Table II. Results of biochemical assay of acid phosphatase and nonspecific esterases with their standard deviations

| Organs | Enzyme type | Enzyme activity in mU/mg of protein | | |
|---|---|---|---|---|
| | | Control | DMN | MMS |
| Liver | Acid. phosphatase | 1.10 ± 0.14 | 0.95 ± 0.24 | 1.00 ± 0.18 |
| | Esterases | 4.70 ± 0.90 | 4.90 ± 1.50 | 4.20 ± 1.20 |
| Kidney | Acid. phosphatase | 0.78 ± 0.18 | 0.79 ± 0.15 | 0.72 ± 0.10 |
| | Esterases | 4.90 ± 0.30 | 4.85 ± 0.80 | 4.40 ± 0.85 |
| Testis | Acid. phosphatase | 0.18 ± 0.09 | 0.19 ± 0.12 | 0.18 ± 0.12 |
| | Esterases | 5.40 ± 0.40 | 5.50 ± 1.50 | 5.00 ± 0.60 |

mU = international milliunit. Post-coupling technique was employed.

experimental animals (DMN and MMS groups), than was observed amongst the controls. As the large standard deviations in the experimental groups overlapped those of the latter, no statistically significant differences were noted between them. The bioassay data are presented in Table II.

*Discussion.* In view of the available reports that larger doses of alkylating agents than used in the present investigation are required for experimental pathogenesis in rats, the findings noted may be of significance. HULTIN et al.[10] reported that the smallest dose of DMN necessary to produce definite necrosis of the liver is about 20 mg/kg. The present findings suggest that hepatic involvement, as evinced by scattered areas of centrilobular necrosis and enzyme deficient islands, can result from administration of the chemical at half the stated dosage. Such occurance of morphological and enzymological changes even in a small proportion of experimental animals underlines the role of individual susceptibility in the causation of lesions by the alkylating agents. The bioassay data exhibiting wide variations in enzyme levels amongst the individual experimental animals are corroborative in this regard. However, the absence of any statistically significant difference in enzyme activities between the experimental and control groups, as noted earlier, is relevant to the biochemical findings reported by SLATER et al.[7]. Working on hepatic lysosomal hydrolases following necrotizing doses of hepatotoxins, including DMN, these authors observed little alteration in lysosomal activity in the prenecrotic and in the early necrotic stages of hepatic involvement and concluded that the lysosomes probably play no role in the early development of hepatic lesions. Changes in activity that were observed after 10 h were attributed to the aftermath of the necrotic process, rather than with its initiation.

The selective loss of the terminal cathodal esterase of the testis in 3 DMN-treated animals is also significant, as this isozyme seems characteristic of the organ. In all zymograms with both unsubstituted and substituted naphthyl esters this particular variant features prominently. Besides, its capacity to produce hybrid esterase species is also distinctive[11]. The present finding points to the fact that cytochemical changes may occur from administration of alkylating agents before any morphological changes become evident and that such subtle changes may be detectable by appropriate analysis of the constituent isozyme systems.

*Zusammenfassung.* Ein kleiner Teil der mit Dimethylnitrosamin (10 mg/kg) behandelten Ratten zeigte Lebernekrose und Suppression eines Esterisozyms in den Hoden.

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## Intramitochondrial Bodies in the B-Cells of Rat's Pancreas Treated by Glybenclamide

Intramitochondrial bodies, different in shape, size and structure, and bound to the matrix or the cristae, have been described in the cells of various animals. They are detached in different organs in various conditions – normal, pathological or experimental, and in different evolutionary phases – embryonal and postnatal[1–11].

Peculiar intramitochondrial structures are seen in the B-cells of the islets of Langerhans of the pancreas of the rat, treated with glybenclamide (HB 419). A single dose of glybenclamide (7 mg/kg) was given orally to 9 white rats. The animals were sacrificed by decapitation on the 2nd, 8th and 24th h. 2 rats were used as controls. The pancreas was removed immediately and fixed in 1% osmium tetroxide in Millonig's buffer at pH 7.4 for 1 h at 4 °C. After a fast rinse in the Millonig's buffer, the specimens were dehydrated and embedded in Durcopan. Reichert ultramicrotome was used to obtain specimens. The stains used were uranyl acetate after WATSON[12] and lead citrate after REYNOLDS[13]. Sections were examined by Hitachi HU-11 A electron microscope.

