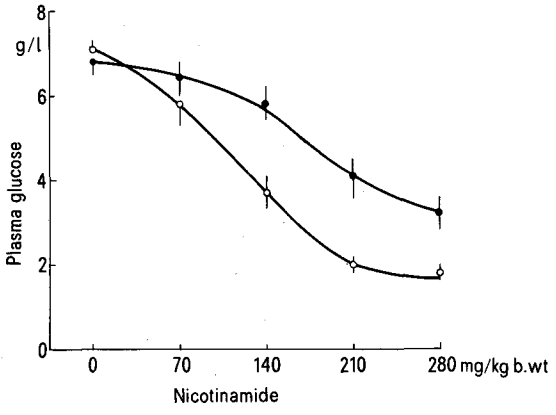

Fig. 2. Plasma glucose concentrations in 70 g (○) or 130 g (●) male Wistar albino rats 1 day after the i.v. injection of 80 mg/kg b.wt streptozotocin and nicotinamide (see figure 1). Means of 6 cases are given. Vertical bars represent 1 or 2 × SEM.

blood glucose levels attained in the unprotected animals (figure 1). In fact, the levels of plasma glucose attained in the animals which received SZ but not nicotinamide did not significantly differ, except in the case of the highest SZ dose (80 mg/kg b.wt). However, different amounts of nicotinamide were required to provide full protection in the different experimental groups (70–140 mg/kg in 50 mg/kg SZ given rats; 210 or 280 mg/kg in rats given 60 or 70 mg/kg SZ respectively). When 80 mg/kg SZ were given, even the largest dose of nicotinamide provided partial protection only.

Most likely, the fact that protection changes together with the relative dosages of both nicotinamide and SZ is not a consequence of an interaction between these 2 molecules at the level of cell receptors. Probably, the obtainable protection depends on the extent of the beta cell damage, which, in its turn, depends on the size of SZ dose. Evidence supporting this statement comes from our second experiment (figure 2): younger animals (70 g b.wt) – which are less sensitive to SZ[11] – are protected by nicotinamide much better than older animals (130 g b.wt) given the same dose (80 mg/kg b.wt) of SZ. Incidentally, this finding can help to explain conflicting results previously reported[7, 9, 10].

Finally, we explored whether similar experimental designs could let us time when the pancreatic lesions induced by SZ become irreversible. 0, 70, 140 or 240 mg/kg nicotinamide were given to 130 g rats 75, 45, 15 min before or 45, 75 min after the injection of a medium dose (60 mg/kg b.wt) of SZ (figure 3). Changes of blood glucose were similar and full protection obtained at 140–210 mg/kg b.wt nicotinamide in all cases except in that of the latest nicotinamide administration (75 min after SZ injection). At this interval, the protective effect of nicotinamide is remarkably reduced (but still significant at the highest nicotinamide dosage). If this is the lag that SZ-induced lesions require to become irreversible in most beta cells, we should comment that no-return changes appear much later than the earliest signs of cell lesion detectable at the electron microscope (Z. Gori, personal communication). Also, they seem to be simultaneous with (or shortly follow) NAD depletion[4] and are accompanied by the first significant impairment of insulin secretion[12].

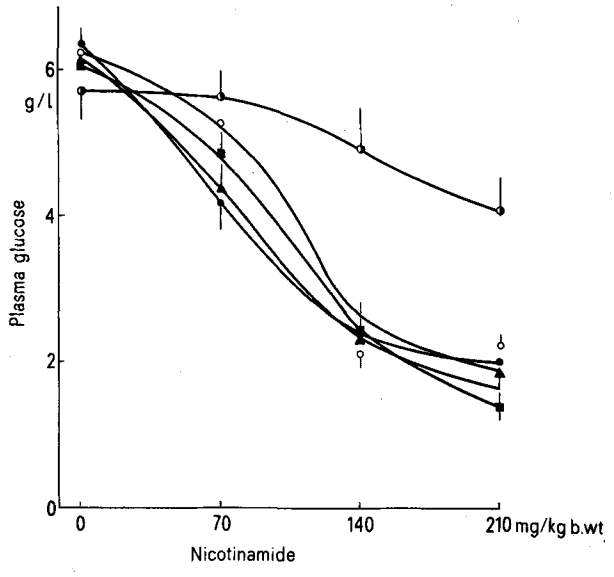

Fig. 3. Plasma glucose concentration in 130 g male Wistar albino rats 1 day after the injection of 60 mg/kg b.wt streptozotocin. Nicotinamide was administered at the dosage indicated on the abscissa respectively 75 min (▲), 45 min (●) or 15 min (■) before or 45 min (○) or 75 min (◑) after the streptozotocin injection. Means of 6 cases are given. Vertical bars represent 1 or 2 × SEM but are omitted when symbols overlap.

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Endocrine organs of different larval instars of Spodoptera (Prodenia) litura Fabricius (Lepidoptera: Noctuidae)

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Summary. A_1 and A_2 cells of the median neurosecretory group are filled with PF stained neurosecretory material between the first 24 to 48 h of the earlier moult in each instar followed by immediate release before the next moult at 72 h, while loaded posterior and lateral neurosecretory cells also appear from the 3rd and 5th instars onwards. Corpora cardiaca, corpora allata and prothoracic glands increase in size in each instar.

Various lepidopterous larvae such as Hyalophora cecropia[1, 2], Bombyx mori Linn[3, 4]. and its race, nistari[5], Ostrinia nubilalis[6] Heliothis zea[7] and 13 genera of 8 families[8] have been studied in respect to the morphology and histology of the retrocerebral complex and prothoracic glands. However, this paper presents the changes in the form of the endocrine organs in various larval instars of Spodoptera litura ultimately leading to moulting in them.

Material and methods. The larvae of Spodoptera litura were reared on soybean leaves from the eggs laid in the

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