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Three-dimensional ultrastructure of synoviocytes in the knee joint of rabbit and morphological changes in osteoarthritis model

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The synovial intima is a cell-rich layer consisting of two morphologically different cell types: cells of macrophagic origin (type A synoviocytes) and fibroblast-like F cells (type B synoviocytes). The synovial macrophages can phagocytose actively cell debris and wastes wandering in the joint cavity to refresh the components of synovial fluid. F cells are proper cells, which secrete collagen, fibronectin, hyaluronan and other proteoglycans into the interstitium and joint cavity. The present study, mainly using scanning electron microscope (SEM), deals with the distribution and morphology of the two types of synoviocytes in whole synovial intima of the normal knee joint in rabbits, and the definite morphological changes of synoviocytes in an osteoarthritis (OA) model of rabbits.

Major population of the cells lining in the synovial intima were F cells characterized by possessing unique cytoplasmic processes. F cells were classified into flat type and dendritic type. The flat type F cells were densely arranged to cover the surface of synovial membrane like an epithelium. The cell bodies were superficially located in the synovial intima and projected flat and short processes. The surface of cell bodies and processes were smooth in appearance, without developed microvilli and microplicae. In dendritic type F cells, the cell bodies were located deeply in the synovial intima, and dispersed with wide interstitial spaces between cell bodies. The cells extended a major projection toward the surface of the synovial intima, where the projec-

tion branched out radially. Thus, a characteristic meshwork composed of long and thick processes was formed on the surface of synovium. Some dendritic type of F cells, which were observed in the fatty tissue around patella, possessed the projections protruding vertically toward joint cavity. The tips of these projections were covered with a tuft of long microvilli. These two types of F cells changed their morphology gradually, and the intermediate forms appeared in middle region. Therefore, the projections of F cells tended to become shorter and flatter, and the cell body tended to be located more superficially as leaving from the patella and the tendon of quadriceps femoris muscle. Many macrophages, spherical or slender in shape, were densely distributed on the fatty tissue around patella, while macrophages were rarely seen in other regions. The differences in distribution and cell shape may reflect secretory and phagocytotic activities and resistance to the impact associated with joint movement. It is necessary to keep in mind this difference, when we fully understand morphological changes of synoviocytes in pathological cases such as arthritis.

An osteoarthritis (OA) model was produced by transection of anterior cruciate ligament. At 2 and 4 weeks after surgery, both types of synoviocytes increased in number and changed their morphology. Changes of number were more remarkable in macrophages, and possibly induced by shifting of bone marrow-derived monocytes. These

macrophages developed many vacuoles in their cytoplasm. It is suggested that macrophages play important roles in phagocytosis of cell debris and wastes during the early stage of OA. On the other hand, F cells first increased in number by mitosis in the synovial intima to be densely arranged and later changed their morphology. F cells in OA model extended longer cytoplasmic processes and formed microvilli and micropliae on

their cell surface. Under TEM, many rough endoplasmic reticulum were observed in the cytoplasm of F cells. These morphological changes of F cells may reflect their stimulated activities in secretion and absorption. It is suggested that the ultrastructural changes of synoviocytes reflect pathological conditions of synovial membrane, and synoviocytes play some important roles in pathogenesis of OA.

Ca²⁺-activated K⁺ channels involved in muscarinically stimulated salivary secretion in ruminant parotid acinar cells

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The aim of the present study was to characterize Ca²⁺-activated K⁺ channels responsible for muscarinically evoked K⁺ efflux driven by HCO₃⁻ transport in bovine parotid acinar cells.

Net K⁺ efflux from perfused bovine parotid gland fragments was measured using flame photometry. Acetylcholine (ACh) perfused over the fragments for 3 min caused a transient net K⁺ efflux in a concentration-dependent manner. The ACh (10 μM) -induced K⁺ efflux was abolished by the muscarinic antagonist atropine (1 μM) and strongly inhibited by removal of CaCl₂ from the perfusing solution (with addition of 0.5 mM EGTA). The Ca²⁺ ionophore, A 23187 (3 μM) plus added Ca²⁺ was able to mimic the ACh-induced response. Net K⁺ efflux evoked by ACh (10 μM) or by A 23187 (3 μM) was inhibited by Ca²⁺-dependent K⁺ channel blockers, quinine (1 mM) and Ba²⁺ (1 mM), but not by tetraethylammonium (TEA) (10 mM), a blocker of large-conductance, voltage- and Ca²⁺

-dependent K⁺ channels (Maxi-K⁺ channels). Removal of HCO₃⁻ (25 mM) / CO₂ (5%) from the perfusing solution abolished ACh (10 μM) or A 23187 (3 μM) -induced net K⁺ efflux. In sharp contrast to the HCO₃⁻/CO₂ requirement for the K⁺ efflux in bovine parotid fragments, in the rat submandibular gland fragments there was no significant difference between the ACh (10 μM) -induced K⁺ efflux observed in normal and HCO₃⁻/CO₂-free perfusate. Acetazolamide (1 mM), an inhibitor of carbonic anhydrase reduced the ACh-induced K⁺ efflux by 30% in bovine parotid fragments.

In whole-cell patch clamp experiments, bovine parotid acinar cells exhibited both outwardly and inwardly rectifying conductances, attributable to TEA-blockable Maxi-K⁺ channels and inward rectifier K⁺ channels respectively, when the cells were dialyzed with a K-glutamate-rich pipette solution containing 100 nM or less than 1 nM free Ca²⁺. When the cells were dialyzed in a bath solution contain-