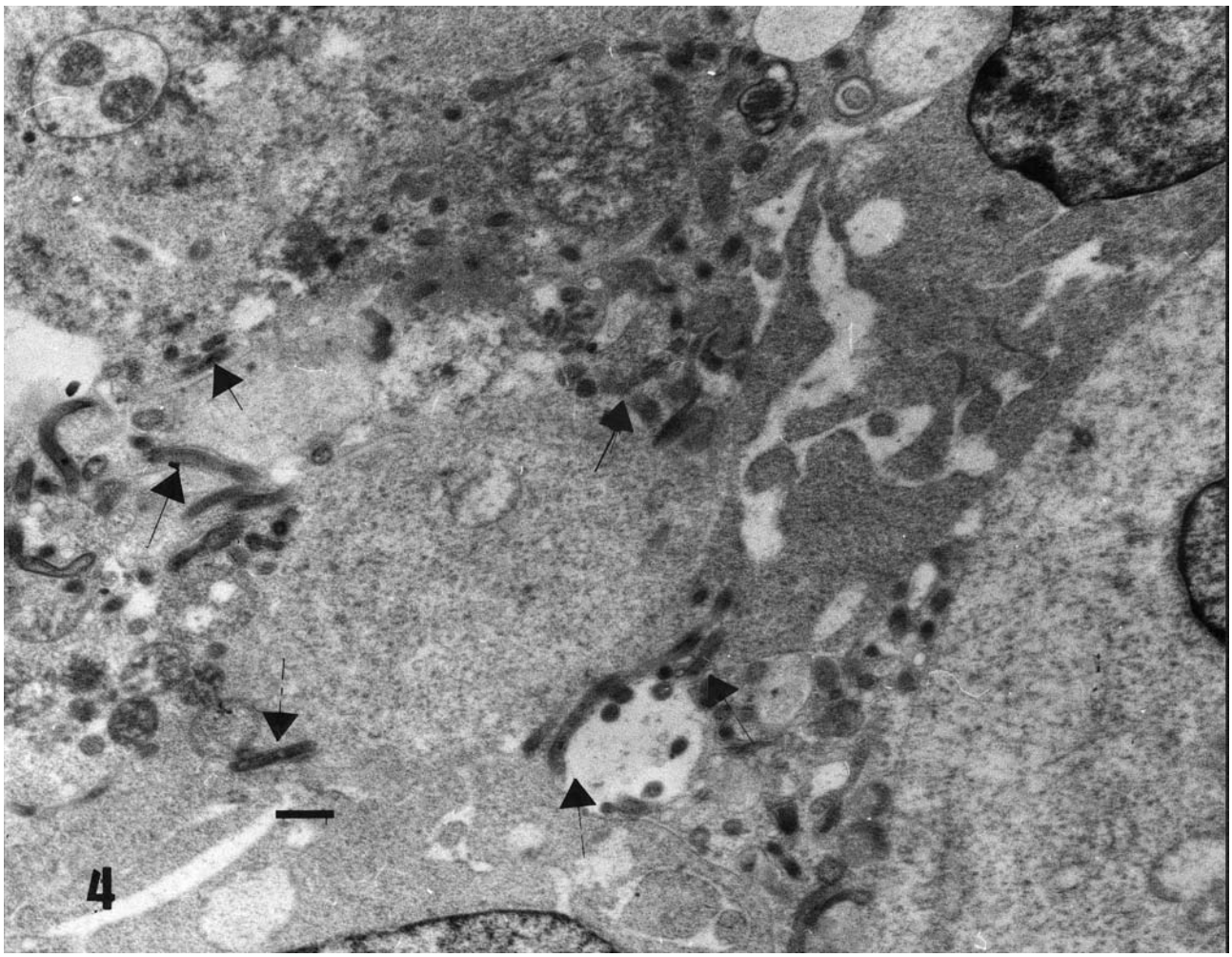

Fig. 5. Micrograph shows a transverse section of P3HR-1 cells clumped in syncytium-like formation and invagination or extension of borreliae into the cytoplasm. Some borreliae were inside (arrow) the host vacuoles (original magnification 14 500x, Ua-Lc). Bar = 600 μm.

