Parasitic forms of a myxosporean in the kidney of *Lampetra japonica* : An ultrastructural study

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No information have been available on myxosporea in cyclostomata. The present study was originally planned to investigate "macrophagic cells" in the kidney of arctic lamprey *Lampetra japonica.* Subsequent ultrastructural study revealed that the "macrophagic cells" were unidentified myxosporean trophozoites parasitized at high prevalences in the fish kidney. The trophozoites (pseudoplasmodia with/without sporoblasts) existed predominantly in the lumen of proximal urinary tubules, but were rarely found in any other regions of the kidney. Since no mature spores were found, exact identification of species was impossible. Two types of parasitic forms were observed in proximal urinary tubules: one attaching to the epithelial cells of renal tubules, and the other free-floating in the lumen of tubules. Ultrastructurally, the attaching trophozoites developed microvilli-like projections towards the apical surface of epithelial cells and interdigitated with microvilli of the brush border. In contrast, the whole surface of floating trophozoites was smooth without any cell projections. The developed projections of trophozoites may contribute to their firm attachment to the epithelial cells and/or absorption of nutrients via the epithelial cells. Against the myxosporean infection, the lamprey as the host provoked a local immune reaction by disposition of numerous lymphocytes and macrophages into the epithelium of urinary tubules.

Contribution of dihydropyridine-insensitive mechanisms to Ca^{2+} influx in rat pancreatic β cells

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1. The present study was carried out to clarify the role of Ca^{2+} influx through dihydropyridine-insensitive mechanisms in stimulus-secretion coupling in rat pancreatic β cells. To characterize the dihydropyridine-insensitive Ca^{2+} influx, intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$) was directly monitored by a microfluorometric method using Fura-2 in isolated perifused preparations of rat pancreatic islets. The changes in $[Ca^{2+}]_i$ induced by high concentrations of glucose and potassium ion (K^+) were analyzed quantitatively, and influence of Ca^{2+} entry blockers upon $[Ca^{2+}]_i$ changes was examined.

2. Stimulation of pancreatic islets with 20 mM glucose caused a biphasic $[Ca^{2+}]_i$ dynamics: a transient increase followed by a sustained in-