AMQ Results: BORRELIA BURGDORFERI LYME    9 Abstracts


TI - The prevalence of spirochete Borrelia burgdorferi sensu lato in ticks Ixodes ricinus and mosquitoes Aedes spp. within a selected recreational area in the city of Szczecin (Poland).

PG - 105-8

AB - The aim of this study was to determine the prevalence of spirochete Borrelia burgdorferi s.l. in ticks Ixodes ricinus and mosquitoes Aedes spp. within the Bukowa Forest, collected between 2000 and 2001. The study covered 215 ticks (193 nymphs and 22 adults) and 947 mosquitoes (female of the genus Aedes). Spirochetes of Borrelia burgdorferi s.l. were detected in the arthropods studied with the method of indirect immunofluorescence assay (IFA). Positive readings of the immunological reaction were stated in 17.7 % of the collected nymphs and adult forms of Ixodes ricinus, and in 0.8 % of mosquito females of the genus Aedes. The number of B. burgdorferi observed in a view field (400 x) of microscopic preparations of all infected mosquitoes and about 10 % of the infected ticks, ranged from 1-10. This number in 50 % of the nymphs was from 11-50 spirochetes. View fields of the preparations of the other 50 % of nymphs and adult forms featured more then 50 spirochetes. The observed low values of the prevalence and infection intensity of female mosquitoes Aedes spp. compared to ticks suggest that the former do not pose a serious epidemiological threat in the spreading of Lyme disease.

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Ixodes ricinus as a vector of Borrelia burgdorferi sensu lato, Anaplasma phagocytophilum and Babesia microti in urban and suburban forests.

AB - In the suburban and urban forests in the cities of Gdansk, Sopot and Gdynia (northern Poland), Ixodes ricinus ticks should be considered as the vector of pathogenic microorganisms that may cause significant diseases in wild and domestic animals and humans. These microorganisms include etiologic agents of Lyme disease, human anaplasmosis (HA) and babesiosis: Borrelia burgdorferi sensu lato, Anaplasma phagocytophilum and Babesia microti, respectively.

DNA extracts from 701 ticks collected in 15 localities were examined by PCR for the simultaneous detection of these 3 pathogens. Overall, 14% were infected with A. phagocytophilum followed by 12.4% with B. burgdorferi s.l. and 2.3% with B. microti. In total, the percentage of infected females (32.9%) was 2.4 times higher than in males (13.7%) and 3.2 times higher than in nymphs (10.3%). Among adult ticks (n = 303), 8.3% were dually infected with A. phagocytophilum and B. burgdorferi s.l., 2.0% with the agent of human anaplasmosis and B. microti and 0.3% with borreliae and B. microti.

Pre-treatment and post-treatment assessment of the C(6) test in patients with persistent symptoms and a history of Lyme borreliosis.

AB - It was recently reported that antibody to C(6), a peptide that reproduces an invariable region of the VlsE lipoprotein of Borrelia burgdorferi, declined in titers by a factor of
four or more in a significant proportion of patients after successful antibiotic treatment of acute localized or disseminated Lyme borreliosis.

The present study evaluated the C(6) test as a predictor of therapy outcome in a population of patients with post-treatment Lyme disease syndrome. The serum specimens tested were from patients with well-documented, previously treated Lyme borreliosis who had persistent musculoskeletal or neurocognitive symptoms. All of the patients had participated in a recent double-blind, placebo-controlled antibiotic trial in which serum samples were collected at baseline and 6 months thereafter, i.show $132#e. 3 months following treatment termination. In this patient population no correlation was found between a decline of C(6) antibody titer of any magnitude and treatment or clinical outcome.

Antibodies to C(6) persisted in these patients with post-treatment Lyme disease syndrome following treatment, albeit at a markedly lower prevalence and titer than in untreated patients with acute disseminated Lyme disease. The results indicate that C(6) antibody cannot be used to assess treatment outcome or the presence of active infection in this population.

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TI - Outer surface lipoproteins of Borrelia burgdorferi vary in their ability to induce experimental joint injury.

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AB - OBJECTIVE: To examine the ability of bacterial lipoproteins from the spirochete Borrelia burgdorferi to cause in vivo tissue injury (arthritis).

METHODS: Outer surface proteins (OSPs) from B burgdorferi were used in a rat model of antigen-induced allergic arthritis. Intra-articular challenge with recombinant OspA, OspB, and OspC in nonlipidated (peptide) and lipidated forms was performed in the left knee joint; the contralateral joint received buffer as control. Inflammation was monitored by technetium scintigraphy and histology.

RESULTS: Nonlipidated (peptide) OspA, OspB, and OspC did not induce arthritis; the only exception was polymerized OspA, which was tested in preimmunized rats. Lipidated OspA from 2 different strains and lipidated OspC induced severe arthritis, whereas lipidated OspB failed to induce injury. A synthetic analog of the OSP lipid modification, lipopeptide Pam(3)Cys-Ser-Lys(4)-OH, either alone or coupled to bovine serum albumin, also failed to induce injury. Injury did not develop in control groups that were given the appropriate buffers or lipopolysaccharide. This showed that lipidated borrelial OSPs can be potent arthritogens but vary greatly with respect to their injury-inducing potential. The possession of a lipid modification is essential but is not sufficient to render an OSP arthritogenic.