of borrelial strains with P3HR-1 cells confirmed these observations. It has been found that *B. burgdorferi sensu stricto* cause lysis of B lymphocytes (Dorward et al., 1997). Epstein-Barr virus is considered to be a human B-lymphotropic virus (Strauss, 1993). Our present findings of the different behaviour and effect of different strains of *B. burgdorferi sensu lato* (Busch et al., 1996; Vasiliu et al., 1998) on replication of EBV in lymphoblastoid cells conform to some earlier studies. Norris et al. (1995) reported differences in virulence between low- and high-passaged borrelial strains. Distinct interactions have been described for various strains of Borrelia with cultured phagocytes, dependent on the presence of the outer-membrane proteins (Carrol and Gherardini, 1996). Different genospecies are associated with distinct clinical symptoms (Van Dam et al., 1993).

Neuroborreliosis as found by Marconi et al. (1999) should be associated with *B. garinii*-type 4. Primary infection by EBV occurs in early chilhood, is mostly asymptomatic (Roubalová et al., 1997) and serves for the development of specific antibodies, whereas primary infection with tick-borne Borrelia is mostly symptomatic but very often without development of specific antibodies, namely in the course of antibiotic treatment. After primary infection EBV is latently present in the organism life-long. Latent Lyme neuroborreliosis (Pfister et al., 1989) was also proved by detection of Borrelia in healthy skin and in the cerebrospinal fluids. Tai et al. (1994) demonstrated that Borreliae have the capacity to attach to different cells and that carbohydrate receptors are involved in their adhesion to eukaryotic cells. Coiling phagocytosis of Borreliae as a basic mechanism of borrelial internalization is controlled by actin polymerization (Linder et al., 2001).

It appears that the clinical outcome of borreliosis depends on a lot of factors also including the presence of viruses in the host, which can complicate the course of infection when they are activated. We have demonstrated that association and internalization of Borrelia by lymphoblastoid cells can cause induction of the lytic virus cyclus. *B. garinii* had a stronger inducing effect on EBV than *B. afzelii.*

