Contrasting emergence of Lyme disease across ecosystems

Atle Mysterud1, William Ryan Easterday1, Vetle Malmer Stigum1, Anders Bjørnsgaard Aas1,2, Erling L. Meisingset3 & Hildegunn Viljugrein1,4

Global environmental changes are causing Lyme disease to emerge in Europe. The life cycle of *Ixodes ricinus*, the tick vector of Lyme disease, involves an ontogenetic niche shift, from the larval and nymphal stages utilizing a wide range of hosts, picking up the pathogens causing Lyme disease from small vertebrates, to the adult stage depending on larger (non-transmission) hosts, typically deer. Because of this complexity the role of different host species for emergence of Lyme disease remains controversial. Here, by analysing long-term data on incidence in humans over a broad geographical scale in Norway, we show that both high spatial and temporal deer population density increase Lyme disease incidence. However, the trajectories of deer population sizes play an overall limited role for the recent emergence of the disease. Our study suggests that managing deer populations will have some effect on disease incidence, but that Lyme disease may nevertheless increase as multiple drivers are involved.

1 Centre for Ecological and Evolutionary Synthesis (CEES), Department of Biosciences, University of Oslo, PO Box 1066 Blindern, NO-0316 Oslo, Norway.  
2 Section for Genetics and Evolutionary Biology (EVOGENE), Department of Biosciences, University of Oslo, PO Box 1066 Blindern, NO-0316 Oslo, Norway.  
3 Norwegian Institute of Bioeconomy Research, Division of Forest and Forest Resources, Tingvoll Gard, NO-6630 Tingvoll, Norway.  
4 Norwegian Veterinary Institute, PO Box 750 Sentrum, NO-0106 Oslo, Norway. Correspondence and requests for materials should be addressed to A.M. (email: atle.mysterud@ibv.uio.no).
Global environmental changes affect several drivers of emergence of vector-borne diseases that are typically difficult to unravel in terms of their role in complex epidemiological networks. The blacklegged tick *Ixodes scapularis* in North America and the sheep tick *Ixodes ricinus* in Europe are the most important vectors of spirochetes within the *Borrelia burgdorferi* sensu lato (s.l) complex, the causal agent of Lyme disease (LD) and, of several other pathogens causing human and animal diseases. Climate warming and land use change are contributing to the tick distribution to expand upwards in elevation and towards higher latitudes in Northern Europe. The hazard of LD for humans is linked to both vector abundance and infection prevalence. These factors are determined by the abundance and composition of different-sized vertebrate hosts suitable for different life stages of ticks and whether they are competent reservoirs of the pathogen.

The life cycle of these ticks involves three stages, each require a blood meal to moult into the next stage or reproduce. Larvae and nymphs parasitize a wide range of different sized vertebrate hosts, picking up the pathogen from small vertebrates, while adult female ticks require a meal from a large vertebrate (> 1 kg) for reproduction. Hence, the ontogenetic niche shift leads to a complicated tick population regulation that depends on the availability of both large (critical for adults) and small (used by larvae) hosts. In North America, access to white-tailed deer *Odocoileus virginianus* or other large hosts are required for tick reproduction, but annual fluctuations in deer numbers play a minor role in limiting LD under conditions of moderate to high deer density. The red deer *Cervus elaphus* and roe deer *Capreolus capreolus* have markedly expanded over recent decades and are the most widely distributed large hosts available for ticks in Europe. There is evidence that deer in Europe amplify tick populations, but the net effect on disease risk and incidence remains to be established.

The ratio of competent to incompetent transmission hosts is central for the prevalence of pathogens in the tick population. In the United States, the main reservoirs of *B. burgdorferi* s.l. is the white-footed mouse *Peromyscus leucopus*, eastern chipmunk *Tamias striatus* and shrews with a wide distribution. Hence, with only three main transmission hosts, any additional host species in the community may lead to a higher proportion of larvae feeding on non-transmission hosts, lowering pathogen prevalence in the tick population. This is termed a dilution effect or buffering of disease with increasing biodiversity. Deer are regarded as incompetent transmission hosts, but must feed a sufficiently large amount of tick larvae to cause dilution of the pathogen in the vector population to be termed a dilution host.

