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Review

Thirty years of tick population genetics: A comprehensive review

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ABSTRACT

Population genetic studies provide insights into the basic biology of arthropod disease vectors by estimating dispersal patterns and their potential to spread pathogens. In wingless vectors, such as ticks, gene flow will be defined in large part by the mobility of their hosts. However, tick behaviors and life cycle strategies can limit their dispersal even on highly mobile hosts and lead to an increase in genetic structure. In this review we synthesize the published literature from three decades of tick population genetic studies. Based on studies from 22 tick species (including representatives from *Amblyomma*, *Bothriocroton*, *Dermacentor*, *Ixodes*, *Ornithodoros*, and *Rhipicephalus*), observed levels of population genetic structure in ticks varied from no structure to very high levels. In about half of the species (including representatives from *Amblyomma*, *Bothriocroton*, *Dermacentor*, and *Ornithodoros*), tick genetic structure appeared to be determined primarily by the movement capacity of hosts, with low gene flow observed in ticks that use smaller bodied less mobile hosts and high gene flow in ticks using highly mobile hosts. In a number of other species (primarily from *Ixodes*, *Ornithodoros*, and *Rhipicephalus*), behavioral limitations to gene flow appeared to result in greater genetic structure than expected based upon host movement capability alone. We also discuss the strengths and limitations of genetic markers and their applicability to ticks and suggest possible analyses when planning population genetic studies for ticks.

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1. Introduction

In 1995, Tabachnick and Black made an appeal for molecular genetic studies of arthropod vectors that would fill gaps in our understanding of dispersal and gene flow in vector populations (Tabachnick and Black, 1995a). The importance of genetic variation in arthropod vectors was also reviewed by Gooding around this time (Gooding, 1996), with a focus on the vector competence of genetically structured mosquito populations. Until then, few studies specifically focused on tick population genetics had been undertaken (Bull et al., 1984; Hilburn and Sattler, 1986b; Wallis and Miller, 1983) but it was already obvious that dispersal and pathogen specificity for tick subpopulations could be important considerations for disease control efforts (Kubasu, 1992; Sattler et al., 1986). In more recent works, the application of genetics to tick evolution and host specialization has been reviewed (Barker and Murrell, 2002; McCoy et al., 2013) and a discussion of the relationship of tick genetic structure to human disease epidemiology has been published by McCoy (2008). These reviews have focused on central issues in vector biology, including the use of molecular tools to identify cryptic species, the importance of quantifying population genetic variation, and the role of vector dispersal on epidemiology and genetic structure of vector-borne pathogens. Our goal in this review is to distill major conclusions drawn from studies of tick population genetics and contrast the biological scenarios that may influence the patterns of genetic variation observed in ticks.

Investigating tick genetics is important because among arthropods these parasites vector the widest variety of pathogens known, leading to public health issues and economic losses in livestock production (Hill and Wikel, 2005; Jongejan and Uilenberg, 2004; Pagel Van Zee et al., 2007; Parola and Raoult, 2001). Only mosquitoes are of greater importance as vectors of human pathogens (Hill and Wikel, 2005; Mixson et al., 2006; Sonenshine, 1991). Ticks are capable of transmitting disease-causing protozoa, viruses, and bacteria. Babesiosis (*Babesia* spp.) and theileriosis (*Theileria* spp.) are the two main types of protozoan parasites transmitted by ticks. These pathogens affect cattle (*Babesia bovis*, *Babesia bigemina*, *Babesia divergens*, *Babesia major*, *Babesia ovata*, *Theileria parva* and *Theileria annulata*), humans and rodents (*Babesia microti*), and many other mammals; the discovery and phylogenetic description of these apicomplexan protozoans is still underway (Schnittger et al., 2012). Among the arboviruses (arthropod-borne viruses) transmitted by ticks are the flaviviruses, which cause tick-borne encephalitis (TBE), and *Orbivirus* spp., the etiological agent of Colorado tick fever. Important bacterial disease agents transmitted by ticks include Lyme disease (*Borrelia burgdorferi*), Rocky Mountain spotted fever (*Rickettsia rickettsii*), Boutenneuse fever (*Rickettsia conorii*), Q-fever (*Coxiella burnetii*), ehrlichiosis (*Ehrlichia* spp.), anaplasmosis (*Anaplasma* spp.), relapsing fever (*Borrelia* spp.), and tularemia (*Francisella tularensis*) (Sonenshine, 1993). These examples are just a sample of the most well-known disease-causing pathogens that ticks transmit, and many more exist.

As important vectors of pathogens, a large focus has been placed on the effectiveness of tick control for reducing pathogen transmission. However, much of this work has been done without considering tick population dynamics and dispersal. Population genetic studies provide insights into the evolutionary forces driving current and past gene flow, the distribution of species, and host adaptations that might influence disease specificity and resistance to chemical acaricides. Furthermore, local adaptation can occur in vector and pathogen populations that can have consequences for epidemiology. For instance, intraspecific variation in transmission ability is known from *Aedes albopictus* mosquitoes that transmit arboviruses (Mitchell, 1991). As obligate parasites and vectors, ticks have a direct influence on their hosts and the pathogens they transmit (Anderson and Magnarelli, 2008; Hill and Wikel, 2005; Pagel Van Zee et al., 2007; Sonenshine, 1993). Each member of a host–vector–pathogen system is affected by the other players; this combined effect can influence genetic variation in ticks (Jongejan et al., 2007).

Tick population genetic studies serve as a logical bridge between the basic biology of vectors and the investigation of tick-borne pathogens. First, estimates of genetic structure and dispersal in ticks may serve as a surrogate for dispersal estimates of tick-borne pathogens. Second, quantifying gene flow (the outcome of successful dispersal) in ticks has become a central question because genetic structure of these parasites often does not parallel that of their host. Third, these studies may reveal non-random mating patterns in ticks, such as inbreeding and the development of host-specific races, which can have important implications for pathogen transmission. Finally, studies that combine host, vector, and pathogen genetics facilitate an understanding of the evolutionary processes linking species across trophic levels.

2. Search methods

We started our literature review with the following key words: "ticks and genetics", "ticks and population structure", "ticks and population genetics", and "ticks and DNA". This search included all population genetic studies found using PUBMED up to November, 2014. We also manually browsed Literature Cited sections from tick genetic studies to find additional publications focused on tick population genetics.

A population genetics study was defined as one that used molecular markers to address the questions of genetic variability, genetic population structure, gene flow, and/or genetic isolation by distance in one or more tick species. We did not include the large body of work focused on phylogenetics (Estrada-Pena et al., 2010; Nava et al., 2009) or whole genome sequencing and functional genomics, although we recognize that next-generation approaches will greatly advance our knowledge of tick biology and evolution (Andreotti et al., 2011; Rachinsky et al., 2008). We chose to group and discuss population-level studies according to taxonomy. This organization allows a comparison of closely related species across different geographical locations and genetic markers. A discussion of the utility of genetic markers for tick population genetic studies is also provided at the end of this review.

3. Molecular studies in six tick genera

In our literature review we found population genetic studies for 22 tick species from six genera (*Amblyomma*, *Bothriocroton*, *Dermacentor*, *Ixodes*, *Ornithodoros*, and *Rhipicephalus*) representing the two major tick families, Argasidae and Ixodidae (Table 1). Although all of these papers include molecular information related to genetic variation and its distribution, we were surprised to find that many studies did not address population genetics explicitly in terms of using formal analyses specific to this discipline (e.g., fixation indices such as F_{ST} and F_{IS}, AMOVA, and population assignment). In part, this is because a number of studies have focused on biogeographical patterns of older divergence within a species rather than finer-scale population structure among contemporary populations. We point out that an essential step in any genetic study is to choose molecular markers with an appropriate mutation rate for the evolutionary scale being examined. Conserved genes such as nuclear 18S or mitochondrial 16S change slowly over time and are best suited for looking at more ancient divergence among species or subspecies. Markers with a faster mutation rate (such as microsatellite repeat loci) are more

Table 1
Summary of tick population genetic studies, including the marker systems used for each species.

