Joint alterations in the chronic phase of erysipelas polyarthritis in pigs have been studied widely (Sikes et al., 1966; Schulz et al., 1975; Trautwein et al., 1980). However, little morphological information is available concerning the initial acute phase of arthritis. In spite of extensive literature on this subject, the pathogenesis of polyarthritis has not been completely elucidated. Thus, the mechanism of polyarthritis with ankylosis in the final stage cannot be explained satisfactorily. Some speculate on polyarthritis, f.e. the slow progressive course and the morphologic changes, are comparable to human Rheumatoid Arthritis.

Material and Methods: In our experiment, 10 gnotobiotic piglets and 20 specific pathogenfree pigs, 3–25 months post infection were injected into the tail, joint with viable Erysipelothrix bacteria, type B, strain178. The gnotobiotic pigs were killed 30 days post infection and the specific pathogenfree pigs 3–25 months post infection. We want to demonstrate articular lesions in joints that were injected with bacteria. The joint particularly refers to lesions in the capital, elbow, stifle, and the right tail joint. Using light microscopy, transmission and scanning electron microscopy, we could see the development of cartilage lesions and the synovial lining cells (SLC) may be detected. In the secondary chronic phase we are able to demonstrate the destructive effect of the pannus which leads to additional macroscopic cartilage defects.

In the bacteraemic phase 1–3 days post infection, we observe large numbers of Erysipelothrix bacteria on the endothelial surface of vessels. These bacteria induce a vascular syndrome with severe disturbance of permeability. During this phase the bacteria penetrate through the vascular wall and invade the perivascular tissue. 2–3 days post infection the bacteria invade the superficial cartilage layers and at this stage the chondrocytes appear normal. Electron microscopically, the bacteria are located between collagen fibrils of the superficially loosened ground substance. 4–5 days post infection the bacteria seem to be in close contact with the perivascular tissue. At the same time polymorphonuclear leucocytes (PMNs) invade the cartilage matrix and are in close contact with chondrocytes. This cartilage surface becomes covered by a thin layer of fibrin. Using the scanning electron microscope we recognize under the fibrin layer the beginning aseptic necrosis. In this phase of the infection, the cartilage lesion becomes obvious. The main changes in the cartilage surface are associated with the fibrin layer and the fibrin matrix. In this phase the fibrin layer appears normal macroscopically. Severe cartilage alterations, which can be seen macroscopically, are associated with severe proliferation of SLC and invasive pannus.


Conclusion. The initial cartilage alteration, seen in experimental erysipelas arthritis is caused by direct action of bacteria, correlated with severe aseptic necrosis of chondrocytes and fibrin matrix. Important early changes are demarcation of collagen fibrils and degeneration of cartilage. In this early phase the cartilage surface appears normal macroscopically. Severe cartilage alterations, which can be seen macroscopically, are associated with severe proliferation of SLC and invasive pannus.