In the United States, LD has been central to the biodiversity buffers disease paradigm. The effectiveness of biodiversity diluting LD and other diseases has been heavily debated. The buffering effect of biodiversity for LD has not been tested outside the United States. The LD systems differ across continents in terms of hosts, vectors and pathogens. In Europe, there are several pathogenic genospecies of *B. burgdorferi* s.l. Most human cases in Europe are caused by *Borrelia afzelii* having a small mammal reservoir and *Borrelia garinii* having a bird reservoir, while *B. burgdorferi* sensu stricto causing LD in North America is present, but less common. The larger base of reservoir hosts including both small mammals and birds in Europe suggests that dilution due to biodiversity is less likely, and that rather the relative densities of transmission to non-transmission hosts will determine hazard. However, long-term and spatially extensive empirical data have been lacking from Northern Europe where LD is emerging.

We here analyse long-term data (1991–2012) on LD incidence in humans over a broad geographical scale (416 municipalities in Norway; latitudinal range 57° 58’ N–71° 08’ N) at the northern limits covering areas with recently emerging LD. This is made possible since systemic LD has status as a notifiable disease in Norway. We first tested if LD was emerging (an increase in incidence over time) and whether there was regional variation in level of emergence. An important aspect of this study is its geographical coverage because it includes regions with contrasting trajectories of population sizes of deer, in part, by different harvesting management regimes. These regions differ sufficiently to classify them as different ecosystems (see Methods).

Hence, we relate the incidence of LD to the spatial contrasts and temporal variation in deer population density across and within four regions. In particular, we highlight the contrast between region West and South, with a steep increase and a decline in deer numbers over the monitoring period, respectively. We hypothesized that LD incidence may increase with higher deer population density in (P1a) space and (P1b) time, as deer are important tick reproduction hosts (LD-deer limitation hypothesis or H1 below.) We further tested whether an increasing deer density caused endemic LD incidence over time, that is, an emergence, together with other drivers (P2) or whether an increasing deer density was sufficiently strong to (P3a) fully explain the emergence across ecosystems. Finally, we tested whether biodiversity buffered LD incidence, predicting (P3b) a lower LD incidence in areas with higher mammalian host species richness (biodiversity buffers disease hypothesis or H2 below). We statistically took into account the impact of climate, land use, a proxy for rodent abundances, and a proxy for human connectedness to the landscape. Finally, in the West region, which has experienced a marked increase in red deer, we used experimental data (exclosures), spatially extensive flagging data for questing (that is, host seeking) sheep tick abundance, tick load on red deer ears and pathogen prevalence to document the mechanisms by which red deer density might affect LD incidence. We find that both high spatial and temporal deer population densities increase LD incidence, but that the trajectories of deer population sizes could not fully explain the recent emergence of LD. We conclude that

| Parameter | Estimate | S.E. | Z | P |
|-----------|----------|-----|---|---|
| Intercept | −11.831  | 0.149 | −79.4 | <0.001 |
| log(spatial deer density + 0.1) | 0.711 | 0.103 | 6.91 | <0.001 |
| Temporal deer density | 0.085 | 0.039 | 2.20 | 0.028 |
| Region (North versus West) | −1.174 | 0.305 | −3.85 | <0.001 |
| Region (South versus West) | 0.830 | 0.201 | 4.12 | <0.001 |
| Year | 0.638 | 0.066 | 9.69 | <0.001 |
| Spatial autocorrelation (lag 1) | 0.276 | 0.083 | 3.33 | <0.001 |
| Latitude | −0.500 | 0.172 | −2.90 | 0.004 |
| NAO-JJA (lag 1) | 0.197 | 0.033 | 5.95 | <0.001 |
| NAO-MAM (lag 2) | −0.125 | 0.026 | −4.79 | <0.001 |
| NAO-DJF (lag 1) | 0.131 | 0.026 | 4.98 | <0.001 |
| Year × region (North versus West) | 0.170 | 0.152 | 1.12 | 0.262 |
| Year × region (South versus West) | −0.331 | 0.073 | 4.56 | <0.001 |
| Year × region (East versus West) | 0.035 | 0.110 | 0.32 | 0.749 |

DIF: December–January–February; JJA: June–July–August; MAM: March–April–May; NAO, North Atlantic Oscillations. Parameter estimates of the most parsimonious negative binomial model explaining the incidence of Lyme disease during 1991–2012 in Norway. All numeric variables were standardized. Baseline for region is West.
managing deer populations will have some effect on LD incidence, but that such actions are not necessarily sufficient to hinder emergence as multiple drivers are involved.