Taxa	Marker	Summary	Citations
Argasidae			
Ornithodoros capensis	DNA sequence (16S/18S)	Multiple divergent lineages introduced to Cape Verde Islands via dispersal on highly mobile hosts (seabirds)	Gomez-Diaz et al. (2012)
Ornithodoros coriaceus	DNA sequence (16S)	Multiple divergent lineages on moderately mobile hosts (deer, cattle)	Teglas et al. (2005)
Ornithodoros sonrai	DNA sequence (16S/18S)	Ancient divergence among four genetic groups in West Africa	Vial et al. (2006)
Ixodidae			
Amblyomma americanum	DNA sequence (ITS2/16S), isozymes	Low structure on highly mobile hosts (birds, canines, deer)	Hilburn and Sattler (1986b), Mixson et al. (2006), Reichard et al. (2005), Trout et al. (2010)
Amblyomma dissimile	Isozymes	High structure on low mobility hosts (reptiles, salamanders, small mammals)	Lampo et al. (1998)
Amblyomma dubitatum	DNA sequence (16S)	Low 16S variation; use of multiple hosts (capybaras, livestock) may reduce structure	Nava et al. (2010)
Amblyomma maculatum	DNA sequence (16S)	Low structure on moderately mobile hosts (deer, cattle, birds); also low host specificity and recent population expansion	Ferrari et al. (2013)
Amblyomma triste	DNA sequence (16S)	Low structure on moderately mobile hosts (deer, livestock); also low host-specificity	Guglielmone et al. (2013)
Amblyomma variegatum	DNA sequence (D-loop/12S/16S/*cytb*)	Two major lineages in Africa; East African lineage has spread to nearby islands while West African lineage has spread globally; local populations on human-transported hosts show very low structure	Beati et al. (2012), Stachurski et al. (2013)
Bothriocroton hydrosauri	Microsatellites	Hierarchical structure within infrapopulations and among hosts in close proximity due to ripple effect of successful tick sibling groups	Guzinski et al. (2008, 2009)
Dermacentor albipictus	Microsatellites, DNA sequence (ITS2, *lys*, 16S, COI)	Two major mitochondrial lineages, possibly from mtDNA introgression from *D. nitens*; microsatellites predict three recently diverged genetic groups in Alberta	Crosbie et al. (1998), Leo et al. (2010, 2012, 2014)
Dermacentor andersoni	AFLPs,DNA sequence (12S/16S)	Variable structure despite highly mobile hosts (deer and elk)	Araya-Anchetta et al., 2013; Lysyk and Scoles, 2008; Patterson et al., 2009
Dermacentor variabilis	Microsatellites, AFLPs, DNA sequence (16S)	Low structure on highly mobile hosts (canines) but isolated populations are highly differentiated	Araya-Anchetta et al. (2013), de la Fuente et al. (2005), Dharmarajan et al. (2010b), Krakowetz et al. (2010)
Ixodes arboricola	Microsatellites	Structure at two hierarchical levels: 1) among nesting boxes (due to larval sibling groups, and 2) structure among woodlots	Van Houtte et al. (2013), Van Oosten et al. (2014)
Ixodes ricinus	Microsatellites, DNA sequence (multiple loci from nDNA and mtDNA), isozymes	Variable structure depending on scale and markers used; high structure observed among host-specific races; also sex-biased dispersal is known	Casati et al. (2008), De Meeûs et al. (2002), Delaye et al. (1997, 1998), Dinnis et al. (2014), Healy (1979a,b), Kempf et al. (2009b, 2010, 2011), Noureddine et al. (2011)
Ixodes pacificus	DNA sequence (COIII), allozymes	High structure on highly mobile hosts	Kain et al. (1997, 1999), McLain et al. (1995), Wesson et al. (1993)
Ixodes scapularis	Microsatellites, DNA sequence (16S/*cytc*)	High structure on highly mobile hosts	Humphrey et al. (2010), Kempf et al. (2009b, 2010, 2011), Krakowetz et al. (2011), McLain et al. (1995), Norris et al. (1996), Qiu et al. (2002), Rich et al. (1995), Wesson et al. (1993)
Ixodes texanus	Microsatellites	High structure results from nidiculous behavior and kin structure despite a moderately mobile host (raccoon)	Dharmarajan et al. (2010a)
Ixodes uriae	Microsatellites, DNA sequence (COIII)	Recent development of host races within nesting colonies	Dietrich et al. (2012a,b, 2013, 2014), Kempf et al. (2009a), McCoy et al. (1999), McCoy and Tirard (2000), McCoy et al. (2001, 2003a,b, 2005b, 2012), McCoy and Tirard (2000, 2002)
Rhipicephalus annulatus	Microsatellites	Single host ticks that display variable levels of structure on moderately mobile hosts (cattle, deer, red deer)	Araya-Anchetta (2012)
Rhipicephalus australis (formerly *R. microplus*)	Microsatellites	Single host ticks that display variable levels of structure in Australia; development of host races on New Caledonian cattle and deer	Chevillon et al. (2007, 2013), Cutullé et al. (2009), de Meeus et al. (2010), Koffi et al. (2006a,b)
Rhipicephalus microplus	Microsatellites, isozymes, DNA sequence (*para*-sodium ion channel)	Single host ticks that display variable levels of structure on moderately mobile hosts (cattle, deer); permethrin resistant genotypes increasing in Mexico and the U.S.	Busch et al. (2014), Sattler et al. (1986), Stone et al. (2014)

appropriate for evaluating recent differentiation among populations (Avise, 2004). Table 2 and Section 5 provide additional information on the relative mutation rates of molecular markers for population genetic studies. In this section, we will discuss molecular methods, questions, and analyses used to investigate genetic variation in each genus.

3.1. Argasidae (soft ticks)

Ornithodoros ticks belong to the family Argasidae (soft ticks). Ticks in this genus are responsible for the transmission of several pathogens, including human relapsing fever (*Borrelia* spp.), African swine fever virus, and a currently unknown agent associated with

Table 2
Main characteristics of molecular markers used to analyze tick vectors.

Marker	Type of dominance	Automation possible	Mutation rate	Level of homoplasy	Cross study comparisons	Previous sequence knowledge required	Development cost	Greatest disadvantage
Allozymes (proteins)	Codominant	No	Low	Intermediate	Easy	No	Low	Poor resolution for population genetics due to low variation
AFLPs	Dominant (presence/absence)	Yes	Variable	High	Intermediate	No	Intermediate	Homoplasy
Microsatellites	Codominant	Yes	High	Intermediate	Intermediate	Yes	High	Presence of null alleles
Mitochondrial gene sequences	Codominant (single haplotype per individual)	Yes	Low-Med (locus-dependent)	Low-intermediate	Easy	Yes	Intermediate	Maternal lineage only; mutational saturation leads to homoplasy at broad evolutionary scales
Nuclear gene sequences	Codominant	Yes	Low-Med (locus-dependent)	Low-intermediate	Easy	Yes	Intermediate	Alleles in a heterozygous state require additional cloning and sequencing to resolve
SNPs	Codominant	Yes	Low	Low	Easy	Yes (but not for whole-genome sequencing)	High	Ascertainment bias

epizootic bovine abortion (EBA). Each *Borrelia* species causing human relapsing fever in Africa is considered to be specific to a different *Ornithodoros* lineage. Therefore, identifying each tick group with genetic tools is an important application for public health. A sequencing approach was used to study *Ornithodoros sonrai* sampled from 14 sites (n = 29 specimens) in the west African countries of Senegal and Mauritania (Vial et al., 2006). One mitochondrial gene region (16S rRNA) and one nuclear gene region (18S rRNA) were chosen for this phylogeographic analysis. Parsimony analysis identified four distinct genetic groups within *O. sonrai* with different geographical distributions (Vial et al., 2006). The clear genetic delineation among these four groups validated an early breeding experiment that yielded poor fecundity in crosses that involved parents from different geographical areas (Chabaud, 1954). Furthermore, the authors noted variation in the infection rates of *Borrelia* among the four *O. sonrai* lineages, which could suggest an important difference in their vector competence for *Borrelia* (Vial et al., 2006). Although the small sample sizes per population in this study (n = 1–3) did not make it possible to quantify population differentiation among sample sites, the 16S and 18S gene sequences provided valuable insights into the biogeography of present-day populations.

A more in-depth study has been performed on a related species, *Ornithodoros coriaceus*, which was sampled from 14 populations in the western U.S. (California, Nevada and Oregon) (Teglas et al., 2005). Using a 420 bp region of the 16S gene, the authors observed an extremely high level of population structure. An analysis of molecular variance (AMOVA) estimated that 85% of the genetic variation was partitioned among populations, which suggests almost no gene flow occurred among the sampled sites. This genetic pattern has implications for livestock health, because epizootic bovine abortion is transmitted by *O. coriaceus* and the disease had spread into Nevada and Oregon from California. The severe limitation of tick dispersal inferred by 16S data suggests that EBA was not spread by infected ticks, since the movement of ticks would likely have resulted in gene flow. Instead, the disease probably spread through infected cattle that were transported by humans. The etiological agent for EBA remains unknown but it most likely entered local tick populations after infected hosts had been transported and pastured.

Genetic data have been invaluable for studying colonization patterns in the *Ornithodoros capensis* complex from Africa and Europe. This species complex includes eight morphospecies of ticks (Dietrich et al., 2012a; Hoogstraal et al., 1976) that parasitize widely-ranging seabirds such as such as gulls, terns, boobies, and petrels (Estrada-Pena et al., 2010). Analysis of 16S and18S gene sequences determined that *O. capensis* ticks from the Cape Verde islands west of Senegal, Africa, exhibit high genetic diversity that can be attributed to multiple independent colonization events (Gómez-Díaz et al., 2012). The genetic sources of Cape Verde ticks include widely separated locations from the Atlantic, Pacific, and Indian Oceans and the 16S/18S data are consistent with deep genetic delineations among ocean basins. These results might not be expected from an Argasid species when considering their high site fidelity and relatively short (20–70 min) feeding times. However, feeding behavior during larval stages may act to promote dispersal and the larvae of many Argasid species feed for several days at a time (Apanaskevich and Oliver, 2014). Thus, it is important to understand the combined effect of all life stages on gene flow before making predictions about genetic structure in Argasid ticks. Interestingly, the authors also identified specific tick-host associations at a local scale and suggested that host switching may occur in the Cape Verde Islands (Gómez-Díaz et al., 2012). A robust test of this possibility will require markers with a faster mutation rate, such as microsatellites.

