whether the N-acetyl peptide is located in the heavy chain or in the light chain; and whether it is situated in the head or the tail of the myosin molecule.

The heavy chain, light chain and heavy meromyosin were digested by pronase, respectively. The acetyl group was determined on an acidic peptides fraction isolated by passing the pronase digest through a cation exchange resin, and the acetyl peptide was purified by chromatography on an anion exchange resin and on thin layers.

On the acidic peptides fraction, 0.96 moles of acetyl group per 2.0×10^5 of heavy chain and 1.86 moles of acetyl group per 3.4×10^5 of heavy meromyosin were determined, respectively. On the other hand, the determination failed to reveal any acetyl group in the acidic peptides fraction of light chain.

On purified N-acetyl peptide, N-acetyl-Ser-Ser-Asp-Ala-Asp, 0.30 moles of the peptide per 2.0×10^5 of heavy chain and 0.25 moles of the peptide per 3.4×10^5 of heavy meromyosin were isolated, respectively. Calculating the yield to take isolation losses of the peptide in purification procedures, the yield of the peptide corresponded to 0.83 moles per 2.0×10^5 of heavy chain and 0.78 moles per 3.4×10^5 of heavy meromyosin, respectively. These results strongly suggest that N-acetyl-Ser-Ser-Asp-Ala-Asp is located in the heavy chain and is situated in the head of myosin molecule.

CHOLECYSTOKININ-PANCREOZYMIN-LIKE EFFECT INDUCED BY INTRA-INTESTINAL ADMINISTRATION OF TRYPSIN INHIBITORS

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1) The effects of synthetic trypsin inhibitors (TIs) on exocrine pancreatic secretion were investigated in anaesthetized rats.

2) Intra-intestinal administration of *p*-aminobenzamidine (*p*-ABA), a synthetic TI, caused a biphasic effect on exocrine pancreatic secretion; a marked increase in protein output to about 11 times as much as the control level accompanying a parallel increase in juice flow in the initial phase, and gradual decline in protein output maintaining the enhanced flow rate in the latter phase. Similar effects were also observed by another synthetic TI, *p*-ethoxycarbonylphenyl-ε-guanidinocaproate phosphate.

3) The initial phase of pancreatic response was diminished after flushing

the mucosal surface of the intestine with physiological saline, whereas the latter phase was almost unaffected.

4) Intra-venous administration of *p*-ABA produced an effect similar to the latter phase; an increase in pancreatic juice with a transient slight increase in protein output. A similar effect was also observed after the intra-intestinal administration of diluted HCl.

5) Since the initial phase resembles the known effect caused by the intra-venous administration of cholecystokinin-pancreozymin (CCK-PZ), it may be due to an increase in CCK-PZ release from a sort of intestinal endocrine cells. Since the latter phase, which resembles the known effect of secretin, was also produced by intra-venous administration of *p*-ABA, it seems to be due to stimulation of the exocrine pancreatic cells.

6) On these and other results, the mechanism of the CCK-PZ-like effect caused by TIs is discussed in connection with the "negative feedback hypothesis" proposed by GREEN & LYMAN (1972).

PATHOLOGIC DIFFERENTIATION BETWEEN LYMPHOID LEUKOSIS AND MAREK'S DISEASE IN CHICKENS LIGHT AND ELECTRON MICROSCOPIC OBSERVATIONS

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Light and electron microscopic observations were conducted in order to clarify the characteristics of the lesions of lymphoid leukosis (LL) and Marek's disease (MD). The materials investigated were collected from culled chickens (field cases) with visible solid tumors in the liver and the other visceral organs. For histopathological investigation, 26 cases of MD and 14 cases of LL were selected. Diagnosis of MD was based on the findings reported by FUJIMOTO et al. (1971), and that of LL was conducted by comparing the lesions found in chickens (5 cases) inoculated experimentally with avian leukosis virus. Three cases of LL and 7 cases of MD were also investigated electron-microscopically.

The tumors of LL in the various visceral organs and tissues were generally composed of a homogeneous population of either hemocytoblastic lymphoid cells or lymphoblastic lymphoid cells. The hemocytoblastic lymphoid cells were characterized by a homogeneous distribution of polyribosomes in the cytoplasm and the presence of giant nucleoli. The size of the lymphoblastic lymphoid cells