**Results**

**Analysis of LD incidence.** The incidence of LD was clearly emerging, but with significant differences in yearly increases across the four regions of Norway (Table 1). LD incidence was related to both the spatial population density of all deer species combined (referred to as deer, $P_{1a}$, Fig. 1) and the temporal component (annual residual relative to mean density on a log scale) of the deer population ($P_{1b}$, Fig. 2), providing support for two of the predictions from the LD-deer limitation hypothesis. First, the temporal increase of the deer population size was

![Figure 1](image_url)
Correlated with the emergence of LD in the region West (Fig. 2c, partial support of $P_{1c}$, see Methods for the working definition of West and all other regional divisions), which has shown a marked density increase in red deer in recent decades (Fig. 2a, Supplementary Table 1 and Supplementary Note 1). In contrast, areas along the South coast and towards the East, which are dominated by roe deer and moose (Alces alces), have seen a decline in their numbers (Fig. 2b). Here incidence was not linked to temporal population density trajectory of all deer species combined (Fig. 2d). However, even for the West region, only a small proportion of the LD emergence could be attributed to the red deer density increase (Fig. 2c; rejecting $P_{1d}$), suggesting that other drivers were involved and important for emergence. There was no evidence that LD was buffered by biodiversity measured as mammal species richness (rejecting $H_{2}$: Table 2). Regions East and North have much lower LD incidence compared with West and South (Table 1), the latter two regions thus providing our main comparison of how deer density correlates with patterns of LD emergence.

The incidence of LD was correlated with proxies of spatial (the distance to the coast) and temporal variation in climate (annual variation in the North Atlantic Oscillation, Table 1). Controlling for the distance to the coast was important as ticks thrive along the coastline, but this variable is also correlated to a higher spatial density of deer (particularly in region South; Supplementary Table 2). The spatial contrast in deer density had a mainly positive effect on the incidence of LD while accounting for distance to the coast (Table 1). However, the effect of spatial deer density is slightly higher than that from the coast is removed from the model (estimated effect 0.711 versus 1.021). Similarly, covariation may cause the yearly trend to capture part of the temporal deer density effect, particularly in the region West. Hence, if running a model using a common year trend for all regions (that is, excluding 'year × region'), the estimated effect of temporal deer density increases (estimated effect 0.085 versus 0.178). Thus, our estimates of the effect of spatial and temporal deer density effects are conservative. Some variables of land use were also important (Table 1), but we failed to link LD to a proxy of rodent abundances, as adding such a proxy did not improve explanatory power of the model based on Akaike information criterion (AIC; Table 2).

We cannot exclude that the increased awareness of LD is leading to higher rates of reporting over time and hence play a role in the emergence pattern. Increased reporting might play a role in the first few years, but rerunning models from 1995 onwards gave qualitatively similar results. Also, the emergence of babesiosis21, anaplasmosis and tick-borne encephalitis22 suggests the existence of some common environmental driver, and clearly the spatial effect of deer density and temporal effect of deer density after controlling for year trends cannot be explained by a potential reporting bias. In the United States, the true LD incidence was 10-fold higher than the official number of reported cases23. As a result of our statistical data only include systemic LD infections, we are likely only seeing the tip of the iceberg in terms of LD incidence.