3.2. Ixodidae (hard ticks)

3.2.1. Amblyomma

Population genetic studies have been published for six *Amblyomma* species: *Amblyomma americanum* (Hilburn and Sattler, 1986b; Mixson et al., 2006; Reichard et al., 2005; Trout et al., 2010), *Amblyomma dissimile* (Lampo et al., 1998), *Amblyomma dubitatum* (Nava et al., 2010), *Amblyomma maculatum* (Ferrari et al., 2013; Ketchum et al., 2009), *Amblyomma triste* (Guglielmone et al., 2013), and *Amblyomma variegatum* (Beati et al., 2012). In general these studies indicate that *Amblyomma* ticks exhibit low genetic variation and high levels of gene flow. We note this generalization is made across tick species with different natural histories, and studies utilizing different sampling scales, sample sizes, and genetic markers. An exception to this pattern is *A. dissimile*, a South American tick that demonstrates high genetic structure (Lampo et al., 1998).

A. americanum, the Lone Star tick, is a vector for *Ehrlichiosis chafensis* and *Rickettsia amblyommii*; the latter is the agent of a spotted fever-like disease (Trout et al., 2010). Hilburn and Sattler studied the genetic variation of nine populations across the US geographical range of *A. americanum*. Using 21 isozymes, they noted an absence of genetic differentiation across all collection sites (Hilburn and Sattler, 1986b). More recent studies have utilized DNA sequences from a 300 bp region of the mitochondrial 16S rRNA gene and found a similar pattern of low genetic differentiation in *A. americanum* populations from multiple ecoregions in the states of Georgia (Mixson et al., 2006) and Arkansas (Trout et al., 2010). Both studies found evidence in support of a population increase after a bottleneck (using Fu's test of neutrality), which supports the possibility of a recent range expansion mediated by widespread dispersal on avian and large mammalian hosts (e.g. canines and white-tailed deer).

Two genetic studies have led to interesting discoveries about the spread of *A. variegatum*, the tropical Bont tick. This species is a vector for *Ehrlichia ruminantium*, which causes heartwater (cowdriosis) in cattle, as well as *Rickettsia africae*, the pathogen causing African tick bite fever. An initial analysis of mitochondrial 12S and D-loop sequences suggested deep divergence between ticks from East and West Africa (Beati et al., 2012). Populations of *A. variegatum* have become established in several Caribbean islands and share close genetic similarity to the West African lineage. In fact, 16S and cytochrome *b* (*cytb*) sequence data suggest that the West African lineage has spread broadly in Africa and invaded Madagascar long ago (Stachurski et al., 2013). This lineage is also found in neighboring islands of the Indian Ocean. In contrast, the East African lineage has not spread as widely. This lineage has invaded only a few islands of the Indian Ocean, including Madagascar. Based on differences in the current distribution of both lineages, the authors hypothesized greater ecological plasticity in the West African *A. variegatum* lineage, which may allow it to become established in a wider range of new habitats (Stachurski et al., 2013).

A. dissimile in South America presents increased genetic structure compared to other *Amblyomma* species (Lampo et al., 1998). In this study, eight isozyme loci were used to assess genetic variation at eleven localities in Venezuela. Host dispersal appears to be the most significant factor for determining gene flow in *A. dissimile* because this tick is associated with amphibians, reptiles, and occasionally small mammals. It is important as a vector for *E. ruminantium* in places like sub-Saharan Africa, the Caribbean, and South America.

3.2.2. Bothriocroton

The genus *Bothriocroton* includes seven tick species indigenous to Australia and nearby Pacific islands. Interestingly, endangered status has been proposed for a species from Papua New Guinea and Indonesia, the echidna tick (*Bothriocroton oudemansi*) (Beati et al., 2008). Four *Bothriocroton* taxa parasitize reptile hosts including *Bothriocroton hydrosauri*, a vector of *Rickettsia honei*. A study of population dynamics in *B. hydrosauri* used nine microsatellite loci (Guzinski et al., 2008) to follow genetic signatures of relatedness on a large skink species (*Tiliqua rugosa*) in southeastern Australia. Spatial genetic structure at two hierarchical levels was observed, the first arising from sibling groups in the infrapopulations (the ticks infesting a single host) of individual lizards and the second due to spatial autocorrelation of tick genotypes on hosts found in close proximity (Guzinski et al., 2009). The authors found a genetic "ripple effect" explained by successful tick clutches dispersing outward from their point of origin.

3.2.3. Dermacentor

The genus *Dermacentor* comprises about 30 species found in the New World, Eurasia, and Africa (Crosbie et al., 1998). Most are three-host ticks but some species are one-host ticks (e.g., *Dermacentor albipictus* and *Dermacentor nitens*). Several *Dermacentor* species are important vectors of pathogens to humans, livestock, and wildlife (Yunker et al., 1986). Two North American species, *Dermacentor andersoni* and *Dermacentor variabilis*, have been the focus of multiple population genetic studies (Araya-Anchetta et al., 2013; de la Fuente et al., 2005; Dharmarajan et al., 2010b; Krakowetz et al., 2010; Lysyk and Scoles, 2008; Patterson et al., 2009). *D. variabilis*, also known as the American dog tick, is widely distributed in North America. Two major clades are known from mitochondrial 16S sequences; Clade A consists of ticks from the central and eastern US and Canada, whereas clade B is only found in the western US (California, Idaho, and Washington) (Krakowetz et al., 2010; Scoles, 2004). This species appears to have experienced a historical population expansion into southern Canada, as evidenced by an excess of rare 16S haplotypes (Krakowetz et al., 2010).

The factors influencing population structure in *D. variabilis* at a local scale were investigated by Dharmarajan et al. (2010b) using 8 microsatellite loci. Adult ticks were collected directly from raccoons in two habitat patches separated by <6 km. The authors found low but sometimes significant levels of genetic structure (F_{ST} 0–0.02), which seemed to be determined by male-biased dispersal in ticks and the small number of sibling groups that infested individual raccoons. Interestingly, tick gene flow was higher among ticks collected from young raccoons compared to adults. This parallels the greater dispersal of young raccoons during the spring months, when *D. variabilis* larvae are actively questing (Dharmarajan et al., 2010b). On a larger geographic scale, observed population structure using amplified fragment length polymorphisms (AFLPs) was high (F_{ST} = 0.30) among three disjunct populations of *D. variabilis* in northwestern US (Araya-Anchetta et al., 2013). These isolated western populations are a result of human-mediated expansion of *D. variabilis* outside its natural range (Stout et al., 1971) where it can hybridize with native *D. andersoni* (Araya-Anchetta et al., 2013).

As observed in *D. variabilis*, the level of gene flow among populations of *D. andersoni* varies depending on the sampling scheme and the molecular marker of choice (Araya-Anchetta et al., 2013; Lysyk and Scoles, 2008). In a comparison between a prairie and a montane population of *D. andersoni* in southern Canada using 16S and 12S sequences, observed population structure was very high (F_{ST} = 0.49) (Lysyk and Scoles, 2008). However, the barriers to gene flow were not strong enough to reduce reproductive compatibility between ticks of both populations as determined by reciprocal crosses (Lysyk and Scoles, 2008). When quantified across nine populations in western US and Canada using AFLPs, the observed genetic structure was moderate (F_{ST} = 0.11) with only a weak signature of isolation-by-distance (r = 0.06) (Araya-Anchetta et al., 2013). The genetic differentiation among *D. andersoni* populations appears higher than expected for a field-dwelling,

hard tick that uses multiple highly mobile hosts (large herbivores). We speculate that this may be due to habitat heterogeneity (e.g. mountain ranges) in the western U.S., or environmental tolerance limitations that reduce the success of dispersing ticks.

The winter tick, *D. albipictus*, parasitizes large ungulates and has a wide geographic range in North America. Mitochondrial gene sequences (COI and 16S) show two deeply diverged mitochondrial lineages (Crosbie et al., 1998; Leo et al., 2010). However, this signature is not observed in nuclear loci (ITS-2 and lysozyme) and one of the mitochondrial lineages may be the result of past hybridization with a congener, possibly *D. nitens*. Since the mid-1990s, *D. albipictus* infestations on elk (*Cervus elaphus*) have increased in the Yukon region of Canada. A recent study of this expansion used DNA sequence from three genes (ITS-2, COI, and 16S) and 14 microsatellite loci (Leo et al., 2012) to predict a genetic origin of the new tick populations (Leo et al., 2014). The most likely source was predicted to be either a gene pool in northern Alberta and British Columbia that is naturally expanding northward, or the result of an elk translocation from central Alberta. Genetic structure was not partitioned among the five host species sampled in this study, thus newly dispersed *D. albipictus* populations may be able to spread freely using any available ungulate hosts.