Dense intramitochondrial bodies, single or multiple (Figure 1), were detected in the mitochondria of some of the B-cells, more frequently in the pancreas of animals sacrificed after 24 h. These bodies were mainly observed in bigger mitochondria. The mitochondria themselves (matrix, cristae and membrane) did not show any

significant alteration. Only occasionally the membrane at one end of the mitochondria showed illdefined 'smeared' appearances. The electron-dense intramitochondrial bodies were of varied length and were sometimes disposed alongside the mitochondrials axis. Their width was about 300 Å; they were located in the matrix and not connected with the mitochondrial cristae which were frequently parallel to them. The ultrastructure of the dense bodies demonstrate a certain periodicity of about 85–90 Å (Figure 2).

In general the intramitochondrial bodies described in the literature are varied in appearance. They are bound to the matrix or to the mitochondrial cristae[5]. Bodies located in the matrix, are described by many authors to be most frequently in liver or kidney cells[1–3, 9].

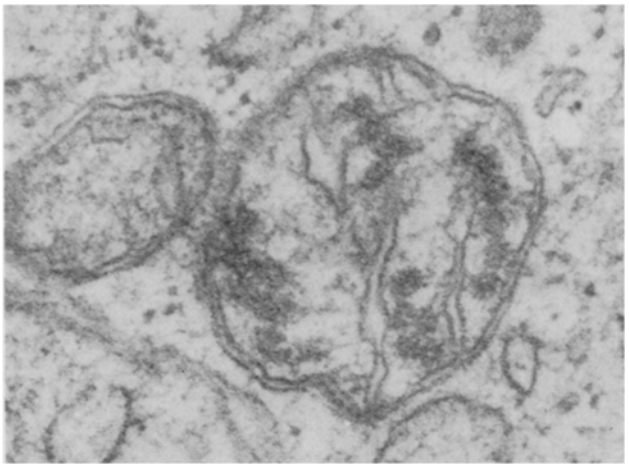

Fig. 1. Mitochondria containing several cross cut intramitochondrial bodies. ×82,000.

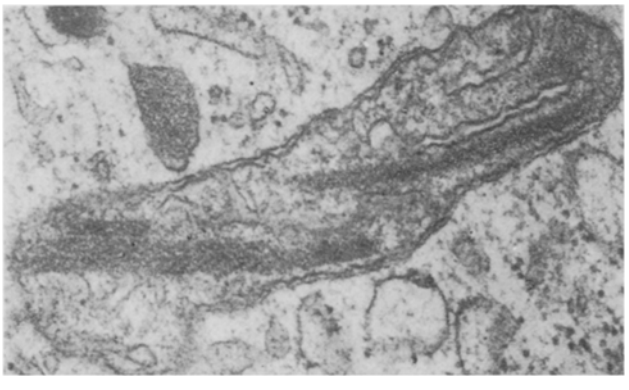

Fig. 2. Intramitochondrial body longitudinally cut. Its ultrastructure shows a certain periodicity. ×60,000.

What is the origin and the function of these structures? After NASS and NASS[14], the fine filaments depicted by them in the matrix of mitochondria in chicken embryos, represent DNA. After other authors, the filament bodies originate from the cristae as a result of disturbance of the phospholipid metabolism most frequently under the influence of some noxious effect[3, 4, 15, 16], or due to the peculiarities of breeding animals under laboratory conditions[9].

The intramitochondrial bodies in the suprarenal cortex and in the testes are considered in relation to their possible participation in hormones formation[6, 17]. After SVOBODA[15] they are an expression of degenerative changes or a manifestation of cell death in normal conditions in normal organisms[4].

The appearance of intramitochondrial bodies in pancreatic B – cells is, in our opinion, related to stimulation – in this case by glybenclamide. It is possibly an expression of activating the enzyme systems in mitochondria for providing the cell with the necessary energy.

*Résumé.* Description des corps intramitochondriaux se trouvant dans les cellules B du pancréas du rat blanc stimulées par le glybenclamide (HB 419). Dans quelques uns de ces corps, une périodicité fut observée. L'apparition des corps intramitochondriaux serait liée à l'activation de certains groupes enzymatiques.

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## Location of the Avian Tumor Virus Group Specific Antigen in the BAI Strain A Virus Associated Myeloblast Cell

The group specific (GS) antigen of the avian sarcoma and leukosis viruses has been found in the soluble fraction of the cells infected with any of the avian tumor viruses[1]. However, none of the studies done so far have shown avian tumor virus GS antigen associated with any subcellular fractions, although electron microscope[2] and fluorescent[3] antibody studies have demonstrated virus elaboration at the cell surface. In this study, using BAI strain A virus associated avian myeloblasts, and a technique for isolation of intact cell membranes, we have demonstrated that GS antigen is in fact associated with cell membrane fraction.