**Tick population and deer density in region West.** The red deer populations in the West region of Norway have rapidly increased in the last decades (Fig. 2a and Supplementary Table 1). We focused on determining the mechanisms behind the correlation between deer density and LD incidence in the two largest counties...
Table 2 | Model selection of Lyme disease incidence.

| Excluding                                      | ΔAIC |
|-----------------------------------------------|------|
| Region                                        | 78.5 |
| Spatial deer density                          | 42.8 |
| Temporal deer density                         | 2.9  |
| Year × region                                 | 25.3 |
| Year × region + temporal deer density         | 52.1 |
| Year                                          | 134.1|
| Square root (distance to coast)               | 76.3 |
| Square root (proportion of residential settlement area) | 53.6 |
| Latitude                                      | 6.6  |
| NAO-DJF (lag 1)                               | 62.4 |
| NAO-MAM (lag 2)                               | 20.9 |
| NAO-JJA (lag 1)                               | 40.9 |
| NAO-DJF (lag 1) + NAO-MAM (lag 2) + NAO-JJA (lag 1) | 67.3 |
| NAO (all) + temporal deer density + year × region | 107.2 |
| NAO (all) + year × temporal deer density       | 214.4|
| Spatial autocorrelation (lag 1)               | 9.3  |
| All fixed effects (intercept and random effects only) | 708.2 |
| All fixed and random effects (intercept only)  | 2,259.6|

| Replacing                                      |      |
| Log (deer density) replacing spatial deer density + temporal deer density | 7.1  |
| Temporal deer density (lag 1) replacing temporal deer density | −0.8 |

| Adding                                          |      |
| Temporal autocorrelation                       | −0.8 |
| Proportion of people living in city            | 1.1  |
| Proportion of agricultural area                | −5.1 |
| Proportion of forest                           | 1.6  |
| Mammal species richness                        | 0.6  |
| Rodent abundance index (lag 1)                 | 0.3  |

|                |      |
| Difference in the AIC, ΔAIC, between the top-ranked (best) model in main Table 1 (ΔAIC < 0.0312) and a model excluding, replacing or adding indicated explanatory variable. Observation unit is number of LD cases in each municipality and year. There are in total 19 years and 416 municipalities with complete set of covariates giving 7,904 observation units over which 2,007 LD cases were found. Top-ranked model is the most parsimonious model with lowest AIC value or AIC not larger than 5 to other models. Parameters directly relevant for the testing of hypothesis were added if improving model fit and retained if significant. Spatial values are at scale of municipality, while temporal values are at scale of year lagged one (lag 1) or two (lag 2) years before to match life cycle of ticks. |

1The top-ranked (best) model is the one parametrized in Table 1 and used as a starting point. We then challenge this model by either excluding those explanatory variables that are in the best model, adding those explanatory variables that are not in the best model, or replacing explanatory variables in the best model with related, correlated terms (that should not be in the same model).

2Compared with top-ranked model run on reduced data set (1 year lacking for this variable).

(Sogn and Fjordane, and More and Romsdal). The exclosure experiment showed that the complete eradication of deer resulted in significantly fewer questing nymphal ticks 3 years after the fences were erected (estimated mean density outside was 7.5 ticks versus 0.7 ticks per 20 m² inside exclosures, Fig. 3a, Z = 4.66, P < 0.001). These results indicate that hosts for adult females limit the tick populations where large vertebrates are totally absent.

The abundance of questing ticks was also linked to spatial contrasts of red deer density mainly caused by management in the West region of Norway (n = 23,298 nymphal ticks from 71 transects). At the finer scale of local management units (n = 21, mean size = 103.6 km²) in More and Romsdal County the abundance of questing ticks was positively related to the red deer density in spring (Fig. 3b and Table 3). While at the broader scale of municipality (n = 9, mean size = 305.2 km² removing mountain areas) in Sogn and Fjordane County (Table 3), where the contrasts in red deer density was about half of More and Romsdal County, the effect was weak and insignificant. There were more questing ticks in May than in August, and the abundance varied from year to year. The positive effect of red deer density in More and Romsdal County was robust when controlling for other environmental factors such as distance to the coast and slope (Table 3). However, distance to the coast and density of red deer is partially correlated; hence, removing distance to the coast as a covariate in the analysis amplified the effect of red deer density even further. Tick load (nymphs) on GPS-marked red deer (n = 49) also increased considerably at high red deer densities (Fig. 3c and Table 5).