3.2.4. Ixodes

As with other ticks, *Ixodes* spp. are relevant to humans because of the pathogens they transmit. A particular group of interest is the *Ixodes ricinus* complex, which is comprised of 14 closely related taxa that collectively are distributed across most of the world (Xu et al., 2003). Species within this complex have been identified as vectors for the causative agents of louping ill viral disease (ovine encephalomyelitis), Russian spring-summer encephalitis (another viral disease), human granulocytic ehrlichiosis (caused by *Ehrlichia* bacteria), and babesiosis (caused by *Babesia* protozoans). One of the most important pathogens transmitted by the *I. ricinus* complex is *Borrelia burgdorferi*, the agent of Lyme disease in North America and Eurasia. *Ixodes* species transmit different genospecies of *B. burgdorferi* according to their geographical distribution (Xu et al., 2003). All of the studies below concern species within the *I. ricinus* complex (Subgenus *Ixodes*).

I. ricinus has a wide distribution across Scandinavia, other parts of Europe (including the British Isles), and Northern Africa (Estrada-Peña, 2001). This is the first tick species to have been used in a population genetic study, in which two isozyme loci (α-GPDH and PGM) were used to look at genetic variation in ticks from four locations in Ireland (Healy, 1979a,b). Diversity was high at both loci and differences between allelic frequencies were found between males and females for α-GPDH but not PGM. However, PGM allele frequencies shifted significantly between spring and autumn samples (Healy, 1979b). This was attributed to a temporal difference in mating phenology between two sympatric populations, which would limit gene flow and lead to an increase in genetic differentiation. These same two allozymes were also used to look for geographic structure in five Swiss populations, but no differentiation was detected (Delaye et al., 1997). An intriguing element of the Irish study was evidence that α-GPDH in females was positively related to walking activity, because heterozygous females displayed greater activity than homozygous females (Healy, 1979a). This makes sense because α-GPDH plays a role in muscle fiber contraction and recovery. This early study provided insight into genes of adaptive importance in natural tick populations.

More recent population genetic studies on *I. ricinus* show a variety of results and can be sorted into two categories: (1) studies designed to investigate older genetic divergence across the entire range of *I. ricinus* by sequencing multiple genes from a small number of ticks per site (Casati et al., 2008; Noureddine et al., 2011), and (2) studies of contemporary population structure at a finer scale that used microsatellite markers and greater sample sizes per site (De Meeûs et al., 2002; Kempf et al., 2009b, 2011, 2010). Studies in the first group used a combined total of six mtDNA genes and four nuclear genes, and found a major genetic division between *I. ricinus* in Eurasia and North Africa. Additional variation within Europe has also been detected using gene sequences from six mitochondrial genes (ATP6, COI, COII, COIII, CYTB, and 12S) that comprise a new multilocus sequence typing (MLST) system for ticks (Dinnis et al., 2014). The second group of studies detected variable genetic structure among populations (F_{ST} 0.01–0.05) at a finer scale. In addition, significant levels of cryptic structure were found that could be attributed to assortative mating and host race formation (see below). The mutation rates of microsatellites are faster than either mitochondrial or nuclear genes and tend to be more informative at the population scale (Avise, 2004; Keim et al., 2004). Gene sequences provide evidence of evolutionary events at a broader scale, such as finding a clear genetic division between North African and Eurasian samples of *I. ricinus* (Noureddine et al., 2011) and the ability to resolve the American and southern clades of *Ixodes scapularis* (Norris et al., 1996; Qiu et al., 2002; Rich et al., 1995; Wesson et al., 1993).

Use of microsatellite markers in *I. ricinus* population studies has led to three major findings. First, this species presents moderate to high genetic structure at a local scale; second, there is a deviation from panmixia (random mating); and third, host use is not random (De Meeûs et al., 2002; Kempf et al., 2009b, 2011, 2010). A genetic analysis of *I. ricinus* ticks collected in Switzerland and Tunisia originally presented no differentiation at a local scale (De Meeûs et al., 2002). However, microsatellite data in *I. ricinus* exhibits a deficit in heterozygotes that is not solely explained by technical issues (e.g., null alleles). Upon further analyses, these researchers discovered population substructuring within Swiss populations that was obscured by a Wahlund effect (Kempf et al., 2010). This dataset also showed a bias in tick dispersal according to sex, which possibly reflects differences in host use between males and females. Another way that population structure can occur in a species is assortative mating (mating between genetically similar individuals) instead of random mating. Assortative mating was observed in two out of four locations in northern France in *I. ricinus* (Kempf et al., 2009b). In addition, a non-random use of hosts was shown in *I. ricinus* ticks collected from birds, rodents, lizards, roe deer, and wild boar (Kempf et al., 2011). Further studies are required to confirm the existence of host races in *I. ricinus* as well as to determine their effect on the pathogens that this species transmits.

Nidicolous species of *Ixodes* can demonstrate high relatedness within individual harborage sites and/or hosts. A deficit of heterozygotes was observed in *Ixodes texanus* infrapopulations collected from raccoons (Dharmarajan et al., 2010a). Three hypotheses were proposed to explain this deficit: technical issues, population structure due to the host or tick life stage, and the presence of a cryptic population structure. The occurrence of null alleles did not explain the heterozygote deficit entirely (similar to *I. ricinus*) and further exploration showed the existence of kin structure among samples of *I. texanus* ticks. This kin structure resulted from breeding group structure among individual raccoon hosts, coupled with a large variance in the reproductive success of adult ticks (Dharmarajan et al., 2010a). Another nidicolous tick, *Ixodes arboricola*, demonstrates high relatedness among larval groups from individual harborage sites (nest cavities in trees and man-made nest boxes) found in woodlots near Antwerp, Belgium. Surprisingly, microsatellite loci developed for this species (Van Houtte et al., 2013) reveal greater genetic diversity and lower relatedness among older instars within the same harborages (Van Oosten et al., 2014). Thus, dispersal appears to occur sometime late in larval development. As

might be expected from a nidicolous tick that parasitizes territorial songbirds such as the great tit (*Parus major*) and blue tit (*Cyanistes caeruleus*), genetic structure was found both at the level of individual nest boxes and among woodlots.

In North America, Lyme disease is mainly transmitted by *I. scapularis* in the east and *Ixodes pacificus* in the west. Previously, *I. scapularis* was considered to be found only in the northeast, whereas a sister species, *Ixodes dammini*, comprised populations in the southeast. However, both are now grouped under one species, *I. scapularis* (Keirans et al., 1996). In the US, genetic variation in 16S sequences is partitioned across two *I. scapularis* lineages: (1) the American clade, found in the northeastern US down to the Carolinas, and (2) the southern clade, which overlaps in the Carolinas and extends into the southeastern states (Norris et al., 1996; Qiu et al., 2002; Rich et al., 1995; Wesson et al., 1993). In Canada, four additional subpopulations have been discovered recently, with frequency differences among mitochondrial cytochrome c haplotypes occurring in central Canada (Alberta to western Ontario), eastern Ontario, Quebec, and the Atlantic Provinces (Mechai et al., 2013). Support for combining *I. scapularis* and *I. dammini* into a single species was found by carefully examining how genetic variation was distributed among the species compared to *I. pacificus* (McLain et al., 1995; Wesson et al., 1993). In both studies, the observed sequence diversity using 16S sequences was greater between *I. scapularis* and *I. pacificus* than that found among the American and southern clades of *I. scapularis*. Further analyses demonstrate greater genetic diversity in the southern *I. scapularis* lineage. The species is now considered to have arisen and diversified in this region and recently expanded to the north (Humphrey et al., 2010; Norris et al., 1996; Rich et al., 1995). These genetic differences between tick lineages seem to influence the ability to carry *B. burgdorferi*, as infection rates for northern *I. scapularis* are higher than for southern ticks and the disease is most prevalent in the north (Qiu et al., 2002).

Despite the dispersal of *I. scapularis* by migratory passerine birds, gene flow is consistently limited across the species range and at a regional scale (Humphrey et al., 2010; Krakowetz et al., 2011; McLain et al., 1995; Norris et al., 1996; Qiu et al., 2002; Trout et al., 2009; Wesson et al., 1993). For example Trout et al. (2009) observed a pronounced Wahlund effect in *I. scapularis* ticks from Arkansas, and marked genetic differences between coastal and inland populations of ticks were observed in North Carolina (Qiu et al., 2002). At a larger scale, population structure of this species has been driven by the differences between the American and southern clades but also by local limitations to gene flow that remain unknown (Humphrey et al., 2010; Krakowetz et al., 2011; Norris et al., 1996; Qiu et al., 2002; Wesson et al., 1993). It is possible that further exploration of the link between the migratory patterns of passerine birds and *I. scapularis* proposed by Krakowetz et al. (2011) will shed light on how gene flow is limited in this tick.

