Several attempts were made to elucidate the possible role of histamine, serotonin, leukotrienes C\(_4\) (LTC\(_4\)) and D\(_4\) (LTD\(_4\)), and prostaglandin E\(_4\) (PGE\(_4\)) as vascular permeability increasing factors involved in 48-hour homologous passive cutaneous anaphylaxis (PCA) in the mouse ear. Increased vascular permeability in the mouse ear caused by the mediator injection or PCA was assessed quantitatively by measuring the amount of extravasated dye. Present results strongly suggest that the most important mediator involved in mouse ear PCA is histamine and that serotonin also plays an important role in the increase of vascular permeability caused by PCA. Despite their potent vascular permeability increasing activity LTC\(_4\), LTD\(_4\) and PGE\(_4\) do not seem to be important.

Autoantibodies in Burkitt’s Lymphoma Patients from the Ugandan Prospective Study.

H. Mori*, G. M. Lenoir, R. M. Franklin

Pre- and post-syndrome sera from five Burkitt’s lymphoma patients who partook in the Ugandan prospective study were investigated with respect to autoantibodies. Neighbours and siblings of these patients served as controls and all of these groups were compared with sera from 50 Caucasian normal controls (CNC). Antibody levels significantly higher than those in CNC were found in all African groups for actin, desmin, vimentin, tubulin, keratin, laminin and collagen type I. Polyclonal B cell activation and high levels of antibodies to p. falciparum were also found. Whereas EBV infected B cells do produce autoantibodies, there is a potentiation of autoantibody formation as a result of infection with malaria, which seems to provide an independent trigger of polyclonal B cell activation.

Autoantibodies in Humans with Cystic or Alveolar Echinococcosis.

Hiroshi Mori*, Barbara Wernli, Niklaus Weiss, Richard M. Franklin

Sera from 16 echinococcosis patients were analyzed with respect to polyclonal B cell activation and autoantibody formation. At least 8 of the sera were from patients who were never in tropical countries and therefore their cases were not complicated by other parasitic diseases. In comparison with a group of 52 healthy controls, these patients had significant levels of antibodies to DNP and haemocyanin, indicators of polyclonal B cell activation. There were also significant differences between control and patient groups with respect to antibodies to dsDNA, histones, actin, vimentin, and desmin. This is the first report of autoantibodies in echinococcosis.