## Effects of bradykinin on uptake and release of substances by mouse peritoneal macrophages

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Non-activated peritoneal macrophages were collected from freshly killed mice and incubated in Roswell Park Memorial Institute (RPMI) medium + 10% foetal calf serum. The suspension was incubated in sterile Petri dishes containing 9 round cover slips. After 1 h, the cover slips were washed and the attached cells cultured overnight. [$^3$H]-Sucrose, $^{45}CaCl_2$ or $^{86}RbCl$ were added at this stage (for measuring release from cells) or at the following incubation (for measuring uptake and release). During the latter incubation cover slips were removed at intervals and the radioactivity in the cells measured by scintillation counting. Bradykinin depressed pinocytosis and release of [$^3$H]-sucrose, and the uptake of $^{45}Ca^{++}$. The efflux of $^{45}Ca^{++}$ was accelerated. Hog pancreatic kallikrein had similar effects. Scanning electron microscopy showed that the ruffle membrane of the macrophages was altered by both agents.

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## Effect of ionizing radiation on prostaglandin 15-OH-dehydrogenase (PGDH)

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Prostaglandins may contribute to some of the clinical symptoms of the radiation syndrome (Mennie, Dalley, Dineen & Collier, 1975). Raised levels were found in several tissues after whole body irradiation of mice (Eisen & Walker, 1976). The possibility that this increase is due to inactivation of PGDH has been investigated. Mice were killed at intervals after whole body exposure to 200-1,000 R of unfiltered x-radiation (230 kV, 15 mA, 140 R/min), and PGDH activity measured in the cytosol fraction of spleens, livers, kidneys, lungs and intestines. In the spleen, the fall in activity was prompt and extensive (up to 100%), and exceeded the loss in spleen weight and protein content. PGDH was only slightly depressed in the kidney, unchanged in the liver, and unchanged or even enhanced (after 1,000 R) in the lung. Four days after exposure to 700 R, PGDH activity in the jejunum was reduced ($P<0.05$), but not changed in duodenum and colon. These results agreed well with prostaglandin formation by isolated segments of gut, which was enhanced only in the jejunum.

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