I. pacificus is responsible for the transmission of *B. burgdorferi* in the western US. Low to moderate gene flow has been observed in this tick vector using eight polymorphic allozymes against 20 populations sampled along the species range (Kain et al., 1997). A possible differentiation between northern and southern regions was particularly strong at one locus. Further analysis of mtDNA sequences of cytochrome oxidase III (COIII) determined the isolation of an *I. pacificus* population in Utah from those found along the North American west coast (Kain et al., 1999). The Utah population carries very low mtDNA sequence polymorphism, possibly as a result of a founding event after Pleistocene glaciation.

The exploration of population structure and host race formation in *Ixodes uriae* is the most comprehensive body of work in tick population genetics (Dietrich et al., 2013, 2014, 2012b; Kempf et al., 2009a; McCoy et al., 2012, 1999, 2002, 2005a, 2001, 2003a, 2005b; McCoy and Tirard, 2000, 2002; McCoy et al., 2003b). This species is part of the *I. ricinus* complex and a vector of the Lyme disease pathogen, *B. burgdorferi*. It is a three-host tick that parasitizes colony-nesting seabirds in circumpolar regions of both hemispheres (McCoy et al., 2001). Four major genetic lineages exist globally, delineated by the far northern and southern regions of the Pacific and Atlantic Oceans (Dietrich et al., 2014). Diversification of ancient mitochondrial lineages in the southern Pacific began around 22 MYA from likely origins in Australia and New Zealand. Northward expansion occurred later in both the Pacific and Atlantic. Genetic diversity is greater in southern populations than northern, as might be expected from their greater age.

Host race formation in *I. uriae* has been recurrent (Dietrich et al., 2014) and analysis of COIII sequences suggests that host races in this tick have developed only recently (Kempf et al., 2009a). Observations of host races in *I. uriae* started with an examination of Atlantic populations found on two species of birds (black-legged kittiwakes, *Rissa tridactila*, and Atlantic puffins, *Fratercula arctica*) that showed greater genetic differentiation between sympatric tick populations on different host species than between allopatric tick populations on the same host (McCoy et al., 2001, 2003a). This provided strong support for the host race hypothesis.

Samples from a greater number of host species across a wider geographic range provided additional evidence for host race formation (Dietrich et al., 2012b; McCoy et al., 2005b). The sampling scheme used included a combination of monospecific and heterospecific colonies of birds sampled in the North Atlantic, North Pacific, and Indian Oceans (Dietrich et al., 2012b; McCoy et al., 2005b). Analysis of eight microsatellite markers (McCoy and Tirard, 2000) revealed genetic isolation among tick populations in the Northern and Southern hemispheres (North Atlantic and Indian Ocean) as well as between North Atlantic and North Pacific populations, which is consistent with the major migratory routes of marine birds (Dietrich et al., 2012b; McCoy et al., 2005b). This allowed tick populations to be grouped according to host species regardless of colony type (mono- or heterospecific) or geographic distance (McCoy et al., 2005b). Interestingly, variation in the degree of host specificity was observed among races of *I. uriae* and correlated with the degree of genetic isolation between races (Dietrich et al., 2012b). These differences likely reflect the influence of host life-history traits on parasite population structure (Dietrich et al., 2013; Kempf et al., 2009a). Furthermore, recent observations have shown that host races are not just genetically isolated but that they present a high degree of morphological variation. It is reasonable to hypothesize that differences between races are adaptive responses to host differences (Dietrich et al., 2013).

A second set of population genetic studies of the *I. uriae* system involves both smaller and greater spatial scales. At a smaller scale, levels of population genetic structure were studied among nests in three different cliffs used by kittiwakes. Genetic differentiation among nests was low but significant among all three cliffs. A deficit of heterozygotes explained population structure in two of the cliffs but in the third cliff it was associated with the density of nest infestation. Also, in a comparison of genetic structure between *I. uriae* ticks and their black-legged kittiwake hosts, no correlation was found between the population structures of twenty-two bird colonies and fourteen tick collection using species-specific microsatellites (McCoy et al., 2005a, 2003b). Observed patterns may reflect differences in evolutionary rates due to disparities in generation times and effective population sizes. In addition, they provide evidence of cryptic movements by kittiwakes during their breeding season (McCoy et al., 2005a). It is interesting to observe how the change from a short to a long-range geographical scale results in a change in the degree of population structure. At a smaller scale, population structure seems to respond to *I. uriae* life history traits,

whereas at a large scale structure will depend on the dispersal capabilities of the host.

3.2.5. Rhipicephalus

Three members of genus *Rhipicephalus* (*Rhipicephalus microplus*, *Rhipicephalus annulatus*, and *Rhipicephalus australis*) are ticks of global economic importance because they transmit cattle fever pathogens that cause high morbidity and mortality (Estrada-Peña et al., 2006; Sattler et al., 1986; Wang et al., 2007). All were formerly members of the genus *Boophilus* but recent molecular evidence places them in the genus *Rhipicephalus* (Murrell and Barker, 2003; Murrell et al., 2000). Furthermore, the populations of *R. microplus* in Australia, Cambodia, New Caledonia, and numerous islands throughout Indonesia are now recognized as a distinct species, *R. australis*, based on 12S/16S sequences (Burger et al., 2014; Estrada-Pena et al., 2012; Labruna et al., 2009). Bovine babesiosis is caused by a number of apicomplexan protozoa, two of which (*B. bovis* and *B. bigemina*) are transmitted by *R. annulatus*, *R. australis*, and *R. microplus* (Bock et al., 2004). These vectors also transmit protozoans in the genus *Theileria* (*Theileria cervi* and *Theileria ovis*) and the bacterial pathogen *Anaplasma marginale*. In Africa, *Rhipicephalus appendiculatus* transmits *T. parva* among domestic cattle and wildlife (Bishop et al., 2004; Walker et al., 2014). Thus, Rhipicephaline ticks have been a major constraint to cattle production throughout tropical and subtropical agricultural systems because of agricultural losses in milk and beef production (Estrada-Peña et al., 2006; Wang et al., 2007). The *Rhipicephalus*–*Babesia* interaction was one of the first vector–pathogen systems to be described in detail (Smith and Kilborne, 1893) and led to the groundbreaking insight that eradicating tick vectors would prevent the spread of this protozoan disease agent (Curtice, 1896). In an effort to eliminate babesiosis from the United States, the Cattle Fever Tick Eradication Program (CFTEP) was initiated in 1906. The program was successful in all states except for Texas where today a tick eradication quarantine area (TEQA) serves as a buffer from cattle imported from Mexico (Bram et al., 2002). Although savings from the eradication of babesiosis have been estimated to be 1 billion US$ per year (Graham and Hourrigan, 1977), *R. microplus* and *R. annulatus* continue to infest cattle in Texas and threaten to invade beyond the TEQA (Giles et al., 2014).

Sattler and coworkers were the first to investigate genetic differentiation between four natural populations and four laboratory colonies of *R. microplus* (the southern cattle tick) from the TEQA, Mexico, and Puerto Rico using isozymes (Sattler et al., 1986). Fifteen polymorphic loci showed levels of heterozygosity similar to other arthropods and high genetic similarity among all strains and populations. Based on this result it was hypothesized that *R. microplus* in North America shares an undifferentiated gene pool (Sattler et al., 1986). This outcome is consistent with mitochondrial evidence (12S/16S) that suggests that a monophyletic tick lineage invaded the Americas (Labruna et al., 2009).

In contrast to the low genetic structure inferred from isozymes and mitochondrial sequences, a recent study using microsatellite markers discovered much greater genetic differentiation among southern cattle tick populations in the TEQA of southern Texas (Busch et al., 2014). Using 11 repeat loci, undifferentiated tick collections were only observed at a local scale (<4 km). Beyond this distance, genetic structure rapidly increased in a pattern of isolation-by-distance. Similar results were observed in 20 TEQA collections of *R. annulatus* using 11 new microsatellite loci developed from a partial genome sequence of *R. annulatus* (Araya-Anchetta, 2012). Isolation-by-distance in both *R. microplus* and *R. annulatus* probably results from high levels of local tick dispersal facilitated by cattle grazing rotations among neighboring properties. Local movements on alternative hosts such as horses (*Equus ferus caballus*), white-tailed deer (*Odocoileus virginianus*), and exotic red deer (*Cervus elaphus*) probably also promote gene flow at a local scale. However, the signature of isolation-by-distance in *R. microplus* is obscured by occasional long-distance movements of ticks mediated by human transport. Well-documented reports by the CFTEP demonstrate that infested cattle can be shipped hundreds of miles before ticks are detected and removed. Although some of these movements could reduce genetic structure over large distances (by spreading genetically similar ticks across the landscape), most appear to increase the average level of genetic structure. In particular, two recent infestation foci north of the TEQA represent distinct genetic groups as shown by Bayesian population assignment methods (Busch et al., 2014). The source of these two genetic groups was not detected in southern Texas, suggesting that their origins may be genetically independent tick populations outside of Texas. Interestingly, one of the new infestations was resistant to permethrin (Stone et al., 2014). The most likely source of this genetic group is Mexico, where permethrin-resistant tick populations have increased dramatically since the introduction of this acaricide in the 1980s (Rosario-Cruz et al., 2009; Santamaria and Fragoso, 1994).

