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Review article

Interactions between *Ixodes* ticks (Ixodidae) and selected bacterial tick-borne pathogens

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ABSTRACT. In Europe, ticks are particularly important vectors of pathogens known as tick-borne pathogens (TBP). TBP can influence hosts, including domestic animals and humans as well as ticks. This review focuses on interactions between hard ticks of the *Ixodes* genera and medically and veterinary significant bacterial pathogens i.e. *Borrelia burgdorferi* s.l., *Anaplasma* spp., and *Rickettsia* spp. The interactions between ticks and bacteria include among others the impact on gene expression and tick behaviour. Infection with TBP may influence tick salivary proteins and midgut receptors. Infection with *B. burgdorferi* s.l. changes the bahaviour of the tick allowing them for longer questing and increased mobility, while *A. phagocytophilum* increases survival in low temperatures by upregulating the expression of antifreeze glycoprotein (IAFGP) and effect on molting success. Whereas *Rickettsia* spp. increases ticks' attraction towards the 900 MHz electromagnetic field.

Keywords: ticks, tick-borne pathogens, interactions, *Borrelia burgdorferi* s.l., *Anaplasma phagocytophilum*, *Rickettsia* spp.

Introduction

Ticks are among the most important vectors transmitting pathogenic microorganisms (tick-borne pathogens – TBP) with an important impact on human and animal health [1–3]. Due to an increase in cases of tick-borne diseases (TBD), ticks and TBP (viruses, bacteria, and protozoa) are of worldwide interest [4]. In the Northern Hemisphere, borreliosis (Lyme disease), caused by bacteria from *Borrelia burgdorferi* sensu lato complex, is the most prevalent tick-borne disease [5]. Other frequently identified tick-borne bacterial pathogens are *Anaplasma phagocytophilum* and *Rickettsia* spp. [5,6].

While analysing relationships between pathogens, vectors, and their hosts, most attention is drawn toward either vector-host or pathogen-host interactions [7,8]. Interactions between pathogens and ticks are less frequently studied, although they are equally important. Knowledge of tick pathogens is relatively new: *Rickettsia* spp. was discovered for the first time in 1925 in soft ticks [9,10]. In 1993 MacLeod and Gordon [11] reported the presence of a tick-borne fever agent (later named *Anaplasma phagocytophilum*) in *Ixodes ricinus*. Some pathogens were described much later, such as *Borrelia burgdorferi* in *Ixodes scapularis* in 1982 [12]. Nonetheless, the interaction between ticks and pathogens has a long history, e.g., a study in Great Britain [13] proved the presence of *Borrelia* spirochaetes in ticks from a museum collection from the XIX century. Poinar [14] in his study suggests that *Borrelia*-like bacteria are present in the gastrointestinal tract of *Ambylomma* spp. tick from amber dated as 15 to 20 million years. The history of tick–pathogen interactions can be now traced back thanks to molecular methods. Analysing genetic differences between bacterial species is helpful in understanding the evolution of these species [15]. Long coexistence and interactions worked in both ways, the pressure of the environment and the presence of the pathogens in the arthropod body led to phenotypic changes in the vector and that became an evolutionary force for pathogens and led to their speciation [15,16].

During long co-evolution pathogens developed various mechanisms that help them survive in ticks and increase their chances of transmission. Interactions between pathogens and ticks happen both on molecular and behavioral levels. However, our knowledge about the interaction between ticks and tick-borne pathogens is still patchy [16]. The aim of this review is to discuss interactions between ticks and tick-borne bacterial pathogens (*Borrelia burgdorferi* s.l., *Anaplasma* spp., and *Rickettsia* spp.).