Our last objective was to estimate the effect of red deer density on the pathogen prevalence in nymphal ticks at different temporal scales (n = 4,557; Table 6). During the main questing period in the spring (May), there was a reduction in pathogen (B. burgdorferi s.l.) prevalence with increasing red deer density (spatial contrast) in both counties. However, the effect was fairly weak (Fig. 3d, Supplementary Fig. 1 and Table 6). In contrast, there was no measurable effect of red deer density on pathogen prevalence in the late questing (August) at this high latitude (Table 6). The prevalence of the pathogen varied largely among years. On the basis of 6 years of data, we were able to link the high pathogen prevalence in nymphs to the high abundances of rodents the previous year in Sogn and Fjordane County, but only in the month of May (Table 6). Analytically combining functions on tick amplification (Tables 4 and 5) and pathogen prevalence (Table 6) to assess number of infected nymphs show that the estimated functions were quite variable and model dependent, thus the overall number of infected nymphs was weakly related or unrelated to red deer density depending on season and year.

Discussion

The main aim of this study was to test whether LD incidences at the northernmost distribution range in Europe have increased over time in recent decades (an emergence), whether patterns of increase differed across ecosystems, and the extent to which emergence could be linked to (H₂) altered abundances of deer.

Reduced abundances of questing ticks have been reported when comparing data from inside and outside deer exclosures in a number of cases15,24,25 or when completely removing deer from islands and thereby rendering ticks with no alternative large hosts25. This outcome is not surprising because the tick population would then be strongly limited by the absence of reproduction hosts for adult females12. However, for mainland populations, it is not feasible and often not desired to remove deer completely and there may be other large hosts present. Our study shows that reducing deer densities within the range currently observed in mainland areas would only marginally affect the incidence of LD. In the region South of Norway, temporal variation in deer densities could not explain LD emergence (Fig. 2b,d). In contrast, in the region West of Norway, increased red deer density played a role for LD emergence (Fig. 2a,c), and we could mechanistically link red deer density to tick amplification through both tick questing abundances and tick load on red deer (Fig. 3). However, temporal trajectories of deer population sizes could not explain all of the increase of LD over time, even in the West region (Fig. 2c), suggesting that another driver is important across the whole region, most likely a warmer climate. The increase in white-tailed deer in the United States was in early phases of recolonization suggesting that another driver is important across the whole region, most likely a warmer climate. The increase in white-tailed deer in the United States was in early phases of recolonization and population growth regarded as important for LD emergence26,27, while more recent studies find no relationship of LD risk12 or incidence13 unless very low deer densities are included in analyses. Our review of studies linking tick abundance
to actual deer densities provides further support for a stronger effect of deer when going from low to medium compared with from medium to high deer population density (Supplementary Table 3). Indeed, a common feature of studies reporting a positive link between tick abundance and deer density is that they include a wide range of densities from well below 10 deer per km² to quite high (Supplementary Table 3). Whether increased density of ticks with increased deer density leads to increased incidence of LD also depends on pathogen levels and exposure. The emergence of LD across contrasting temporal trajectories of deer density in Norway provides strong evidence that the emergence across northern Europe cannot be attributed to deer populations alone. Yet, deer density plays some role when comparing a wide range of densities including from low to high

**Figure 3 | Linking tick abundances and pathogen dilution to deer density.** Relationship between (a) the (log) abundance of questing tick nymphs inside and outside of deer exclosures (10 replicates), (b) the (log) abundance of questing tick nymphs (n = 11,216) and deer population density index, (c) the (log) nymphal tick load on red deer ears (n = 49) and deer population density index, and (d) the prevalence of *Borrelia burgdorferi* sensu lato in ticks (n = 3,324) and deer population density index along the west coast of Norway. For d, data points are proportional to (square root) sample size.