Although cattle are the primary host of *R. annulatus*, *R. australis*, and *R. microplus*, these ticks are highly invasive parasites that can use alternative hosts such as wild ungulates to complete their life cycle. Well-studied examples include white-tailed deer and exotic red deer in North America and rusa deer (*Cervus timorensis*) on New Caledonia. Very low levels of genetic population structure have been observed in *R. australis* (published as *R. microplus*) from across this island using eight microsatellite loci, most likely resulting from the bottleneck that accompanied the recent (1942) introduction of ticks from Australia (Koffi et al., 2006b). A small but significant level of genetic structure can be observed between ticks from rusa deer versus domestic cattle (De Meeus et al., 2010). This result suggests swift sympatric adaptation is underway that could lead to the formation of host-specific races. In contrast, the opposite pattern has been observed in Texas, where *R. microplus* ticks from white-tailed deer and cattle sampled from the same pastures do not show evidence of genetic divergence (Busch et al., 2014). Wild ungulates that occur in regions invaded by cattle fever ticks have become a major inhibition to eradication programs in places like Texas and Mexico, because these alternative hosts are difficult to regulate and treat with acaricides (Pound et al., 2010; Rodriguez-Vivas et al., 2014).

In Australia, Cutullé et al. looked at genetic differences between *R. australis* populations in two regions: Queensland, where this cattle tick is now endemic, and New South Wales, where tick outbreaks occur but are aggressively eradicated (Cutullé et al., 2009). No significant differences were found between these regions using 11 microsatellite markers even though some of the sampled populations were known to have acaricide resistance (most were susceptible). On the other hand, significant differentiation among populations within each region was found, suggesting restrictions to local gene flow.

It is worthwhile to note that the geographical scale of invasion by *R. australis* is much larger in Australia compared to New Caledonia and that genetic structure is likely to be correspondingly greater at the continental scale. Human practices in the movement of cattle are inherently different in each system and this can be expected to influence the genetic patterns found in these locations. Cattle markets in New Caledonia are concentrated mostly in one town, Bourail, and herds from throughout the island are transported to and from this location (Koffi et al., 2006b). Despite a great potential for genetic admixture among tick subpopulations that use cattle, ticks sampled from cattle demonstrate a low but significant pattern of isolation-by-distance, which the authors attribute to large subpopulation sizes and local tick dispersal mediated by cattle rotation among neighboring pastures (De Meeus et al.,

2010; Koffi et al., 2006b). Pairwise estimates of F_{ST} on this island range from 0 to 0.08. In contrast, the range of pairwise F_{ST} values was much greater (0.01–0.46) among *R. australis* populations from Australia (Cutullé et al., 2009).

Compared to ticks on cattle, isolation-by-distance is much stronger in ticks collected from wild Rusa deer in New Caledonia (De Meeus et al., 2010). This appears to be an outcome of their much smaller effective population size (N_e) relative to the tick subpopulation on cattle, which results in greater genetic drift in the tick subpopulation on deer. This study system demonstrates the importance of both genetic drift and dispersal in shaping the isolation-by-distance signature (Chevillon et al., 2013). Another valuable insight gained from studying New Caledonian ticks is that a large variance in adult reproductive success leads to inbreeding, possibly because only a few large sibling groups successfully infest any given cattle herd (Chevillon et al., 2013, 2007).

Overall, host mobility alone (generally moderate to high) probably does not explain the patterns of genetic structure observed in *Rhipicephalus* cattle ticks, since genetic structure appears to vary with each new invasion. It is possible that the life strategy of being a single-host tick may increase genetic structure, or that genetic bottlenecks in newly established infestations may inflate structure due to genetic drift. Also, humans play a significant role in the distribution of these ticks by determining the distance and frequency of cattle (and tick) movement and introducing selective pressures, such as acaricides (Miller et al., 1999).

4. Influence of life history on genetic structure

In the past, ticks were often grouped with other parasites or treated as one large indistinct group. Nevertheless, ticks are a highly diverse group of ectoparasites and many species have peculiar life-history strategies or evolutionary pressures that are expected to leave distinct genetic signatures. An early review of tick allozyme studies (Hilburn and Sattler, 1986a) proposed that the effects of host mobility, population size, and degree of host specificity would be more important for ticks than spatial environmental heterogeneity, as proposed for parasites in general (Price, 1977). It is true that ticks cannot disperse far by themselves (a few meters at most) and gene flow will be highly influenced by the movement capacity of their hosts. Yet behavioral factors of ticks will modify the effect of host movement on tick dispersal. Ticks associated with a mobile host might not actually disperse long distances if feeding times are short or if they use the host during a period of inactivity (such as nidicolous ticks that are mainly associated with nests or burrows) or a season of high site fidelity (such as during nesting or breeding periods).

Ticks that feed on a host species with high mobility might be expected to display relatively high gene flow and low levels of population structure. Tick species associated with hosts that have large ranges will likely be dispersed throughout the host range over time. Furthermore, human transportation of hosts, particularly cattle, horses, and dogs, can be a significant factor that affects gene flow at a regional scale or even leads to ticks dispersing between continents. If tick dispersal is tightly correlated with host movement, then host mobility should serve as a useful surrogate of genetic structure. However, ticks will only benefit from host movement while they are present on the host (i.e. during feeding). A number of other biological factors could play important roles in limiting tick dispersal.

Ticks can be classified as nidicolous (nest-dwelling) or non-nidicolous (field-dwelling), which differ in their mobility and choice of living site. Thus, it is possible that nidicolous behavior will lead to stronger population structure. Nidicolous species (e.g., *I. arboricola* and *I. texanus*) spend most of their time in host-occupied sites such as dens and nests (Anderson and Magnarelli, 2008). The mobility of nest dwellers should be relatively low given that they only occupy places where mates, food, and a favorable habitat are all available. Parthenogenesis is known for at least one nidicolous species, *Ixodes jellisoni* (Lane et al., 1999), and this reproductive strategy should further increase population structure. In contrast, non-nidicolous ticks (such as many *Dermacentor* and *Amblyomma* species) are spread more widely in the field and alternate between times spent feeding on the host and time spent free in the environment. Field dwellers are not limited to harborage sites near dens or nests and, therefore, disperse with fewer restrictions once they find a host. For these reasons, it is reasonable to predict that population structure will be stronger in nidiculous than non-nidicolous ticks. It is also likely that tick groups occupying one nest will be more closely related to each other than to those found in another nest.

Feeding times should influence tick movement and resulting genetic structure. Soft ticks (family Argasidae) typically feed rapidly (20–70 min) so they are less likely to be moved around by their hosts, whereas hard ticks (Ixodidae) feed slowly over a period of days or weeks (Anderson and Magnarelli, 2008; Barker and Murrell, 2002; Sonenshine, 1993). Thus, soft ticks should present relatively greater levels of population structure whereas hard ticks will experience more gene flow and lower levels of structure. Both Argasid and Ixodid ticks generally require a blood meal for reproduction and before proceeding from one life stage to the next (Anderson and Magnarelli, 2008); however, they are different in other aspects. The life cycle of soft ticks consists of four stages: egg, larvae, nymph (with up to eight instars), and adult, with the possibility of multiple blood meals during each post-hatching stage. Hard ticks present the same life stages as soft ticks except that the nymph stage has only one instar and a single blood meal is taken in each stage. Based on these differences in feeding behavior, we predict that most soft ticks will experience relatively low levels of gene flow accompanied by high population structure. Of course exceptions can be found, such as *Ornithodoros* species with larvae that feed for long periods of time on seabirds and *Otobius megnini* (the spinose ear tick), which can feed for over 6 months on highly mobile hosts such as livestock and wild ungulates (Apanaskevich and Oliver, 2014; Hooker et al., 1912).

Ticks with strong host specificity will likely display greater genetic structure than those that use a range of host species. In other words, one-host species should have greater genetic structure and less gene flow than those that use two or three host species. One-host ticks remain on a single host for all of their blood meals, dropping to the ground to lay eggs only after completion of the adult blood meal. For example, *R. microplus* spends a large part of its life on a single host individual, usually a domestic cow (*Bos taurus*/*Bos indicus*). Once larvae acquire a host they remain on it to take larval, nymphal, and adult blood meals; mating occurs on the host and females detach only after repletion to lay their eggs on the ground. In contrast, three-host ticks (such as many *Dermacentor* species) feed on a different host in each life stage, dropping to the ground to molt between each host (Anderson and Magnarelli, 2008; Sonenshine, 1993). Each life stage attaches to a host to feed and then drops off to molt in the environment. Depending on the species, adults can mate on or off their host. As stated previously, mobility of the host influences dispersal of the ticks only as long as they are attached to the host. Because of this we expect that, in general, ticks that use one host species will have stronger population differentiation and less gene flow than those using two or three host species, but of course this depends on the mobility of the hosts at each life stage. Selecting a single host may be advantageous because the risk of questing at multiple points of the life cycle is eliminated. Interestingly, this type of selective advantage may also predispose single-host ticks to host-race formation, as seems to be occurring in *R. australis* from

New Caledonia (De Meeus et al., 2010). Multi-host ticks can also develop specificity to a single host species, such as *I. uriae* (Dietrich et al., 2014) and possibly *O. capensis* (Gomez-Diaz et al., 2012). Regardless of how host races develop, genetic structure among races should increase over time because of limited genetic exchange.