Borrelia burgdorferi sensu lato

Gram negative bacteria of the genus *Borrelia* are vector-transmitted spirochaetes that cause disease in humans and animals. *Borrelia* species can be divided into two groups: Lyme disease (Lyme borreliosis – LB) and relapsing fever (RF) [17]. LB group is known to be transmitted by hard ticks, whereas the RF group is typically associated with soft ticks [15]. However, *Borrelia miyamotoi* from the RF group firstly identified in the hard tick *Ixodes persulcatus* [18], is increasingly detected in other ticks species including *Ixodes ricinus* [19,20]. Lyme disease is the most commonly diagnosed tick-borne disease in the Northern Hemisphere [21–23]. It is caused by bacteria of *Borrelia burgdorferi* sensu lato (s.l.) complex. The complex includes 22 genospecies out of which 11 are present in Europe [24]. Five out of them: *B. afzelii*, *B. garinii*, *B. burgdorferi* sensu stricto (s.s.), *B. bavariensis* and *B. spielmanii* are proven to cause borreliosis in humans [25], and *Borrelia lusitaniae* can cause borrelia-like symptoms [26]. In Europe *B. garinii* and *B. afzelii* are a main cause of borreliosis in humans, while in the United States *B. burgdorferi* s.s. is responsible for most cases [27], and in Asia *B. garinii* [28]. The first symptom most commonly associated with Lyme disease is erythema migrans, although it is present only in 80% of cases in the early stage of the disease [29]. As the disease progresses it can lead to a broad range of symptoms.

In Europe, there are noted around 230 000 new cases of borreliosis each year [30], whereas in the United States annual number is 30 000, however, it is considered that the real number of cases can be ten times higher [31,32]. In recent years (2016–2019) in Poland around 50 cases per 100 000 inhabitants were reported annually [33]. The lower number of cases reported worldwide in the years 2020–2021 is probably related to limited access to health care and not to an actual decrease in the incidence of the disease [34–36].

In Europe *B. burgdorferi* s.l. spirochaetes are transmitted mainly by *Ixodes ricinus* ticks, and in Asia by *Ixodes persulcatus* [22]. In the US the main vector is *Ixodes scapularis*, with the exception of the West Coast, where spirochaetes are transmitted by *Ixodes pacificus* [22]. Reservoir of *B. burgdorferi* s.l. are small mammals and birds as well as lizards [37]. In the US, the prevalence of *B. burgdorferi* s.l. in ticks is usually in a range from 3 to 15%, depending on the region [38–40]. Also in Europe, the prevalence depends on the region and is usually less than twenty percent, with higher rates in central Europe [41,42]. In Poland, according to the literature data, it can be from few to a dozen percent [43–45]. In Europe most frequently identified genospecies were *B. afzelii* and *B. garinii* [46], while in the US *B. burgdorferi* s.s. and recently, since 2013 in the Upper Midwest *B. mayonii* [23,47].

Ticks acquire *B. burgdorferi* s.l. during feeding when spirochaetes enter the tick body with blood. Firstly spirochaetes stay in the tick midgut and later are transferred to salivary glands in order to invade the new host. Hence most of the interactions between ticks and *B. burgdorferi* s.l. will happen either in the tick midgut or in salivary glands. However, there are some interactions that are more complex and cannot be classified into any of those groups. Most of the interactions are based on changes in the expression of certain genes of proteins in various parts of the tick body as well as the reaction of those proteins with bacterial cells. The presence of spirochaetes in ticks can change the expression of plenty of proteins such as Salp25D, Salp15, Salp20, TSLPI, the tick-histamine release factor tHRF, ISAMP, ISDLP, and receptors TROSPA and Tre31 [6,48–54].

Borrelia burgdorferi s.l. tick saliva-associated interactions

Components of tick saliva are important for the feeding process. They take part in vasodilation and prevent blood cell aggregation. They also minimize irritation and inflammation within the host body [50] and interact with the host immune system [55]. Important compounds of tick saliva are salivary proteins known as Salp. As far as it is known they play a role in suppressing the host immune response during tick feeding and interact with various

immune system elements. Although in the tick saliva, more components such as TSLPI, tHRF, ISAMP are present, recently most research focuses on Salp15 and Salp20.