CONCLUSION: This is the first study to demonstrate that individual lipoproteins from B burgdorferi can induce experimental joint injury in vivo. These results may help elucidate the pathogenesis of Lyme arthritis and, above all, underline the importance of bacterial lipoproteins as major virulence factors.
Central Nervous System Lyme Disease.

A nervous system infection with Borrelia burgdorferi frequently causes meningitis and rarely causes encephalomyelitis. Altered cognitive function also can occur in the absence of central nervous system infection. Recently developed serodiagnostic tools, such as the C6 assay, and appropriate use of Western blotting, promise to improve diagnostic accuracy. Treatment trials have demonstrated the efficacy of relatively brief courses of oral antimicrobial agents, even in peripheral nervous system infection and meningitis. Several well-performed studies have clearly shown that prolonged antimicrobial treatment of "post-Lyme disease" is ineffective. Diagnosis and treatment of Lyme disease continue to improve.

MyD88 plays a unique role in host defense but not arthritis development in Lyme disease.

To assess the contribution of TLR signaling in the host response to Borrelia burgdorferi, mice deficient in the common TLR adaptor protein, myeloid differentiation factor 88 (MyD88), were infected with B. burgdorferi. MyD88-deficient mice harbored extremely high levels of B. burgdorferi in tissues when compared with wild-type littermates and greater amounts of spirochetes in tissues than TLR2-deficient mice. These findings suggest that, in addition to TLR2, other MyD88-dependent pathways play a significant role in the host defense to B. burgdorferi.

MyD88(-/-) mice maintained the ability to produce Abs directed against B. burgdorferi. Partial clearance of spirochetes was evident in long term infection studies and immune sera from MyD88-deficient mice were able to protect naïve mice from infection with B. burgdorferi. Thus, the acquired immune response appeared to be functional in MyD88(-/-) mice, and the inability to control spirochete numbers was due to a failure of cells involved in innate defenses. Although macrophages from MyD88(-/-) mice responded poorly to Borrelia sonicate in vitro, MyD88(-/-) mice still developed an inflammatory arthritis after infection with B. burgdorferi characterized by an influx of neutrophils and mononuclear cells. The findings presented here point to a dichotomy between the recruitment of
Borrelia burgdorferi genes selectively expressed in ticks and mammals.

Lyme disease is a tick-borne infection caused by the spirochete Borrelia burgdorferi. Recent studies have focused on how the Lyme disease bacterium overcomes the challenges faced by an organism that depends on a vector-borne life style. These studies indicate that the spirochete expresses different surface proteins at different stages of its life.

Here, Aravinda de Silva and Erol Fikrig review the evidence for differential gene expression and discuss the implications of these findings for the Lyme disease vaccine that is currently being tested in human trials.

Borrelia burgdorferi and the macrophage: Routine annihilation but occasional haven?

Borrelia burgdorferi, the agent for Lyme disease, has a typical pattern of bacterial interaction with phagocytes: attachment, stimulation of release of inflammatory mediators and, in most cases, ingestion and killing. Spirochetes are killed extracellularly by antibody plus complement via the classical pathway, as well as by phagocytes through apparently nonoxidative means. Yet rare persistent spirochetes (mutants?) have been identified both in patients' tissues and in cells.
Ruth Montgomery and Stephen Malawista here ask: are some Borrelia wolves in sheeps' clothing, evading macrophage anti-microbial action?

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**TI**  - Risk of Borrelia burgdorferi infection in western Switzerland following a tick bite.

AB  - The aim of this study was to define the risk of developing Lyme borreliosis after a tick bite. A survey was conducted from 1993 to 1995 in the western part of Switzerland in a group of patients who presented for treatment of a recent tick bite. Only patients with negative serological tests (enzyme-linked fluorescent assay screening test, and IgG and IgM immunoblots) at the first consultation and for whom a second blood sample was available 2 months later were included in the study. Of the 376 patients included, 266 had no clinical manifestation (group 1) and 110 had a small local cutaneous reaction (<2 cm) (group 2). The tick was available for 160 patients. Seroconversion was observed in 4.5% of 376 patients, 3.4% in group 1 and 7.2% in group 2. Typical erythema migrans, confirmed by seroconversion, was observed in three of 376 (0.8%) patients, while five of 376 (1.3%) patients developed a skin lesion without seroconversion. No other clinical manifestation of Lyme borreliosis was observed among these 376 patients. *Borrelia detection in ticks did not correlate significantly with the risk of Lyme borreliosis. In conclusion, the risk of developing Lyme borreliosis in western Switzerland after a tick bite is low, and therefore, prophylactic antibiotics are not required.*

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