Admittedly, these predictions based on life history are very broad. However, we feel it is important for researchers to recognize that tick dispersal is often limited by behavior and life cycle strategies, and that restricted dispersal should lead to a recognizable genetic signature in tick populations. For instance, we expect that a nidicolous species of soft tick with short feeding times and high specificity for a single host should accumulate greater genetic structure over time than a field-dwelling hard tick species that feeds for weeks at a time and can use multiple host species. We feel that including biological considerations will assist in the experimental design of future investigations and lead to key advances in the growing field of vector population genetics.

Two additional factors (both stochastic) were found to exert an influence on the genetic structure of certain tick species. The first is that individual hosts can be infested with a disproportionately large number of related individuals (sibling groups), which will lead to inbred progeny. Researchers who investigated ticks on a fine spatial scale commonly detected inbreeding when using an appropriate marker (e.g., microsatellites). In studies of *Dermacentor*, *Bothriocroton*, *Ixodes*, and *Rhipicephalus* populations, inbreeding was inferred to be the result of high breeding success in a small number of sibling groups. This equates to a high variance in the reproductive success of individual adults, which produces an ephemeral pattern of fine-scale genetic structure due to inbreeding. The other important factor acting on many tick populations is human-mediated transport of ticks and acaricide selection pressure (Stone et al., 2014). Humans can play a significant role in determining tick genetic structure, especially in those species that infest domesticated animals. Human activities obscure the genetic signature that would normally be found in free-living tick populations, and this needs to be considered when making inferences from genetic data. Genetic work on ticks is increasing and we fully expect future investigations will lead to new testable hypotheses and predictions about what shapes genetic variation in these vectors.

5. Molecular tools for population genetics

Population genetic studies of vectors, or any organism, have two major basic goals: to assess levels of genetic variation and to determine how and why it is distributed over space and time. To answer these questions it is first necessary to sample vector populations across an appropriate geographic range and/or temporal scale (Lowe et al., 2004). Choosing an adequate sample size that allows robust statistical inferences is not always straightforward. A random sample of 25–30 individuals per population is often used in initial surveys and can provide a reasonable estimate of genetic variation. However, the appropriate sample size actually depends on the question being asked, the number of loci and their allelic diversity, the uniformity of allele frequencies, and the magnitude of genetic differentiation among populations. For instance, when F_{ST} among simulated subpopulations is 0.05 or greater, a sample of 20 individuals evaluated at 16 microsatellite loci will accurately estimate the level of genetic structure (Kalinowski, 2005). Obviously, more individuals need to be sampled as the level of differentiation decreases, and Kalinowski found that 100 individuals per population are needed to retain power when F_{ST} is set to 0.01 (with all other parameters unchanged). Another power analysis based on simulations suggested that sample sizes of 50–100 individuals per population should be enough to detect low levels of structure ($F_{ST} \sim 0.01$) when up to 20 loci are used (Ryman et al., 2006). Because a wide variety of questions and statistical tests exist for population genetic studies, Ryman and coauthors suggest a power analysis before initiating genetic studies. They have implemented a user-friendly method to assess the power needed to detect genetic differentiation in the program POWSIM (Ryman and Palm, 2006). In the end, the sample size from field collections will be limited by the finite number of ticks that can be reliably sampled per host or from dragging suitable habitat patches. The availability of funds and lab resources will also determine realistic sample sizes for many genetic studies.

How does one choose an appropriate molecular marker? A number of books (Avise, 2004; Lowe et al., 2004) and in-depth reviews (De Meeûs et al., 2007; Meudt and Clarke, 2007; Selkoe and Toonen, 2006; Sunnucks, 2000; Vignal et al., 2002) are available on this topic. These previous works have identified a number of desirable characteristics in molecular markers used for population genetics. Ideally such markers should be DNA-based, amplifiable using PCR, transferable across species, highly variable, and selectively neutral (Lowe et al., 2004; Schlotterer, 2004; Sunnucks, 2000; Vignal et al., 2002). General considerations for choosing an appropriate marker include what hypotheses are being tested, whether DNA sequence information is available or not, and the variability and resolving power of the marker system (Table 2) (Lowe et al., 2004).

Factors specific to ticks include the amount of tissue available for each individual (most have small body sizes), the quality of preserved material (poorly preserved specimens will not support all molecular methods, especially allozymes), the type of sample (collections taken from a host versus dragging methods or sampling from harborage sites near host dens and nests), and the life stage (collections of larvae may over-represent siblings). In addition, a large arsenal of molecular tools has been developed to screen ticks for pathogens (Sparagano et al., 1999). It is important to remember that there is no single ideal molecular marker for all population genetic studies, but some have proven to be more adequate than others. We will highlight both general and tick-specific considerations that may be helpful in deciding on an appropriate marker type for tick genetic studies.

Allozymes and isozymes possess two desirable attributes for assessing genetic variation: they are inexpensive and alleles are co-dominant (both copies are observable). However, they are generally not recommended for current population genetic studies. Particularly in ticks, allozyme usage is limited by the small body size of most life stages. Because males are smaller than females, a preference for analysis of females has prevailed in the past, which may limit population-level inferences to the sex under study.

Individual gene sequences provide greater resolution than protein data and have become widely popular in tick genetic studies. Mitochondrial genes such as 12S, 16S, CYTB, COI, and COIII and nuclear regions such as 18S, the ribosomal internal transcribed spacers (ITS1 and ITS2), and lysozyme (*lys*) mutate at a rate that is usually informative for species-level phylogenetics and taxonomy. These genes may also be used for broad biogeographic inferences and resolving major genetic discontinuities within a species. However, the level of resolution is usually not fine enough to detect recent divergence or questions related to selection. A potential problem with mtDNA data is the possibility of amplifying mitochondrial pseudogenes in nuclear genomes, also known as numts (Zhang and Hewitt, 1996, 2003). Methods to identify and deal with these pseudogenes have been identified for vertebrates (Triant and DeWoody, 2007). Other caveats in the usage of mtDNA is that it reflects the history of just one of the parents, thus reducing the effective population size fourfold from that of nuclear DNA and

also represents only one locus, which may confound relationships between populations (Zhang and Hewitt, 2003).

The most widely accepted markers for population genetic studies are microsatellites (Barbará et al., 2007; Väli et al., 2008). These are repeats of 2–5 nucleotides that are found across the genome. The main advantages of these loci are that they tend to be highly polymorphic and alleles are codominant. In contrast, some of their disadvantages are a high cost of development, high species specificity, and limitations for cross study comparisons (Table 2). The biggest caveat of microsatellite markers is the presence of null alleles that make heterozygous individuals appear as homozygotes, leading to underestimation of genetic variability and affecting F_{ST} calculations (Chapuis and Estoup, 2007; Jarne and Lagoda, 1996). To date, microsatellite primers have been developed for 12 tick species: *B. hydrosauri* (Guzinski et al., 2008), *D. albipictus* (Leo et al., 2012), *D. variabilis* (Dharmarajan et al., 2009a), *I. arboricola* (Van Houtte et al., 2013), *I. ricinus* (Delaye et al., 1998; Roed et al., 2006), *I. scapularis* (Fagerberg et al., 2001), *I. texanus* (Dharmarajan et al., 2009b), *I. uriae* (McCoy and Tirard, 2000), *R. annulatus* (Araya-Anchetta, 2012), *R. australis* (Chigagure et al., 2000; Cutullé et al., 2009; Koffi et al., 2006a), *R. microplus* (Busch et al., 2014), and *O. coriaceus* (Kirchoff et al., 2008). Many of these cross-amplify in closely related species and thus have broader utility for population genetic studies.

Several types of genetic markers are designed to provide information from many locations throughout the genome simultaneously. These include RFLPs (restriction fragment length polymorphisms, PCR-RFLPs, RAPDs (randomly amplified polymorphic DNA), and AFLPs (amplified fragment length polymorphisms). RFLPs identify genome-wide variation through mutations that alter the ability of restriction enzymes to cut DNA. Because it does not utilize PCR amplification, this technique requires a large amount of high quality genomic DNA (gDNA). To counter this problem, PCR- based RFLPs have been developed that amplify specific DNA fragments; these are subsequently cleaved with restriction enzymes. Popular PCR-RFLP targets in eukaryotes are mitochondrial genes (12S, 16S, and cytochrome oxidase genes) and ITS1/ITS2 of nuclear ribosomal genes (Lowe et al., 2004). Because a specific region of DNA is targeted, the number of genomic loci represented in most PCR-RFLP studies is limited. In contrast, hundreds of RAPD markers can generated using PCR. The advantage to this technique is that no previous knowledge of the genome is necessary. However, RAPDs have been heavily criticized because of their high error rates (up to 60% false bands), low reproducibility within and across laboratories, and difficulty of cross study comparison. For these reasons they are no longer considered to have much utility (Jones et al., 1997; Lowe et al., 2004; Mueller and Wolfenbarger, 1999; Schlotterer, 2004).