Salp15 which was originally identified in *Ixodes scapularis* is also present in the saliva of other Ixodid ticks [54]. Expression of this 15 kDa protein is upregulated upon *B. burgdorfer*i s.l. infection. Salp15 is crucial for spirochaetes' ability to transmit and attack vertebrate hosts. It presents strong immunosuppressive activity weakening the host's response to pathogens. Salp's ability to bind to the receptor CD4 may lead to the inactivation of lymphocytes T CD4(+), stop the proliferation of the T-cells, and impedes IL-2 (interleukin 2) secretion [54]. Additionally, it can connect to OspC (Outer membrane protein C) of *Borrelia* spp. and protect bacteria from cell lysis by disrupting the formation of the MAC (Membrane Attacking Complex) in the bacterial membrane. Spirochaetes connected to Salp15 are more likely to survive in the host, multiply and spread to new hosts [48].

Another important saliva protein is Salp20, which expression is also upregulated by *B. burgdorferi* s.l. infection. Similarly to Slap15 it can interact with the host immune system. Salp20 shows 83% amino acid sequence similarity to Isac (*Ixodes scapularis* anitcomplement protein) and similarly has the ability to impair complement activation. The main mechanism works by blocking the alternative pathway of complement activation and that protects spirochaetes from lysis due to component activation [6]. It is also responsible for slowing down the inflammatory reaction in the host, which also helps *B. burgdorferi* in surviving in the host and protects bacteria from cell lysis [6,50].

Slp25D is a glutathione peroxidase, which expression is upregulated during tick feeding. It is responsible for the reduction of reactive oxygen species, which are the result of neutrophil activation in the host body, due to injury caused by a tick [6]. That activity protects spirochaetes while colonization of a tick midgut [6,49].

Tick Salivary Lectin Pathway Inhibitor known as TSLPI has the ability to inhibit the lectin pathway of complement activation by binding to mannose-binding lectin. That disrupts complement activation and therefore protects spirochaetes from phagocytosis. This mechanism protects *B. burgdorferi* s.l. during transmission and invasion of a new host [53].

A protein called tHRF, short for tick histamine release factor, is also produced in salivary glands. Production of this protein is upregulated by *B. burgdorferi* s.l. presence. Thanks to high homology to natural vertebrate histamine release factors, it can bind to basophils and stimulate histamine release [6,52]. This happens in the later stage of tick feeding, and increasing the blood flow makes it easier for the tick to feed, and also helps *B. burgdorferi* s.l. in transmission [6,16,52].

Ixodes scapularis antimicrobial peptide (ISAMP) presents activity against both Gram-positive and Gram-negative bacteria (higher activity is shown against Gram-negative). Most likely the role of ISAMP is to protect ticks from pathogens that might be ingested during feeding. Expression of this protein is upregulated during infection with *B. burgdorferi* s.l. [6,51].

Interactions of *Borrelia burgdorferi* s.l. in the tick midgut

During feeding on the reservoir host tick acquires *Borrelia* spirochaetes from the vertebrate body. The first place where spirochaetes travel to inside the tick body is their gut. The main challenges in the midgut for *B. burgdorferi* s.l. are to avoid the tick's immunology system response. In order to survive and then later transfer to salivary glands spirochaetes interact with various different components. Main interactions in tick midgut happen between tick receptors TROSPA (tick receptor for OspA) and surface proteins of *Borrelia* spirochaetes, but there are other receptors and components as well, such as Dps-like proteins or GST.

After tick feeding spirochaetes stay in the tick midgut. Until the next tick feeding bacteria experience a lack of nutrients. In order to survive, *Borrelia* spirochaetes produce Dps-like proteins (DNA-binding protein from starved bacteria). Dps is a homolog of bacterial ferritin, a protein responsible for acquiring Ferrum for bacteria. Dps is known to help *B. burgdorferi* s.l. to survive periods of hunger between tick feeding [56].