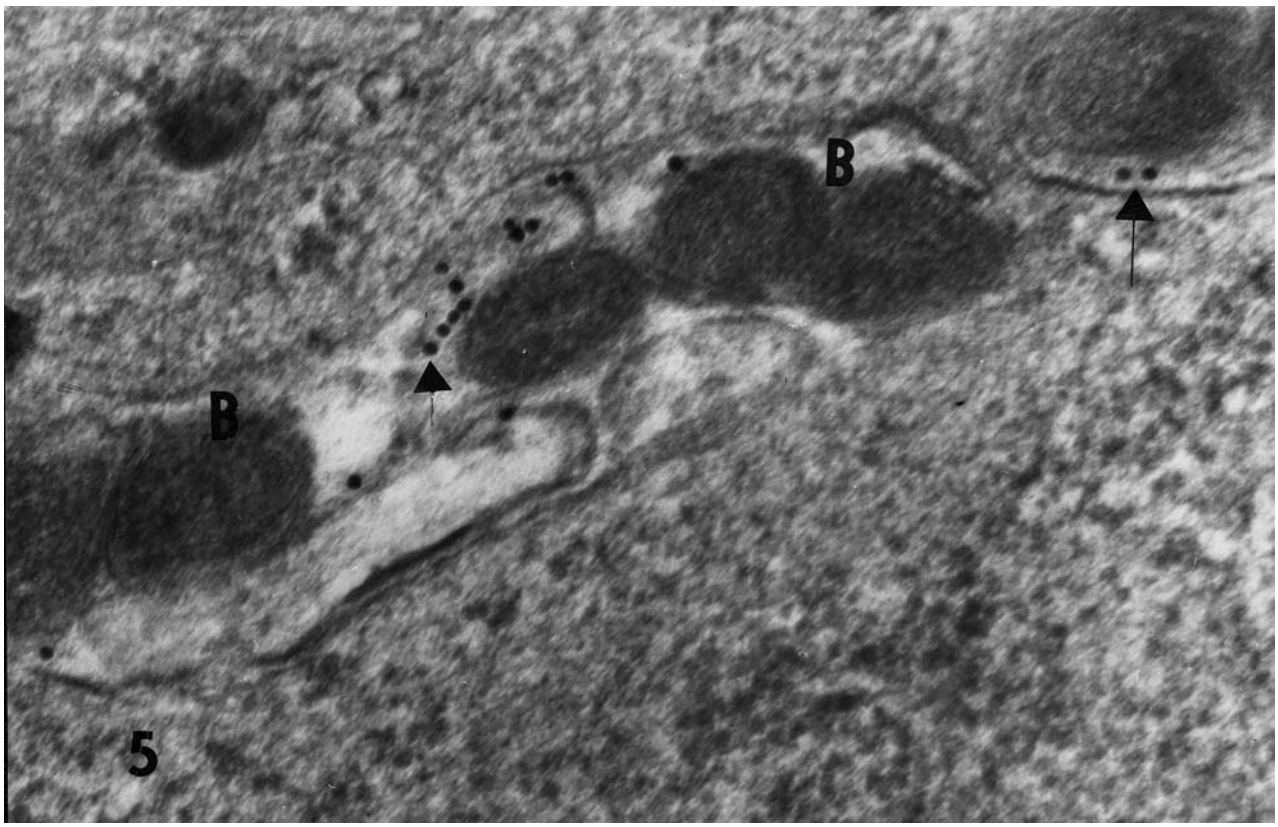

Fig. 6. Thin section micrograph of cross-sectioned *B. garinii* inside the invagination of the cytoplasmic membrane, which forms a tube-like structure protruding nearly to the nucleus of a P3HR-1 cell. EBV particles are marked by gold particles (5 nm) in the reaction with monoclonal anti-ZEBRA antibody (magnification 45 000x, Ua-Lc). Bar = 300 nm.

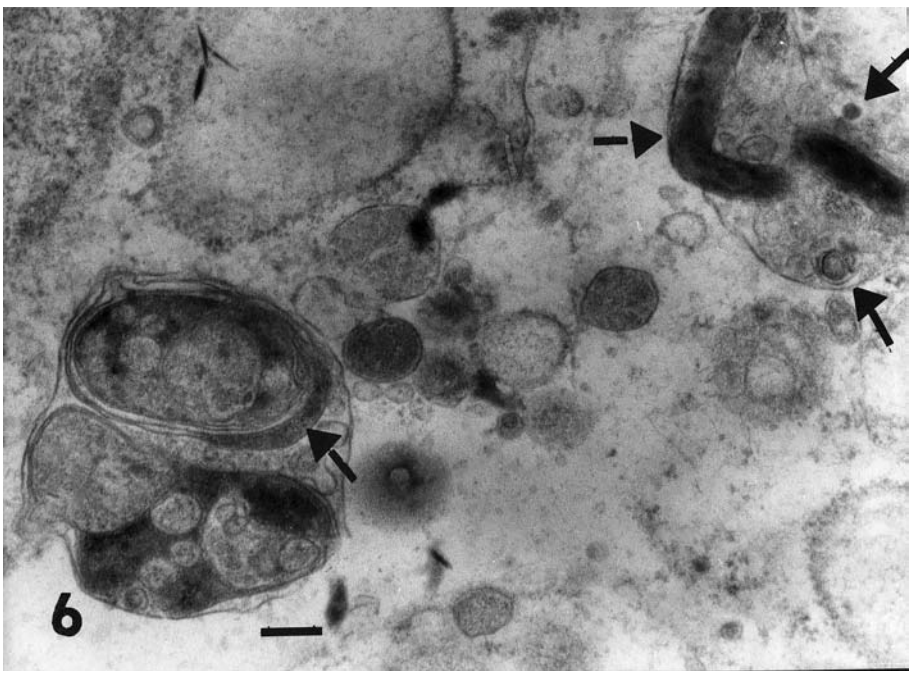

Fig. 7. Convoluted borreliae (arrow) and viruses (arrow) are visible inside the „cellule“ on day 4 of incubation (magnification 34 000x, Ua-Lc). Bar = 280 nm.

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