The AFLP technique was developed 20 years ago (Vos et al., 1995) and since then it has received a lot of focus from the population genetics community. This method is ideal for non-model organisms since it does not require previous knowledge of DNA sequences and thus AFLPs can be used with genetic samples of any organism, including ticks. Because alleles are dominant, they must be scored as 'present' or 'absent'. AFLPs can easily produce hundreds of loci and the level of resolution can be adjusted by modifying the selective primers to expand or reduce the final number of loci (Bonin et al., 2007). Another advantage of AFLPs is that loci can be found in coding and non-coding regions; thus, AFLP loci can be neutral or under selection (Mueller and Wolfenbarger, 1999). This creates the opportunity to design population genomic studies of selection, where populations can be sampled across a variable that is suspected of exerting a selective force on the species (Luikart et al., 2003) (Bonin et al., 2006). A major disadvantage of AFLPs is the ubiquity of homoplasy found across loci within sets of primers (Bonin et al., 2007; Caballero et al., 2008). Hence, it is important to explore levels of homoplasy in any AFLP dataset either by means of sequencing (Bonin et al., 2007) or PCR (O'Hanlon and Peakall, 2000). Tick researchers must also pay attention to the possibility of non-specific amplifications due to host blood and the gut microbial community. There are three possible ways to prevent this issue and at least one or a combination of them should be used. First, DNA extractions can use the tick body without including the gut. Second, host DNA can be run as a control for amplification of non-specific loci. Third, if samples are collected on the host the collection can be done at an early stage before they feed on the host. Ticks that are collected in the field by dragging are usually safe from host contamination because they quickly digest their previous bloodmeal. All ticks should be surface sterilized to reduce contamination from environmental microbes.

Single nucleotide polymorphisms (SNPs) result from a nucleotide change at a single site and are rapidly growing in popularity as molecular markers. A recent comparison of partial genome sequences from *I. scapularis* ($n = 40$) suggests that ticks may have some of the highest SNP densities of all eukaryotes, about one SNP per 14 nucleotide positions (Van Zee et al., 2013). Although four different nucleotide states are possible at any given position, SNPs usually consist of biallelic changes due to their low substitution rate (Vignal et al., 2002). SNPs are found anywhere in the genome in both coding and non-coding regions, which means they can be neutral or under selection. They segregate independently (though they can be linked) and are codominant. SNPs also exhibit a lower mutation rate than microsatellites, around the range of 10^{-8}–10^{-10} per nucleotide per generation, which makes them less prone to exhibit homoplasy but also reduces resolution power (Morin et al., 2004; Vignal et al., 2002). The greatest disadvantage of SNPs is ascertainment bias, which occurs during the marker discovery and selection process. Genetic diversity and population differentiation estimates can be affected by this problem, resulting in misleading conclusions (Pearson et al., 2004). A way to avoid this issue is to select a discovery panel representative of the range of the species (or at least across the desired range of the populations), and by selecting loci with different levels of heterozygosity (Morin et al., 2004). Next-generation sequencing approaches have greatly facilitated the use of SNPs in studies of natural populations (Hagen et al., 2013). Thousands of SNPs can be identified for population genomic studies, similar to AFLPs. As the cost of genome sequencing continues to drop, we predict that SNPs may soon become the most widely used marker type in tick genetic studies.

Population genetic studies offer a large number of powerful analyses to infer levels of genetic connectivity among tick populations (Excoffier and Heckel, 2006). Estimates of population differentiation such as F_{ST}, θ, or φ_{ST} from AMOVA (Excoffier et al., 1992; Weir and Cockerham, 1984) can be used to identify host races and non-random mating patterns (McCoy et al., 2003a,b). Genetic structuring in ticks can occur in a hierarchical fashion, such that variation is partitioned among individuals, infrapopulations (the ticks found on a single host), populations, and even larger scales (regional or continental). Detecting hierarchical levels of genetic subdivision can be accomplished using HIERFSTAT (Goudet, 2005). Identifying major genetic groups and migrants is also possible with Bayesian population assignment (Corander et al., 2003; Manel et al., 2005; Piry et al., 2004; Pritchard et al., 2000). Other important types of genetic analyses include estimates of effective population size (N_e) (Tallmon et al., 2008; Waples and Do, 2008), occurrence of past bottlenecks (Beaumont, 1999; Cornuet and Luikart, 1996; Garza and Williamson, 2001; Luikart et al., 1998), sex-biased dispersal (Fontanillas et al., 2004; Vitalis, 2002), isolation by distance (Bohonak, 2002; Rousset, 1997), genealogy and parentage (Marshall et al., 1998), and/or selection experiments (De Meeûs et al., 2007). These analyses can provide estimates of dispersal in the study species (how far, how much,

and when), identify connectivity or isolation among areas within the range of study, and shed light on vector evolution in populations (McCoy, 2008; Tabachnick and Black, 1995b). Estimates of N_e can shed light on the size of invasive tick populations (e.g., *R. microplus*, *R. australis*, and *A. variegatum*) and we encourage researchers to include these estimates in their studies. We also propose that every study present estimates of genetic diversity, such as the number of alleles, effective number of alleles (weighted by the smallest sample size), observed heterozygosity, and expected heterozygosity at the population and global levels.

In reviewing the available literature from tick population genetics, it is clear that results were heavily influenced by sampling scheme. Analyzing genetic data at different geographical scales within a single study system can reveal intriguing aspects of tick and host ecology. For example, at a scale that covered the entire range of its host, the black-legged kittiwake (*R. tridactila*), population structure in *I. uriae* was high and uncorrelated to host population structure (McCoy et al., 2003a). At a regional scale, tick population structure was low and dependent on host species, whereas at a local scale population structure was explained by tick life history traits (McCoy et al., 2005b, 2003b). Conclusions drawn from a population genetics study are therefore strictly dependent on the scale chosen, which should also be considered when making inferences about the pathogens transmitted by tick vectors. It is our hope that genetic studies in the future will evaluate tick genetic structure at multiple scales and take into account the natural history of each species. When these factors become an important part of a study design, the strength of inference should greatly increase.

6. Conclusions

Ticks are highly successful hematophagous ectoparasites and perhaps it is no surprise that they also serve as vectors for numerous pathogens that affect humans and animals. They are important players in the vector–host–pathogen triangle in which all members interact and affect each other's success (Jongejan et al., 2007). An important question in vector studies is whether the genetic structure of the arthropod can inform patterns of disease transmission. It has been speculated that genetically distinct tick populations may possess distinct vector competency, and that geographic variation within a tick species can influence the ability to acquire, maintain, and transmit pathogens (McLain et al., 1995). Differences in vector competency for the protozoan *T. cervi* have been demonstrated between two populations of *A. americanum* (one wild collection and one laboratory strain) experimentally fed on deer (Reichard et al., 2005; Reichard and Kocan, 2006). Although the specific genetic mechanism for this difference remains unknown, an obvious follow-up to this study would be to test this vector and parasite from geographically diverse locations. A genetic factor determining the susceptibility of *R. appendiculatus* laboratory strains to infection with *T. parva* is being studied (Buscher and Tangus, 1986; Kubasu, 1992; Odongo et al., 2009), but not yet in a population genetics framework. These studies point out the possibility of biological limitations to disease transmission and provide intriguing targets for future studies.

Strong population structure in certain ticks, such as *O. coriaceus*, indicates that some vectors do not disperse long distances even when they use highly mobile hosts (Teglas et al., 2005). This suggests that the movement of infected hosts, rather than infected ticks, may be more important for long-distance disease transmission in these systems. In such cases, disease control efforts could be focused more on screening for infected hosts (cattle) rather than detecting ticks. In contrast, a close relationship between genetic patterns in *I. scapularis* and *B. burgdorferi* demonstrates temporal and spatial coevolution in the northeastern U.S. (Qiu et al., 2002). This situation may be tied to long-distance transmission of the tick vector and control efforts should take this into account. Ultimately, genetic studies improve our comprehension of tick movements, which is relevant to understanding pathogen dispersal and the development of control strategies (De Meeûs et al., 2007; McCoy, 2008; Tabachnick and Black, 1995b). Key information on the distribution of genetic variance may help to explain differences in vectorial capacity, aid in revealing cryptic species or host-races, and deepen our understanding of disease epidemiology.

To date, tick population genetic studies have addressed a series of questions that cannot be addressed with ecological methods alone. Levels of genetic variation have been studied on both a temporal (Busch et al., 2014; Dharmarajan et al., 2010b; Healy, 1979b) and spatial dimension (Koffi et al., 2006b; Lampo et al., 1998; Paulauskas et al., 2006; Vial et al., 2006). Sex-biased distribution of genetic variation (De Meeûs et al., 2002; Healy, 1979b) and the formation of host specific races within a tick species (McCoy et al., 2003a) have also been investigated. However, these studies represent only the tip of the iceberg for understanding the patterns and processes leading to genetic variation in ticks. Future population genetic studies will provide critical insights into the interactions among ticks, the pathogens they transmit, and the hosts on which they feed.

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