Outer surface proteins of *B. burgdorferi* s.l. are particularly important for survival in the tick midgut and transmission to the new host. The most important for infection of ticks are OspA and OspC. Shortly after the next tick feeding spirochaetes, already present in the midgut, start to express on their surface OspA. This protein is responsible for binding with the TROSPA receptor. The connection

between OspA and TROSPA keeps bacteria in the tick midgut and allows bacteria to proliferate [56]. About 72 h later the expression of OspA is lowered while the expression of OspC is upregulated. OspC destroys the OspA–TROSPA bond, which leads to the transport of the spirochaetes to salivary glands, from where they can reach the new host [6,55]. Expression of the TROSPA receptor in tick gut depends on the presence of *B. burgdorferi* s.l., it is upregulated by the spirochaetes and is lowered after the next feeding. Bonding to the TROSPA is crucial to the survival of spirochaetes in the tick [56].

Another receptor in the tick midgut is Tre31, which binds to *Borrelia burgdorferi* surface protein (BBE31). Similarly to TROSPA, Tre31 expression is upregulated during *B. burgdorferi* s.l. infection. Expression of BBE31 is the highest while bacteria are in tick midgut. This mechanism also takes part in the survival of the spirochaetes in the midgut and later transports them to salivary glands [6].

ISDLP (*Ixodes scapularis* dystroglycan-like protein) is also responsible for the transport of the spirochaetes from the midgut to salivary glands. Expression of this protein, on the epithelial cells, is upregulated upon feeding and infection. It can bind *Borrelia* spp. and provide transmission of the bacteria from the midgut to the hemocoel [57].

The presence of *Borrelia* spirochetes in the tick midgut can generate reactive oxygen species, which lead to upregulated GST (glutathione S-transferase) expression in ticks. GTS has antioxidative properties [59].

Borrelia burgdorferi s.l. and tick behaviour

It is a well-known fact that pathogens have the ability to adjust the behaviour of their hosts in order to maximize the chances of finding a new host.

During non-feeding time, ticks are especially susceptible to water loss. The best conditions for ticks are 86 to 96% of relative humidity [60]. While questing in too dry conditions, ticks to avoid dehydration tend to move to lower areas such as the litter layer [60]. Herrmann and Gren [60] proved that ticks infected with *B. burgdorferi* s.l. can survive longer in dry conditions and even prefer lower relative humidity (around 70–75%). Thanks to that ticks can spend longer periods of time on higher vegetation which is beneficial for spirochaetes because of the greater chance for transmission to the new host. The reason for that might be the higher fat content in *Borrelia* infected ticks compared to non-infected ones (up to 12% more fat). Higher fat content can result from bigger meals or decreased activity of infected ticks, the reasons are still unknown [60]. Additionally, it was observed that ticks infected with *B. burgdorferi* s.l. have a longer lifespan, which also contributes to higher chances for transmission of the spirochaetes [60].

It was also shown that *I. ricinus* ticks infected *B. burgdorferi* s.l. present decreased mobility in comparison to non-infected ones [16,61].

Infected with *Borrelia* spp. ticks can differ when it comes to questing height [62]. Infected nymphs have a tendency to climb higher and show increased phototaxis, whereas infected adults are less active and prefer lower heights [16,62]. Reasons for these mechanisms remain unknown but it is suspected that at least in nymphs it can be adaptive, as the change in behaviour increases the chances for *Borrelia* spp. transmission [16,62].

Anaplasma spp.

Species that belong to the *Anaplasma* genus are intracellular pathogens and are transmitted through blood, either by vector or by direct contact [6,63]. They are all intracellular obligate pathogens [64]. Most significant from a medical point of view is *Anaplasma phagocytophilum*, which can cause human granulocytic anaplasmosis (HGA). It was first discovered as a human pathogen in 1990, but it has been known in veterinary medicine since 1932 [65,66]. In HGA bacteria attack neutrophils, which can be seen as small aggregates inside those granulocytes in the blood smear [66,67]. Symptoms of HGA include fever, headache, myalgias, rigours, nausea, but it can be completely asymptomatic as well [64]. The number of cases of HGA in Europe is relatively small, around 300 in total [68]. Whereas in Northern America a steady increase is observed with around 5000 cases yearly [68]. Reservoir of the *A. phagocytophilum* consists of free-living small animals, mostly mammals, such as mice, rats, or hedgehogs, but also big like deers, roe-deers, as well as livestock [63,68,69]. Other species from this genus such as *Anaplasma marginale*, *A. bovi*s or *A. ovis* can cause ehrlichiosis in cattle *(A. ovis* also in sheep), *A. platys* in dogs [6]. In Europe, the main vector of *Anaplasma* spp. is *Ixodes ricinus*, in North America *Ixodes scapularis* and *Ixodes pacificus* [64,69]. The estimated prevalence of *Anaplasma* spp. in ticks in the USA varies from a few to a dozen

percent [70]. Across Europe, the prevalence of *Anaplasma* spp. also differs depending on the region [68]. In Poland, it in can be from a few to over 20% [70–73]. *Anaplasma* spp. interacts with tick saliva components (Salp16, P11) proteins in the tick midgut (CG8, T2, SUB) and also has the ability to manipulate tick behaviour and can affect molting

Anaplasma spp. tick saliva-associated interactions

Through metabolic changes that happen because of the presence of *Anaplasma* spp. in the tick expression of Salp16 in the salivary glands is upregulated [74]. Through activation of the kinase IPI3K and kinase IPAK1 *Anaplasma* spp. leads to actin phosphorylation. Globular actin, which is the result of that process, binds to polymerase RNAPII and TBP protein (TATA – box binding protein). That connection leads to a change in expression [75]. Salp16 mainly impairs neutrophil migration to the place where the tick is feeding and stops them from entering the tick [74]. Salp16 does not interact directly with bacteria in the host body and does not change their ability to infect ticks. It is probably necessary for the transition from tick midgut to its salivary glands, but the nature of that mechanism remains unknown [76].

Salivary gland protein P11 plays an important role in the migration of *Anaplasma* spp. from tick midgut to salivary glands. Expression of P11 is upregulated upon *A. phagocytophilum* infection. It also helps bacteria in infection of the haemocytes in the tick midgut [6,77]. The direct mechanism of P11 activity remains still unknown [77].

Interactions of *Anaplasma* spp. in the tick midgut

The presence of *A. phagocytophilum* lowers the expression of the spectrin alpha chain, known as CG8 in the salivary glands and increases in the tick midgut. CG8 is responsible for binding actin that allows for the connection of the cytoskeleton with the plasma membrane. That mechanism determines the shape of the cell and organelle organization. Increased expression of the CG8 in the tick midgut and cytoskeleton rearrangements are necessary for bacteria to be able to infect midgut cells and from there transit to salivary glands [78].

T2 is a voltage-dependent anion-selective mitochondrial channel that plays a role in mitochondria-mediated apoptosis. Upon *A. phagocytophilum* infection expression of T2 is downregulated both in the salivary glands and midgut, and that results in inhibition of the apoptosis. That mechanism protects bacteria and increases their chances of survival in the tick [78].

Subolesin (SUB) known as 4D8 is a protein present in many tick species, for the first time, it was discovered in *Ixodes scapularis* [6,78,79]. It is expressed in tick salivary glands, midgut, and reproductive organs and is necessary for the development of those body parts [79]. It is also responsible for the activation of tick immune response against bacteria [78]. Upon *Anaplasma* spp. infection expression of SUB is downregulated in nymphs and upregulated in females [6,78].

Anaplasma phagocytophilum and tick behaviour

The presence of *A. phagocytophilum* in a tick increases expression of the *Ixodes scapularis* antifreeze glycoprotein (IAFGP). IAFGP protects ticks from freezing temperatures [80]. Neelakanta et al. [80] proved that ticks infected with *Anaplasma* spp. have higher chances to survive in low (–20°C) temperatures, compared to non-infected ticks and were able to continue being mobile for longer in cold temperatures than non-infected ones. The exact mechanism of IAFGP activity is unknown, and authors [80] proposed three possible scenarios: IAFGP can prevent ice crystals from forming inside the tick, might stop denaturation of macromolecules and membrane disruption, or it can act together with other protective mechanisms such as accumulation of alcohol or glycerol in the cells.

Anaplasma phagocytophilum effect on molting success

Infection with *A. phagocytophilum* may affect the molting success of larvae ticks. According to Ross and Levin [81] the molting success strongly depends on the strain of *A. phagocytophilum*. Authors tested *I. scapularis* larvae molting success after feeding on *A. phagocytophilum* infected mice. For some tested strains they observed decreased molting success correlated to the prevalence of bacteria in the ticks which can be explained either by higher bacterial load or individual characteristics of the bacterial strain. For most of the strains in the study, authors observed no decrease in molting

abilities, or in some cases increase in molting success correlated to higher pathogen prevalence. Authors suggest that in that case presence of *A. phagocytophilum* in the host can possibly increase the quality of the host's blood thus increasing the molting success of ticks fed on infected mice. Overall infection with *A. phagocytophilum* can affect molting success both ways, depending on the bacterial strain.

Rickettsia spp.

Rickettsia spp. are obligate intracellular pathogens that can cause various diseases such as spotted fevers or epidemic typhus [82]. Within the genus of *Rickettsia* species are divided into four groups depending on their properties and pathogenicity: Typhus Group (TG), Spotted Fever Group (SFG), Transitional Group (TRG), and Ancestral Group (AG) [82]. In TG group vectors are either lice (for *Rickettsia provazekii*) or fleas (for *Rickettsia typhi*), in SFG vectors for all species are ticks. In TRG vectors could be ticks, mites, or fleas depending on the species of *Rickettsia*, and in AG group vectors are only ticks [83,84]. The SFG groups more than half of known *Rickettsia* species which are transmitted by different species of ticks. The most frequently identified species from this group in Poland is *Rickettsia helvetica* others are *Rickettsia monacensis*, *Rickettsia slovaca*, and *Rickettsia raoultii* vectored by *I. ricinus* and *Dermacentor reticulatus* [83]. The infection rate in ticks in Poland is around a dozen percent [85]. *Rickettsia* spp. are also widely identified as ticks' symbionts, but the understanding of the nature of this interaction remains unknown [86]. Interactions between ticks and *Rickettsia* spp. are not very well known compared to ticks' interactions with *Anaplasma* or *Borrelia* [16].

A recent study [87] shows that *I. ricinus* ticks infected with *Rickettsia* spp. are about two times more attracted by electromagnetic radiation of 900MHz than ticks free of these bacteria. For ticks coinfected with *Borrelia* spp. and *Rickettsia* spp. attraction by electromagnetic radiation is also stronger compared to non-infected ticks. The exact mechanism of this interaction is still unknown [87].

Conclusion

The overall knowledge about tick-pathogen interaction is still growing. Some of the interactions are well known and described, although most already known interactions need to be researched further. A lot is already known in the case of *B. burgdorferi* s.l. interactions with ticks but in regards to other TBP there are some gaps in the knowledge and understanding of the interactions. As it is nearly impossible to discuss tick-pathogen interactions in separation from interactions with tick microbiota, or host body, new research takes a more holistic approach to the problem. Understanding these interactions is an important step in tackling the problem of tick-borne diseases. It is strongly believed that interactions between pathogens and ticks play a crucial role in the transmission of the pathogens, due to changes in the vector behaviour or their metabolisms.

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