**Table 3 | Analysis of tick abundance in Møre og Romsdal County.**

| Parameter                          | Estimate | S.E.  | Z     | P      | ΔAIC |
|-----------------------------------|----------|-------|-------|--------|------|
| Intercept                         | -0.284   | 0.1808| 1.57  | 0.116  |      |
| Season (spring versus fall)       | 0.1124   | 0.0619| 1.82  | 0.069  | 1.3  |
| Elevation                         | -0.3930  | 0.1158| -3.39 | <0.001 | 9.1  |
| Distance to coast                 | -1.8587  | 0.3193| -5.82 | <0.001 | 34.3 |
| Year (2012 versus 2011)           | 0.0399   | 0.0743| 0.27  | 0.789  | 131.0|
| Year (2013 versus 2011)           | -0.8045  | 0.0786| -10.23| <0.001 |      |
| Slope                             | 0.3107   | 0.0055| 6.03  | <0.001 | 35.0 |
| log(spatial red deer density)     | 0.7778   | 0.2990| 2.60  | 0.009  | 2.2  |
| log(spatial red deer density)²    | 0.4409   | 0.2038| 2.16  | 0.031  | 2.3  |

**Excluding**

- All variables
- All variables and random effects
- Zero-inflation

Parameter estimates from analysis of abundance of questing ticks in spring and autumn with a zero-inflated negative binomial model (AIC = 10,020.8) and ‘transect’ as random terms for the West region, Møre and Romsdal county, Norway. Sample size is 11,216 nymphal ticks from 37 transects for 2 seasons each of 3 years. Each transect consists of 12 survey plots, and observation unit is survey plot for a given transect, season and year (in total n = 2,614). Baseline values are year 2011 and season Fall. ‘Distance to coast’ was entered as the mean for a local management unit. ‘Slope’, ‘elevation’ and ‘distance to coast’ are scaled to mean zero and variance one. The ΔAIC refers to the effect of removing the variable in the given row from the given model and at bottom excluding terms.
Table 4 | Analysis of tick abundance in Sogn og Fjordane County.

| Parameter                          | Estimate | S.E. | Z      | P       | ΔAIC |
|-----------------------------------|----------|------|--------|---------|------|
| Intercept                         | 2.6225   | 0.2912 | 9.005  | <0.001  |      |
| Season (spring versus fall)       | 0.3099   | 0.0567 | 5.470  | <0.001  | 27.3 |
| Year (2010 versus 2009)           | −0.0456  | 0.1028 | −0.444 | 0.657   | 173.5|
| Year (2011 versus 2009)           | −0.5834  | 0.0855 | −6.827 | <0.001  |      |
| Year (2012 versus 2009)           | −0.3151  | 0.1060 | −2.973 | 0.003   |      |
| Year (2013 versus 2009)           | −0.9644  | 0.1202 | −8.024 | <0.001  |      |
| Year (2014 versus 2009)           | −1.1810  | 0.1067 | 0.270  | <0.001  |      |
| Elevation                         | 0.0109   | 0.0784 | 0.139  | 0.889   | 27.5 |
| (Elevation)^2                     | −0.3281  | 0.0590 | −5.565 | 0.000   |      |
| bs(distance to coast)^1           | −8.3404  | 1.2578 | −6.631 | 0.000   | 94.7 |
| bs(distance to coast)^2           | −1.7015  | 1.5149 | −1.123 | 0.261   |      |
| bs(distance to coast)^3           | −5.2523  | 0.8259 | −6.360 | 0.000   |      |
| Slope                             | 0.0402   | 0.0375 | 10.672 | <0.001  | 118.6|
| log(red deer density)             | −0.0328  | 0.0818 | −0.401 | 0.689   | 13.7 |
| log(red deer density)^2           | 0.1894   | 0.0448 | 4.223  | <0.001  | 15.7 |

Excluding

- All variables: 536.7
- All variables and random effects: 2573.0

Replacing

- Year category replaced by year numeric: 33.2
- log(red deer density) (lag 1) replaced by log(spatial deer density): 10.9

Adding

- Zero-inflation: 1.6

Parameter estimates from analysis of abundance of questing ticks with a negative binomial model (AIC = 13,317.1) with ‘transect’ as random terms for the West region, Sogn & Fjordane county, Norway. Sample size is 12,082 nymphal ticks from 34 transects for 2 seasons each of 6 years. Each transect consists of 12 survey plots, and observation unit is survey plot for a given transect, season and year (in total n = 4,419). Continuous variables are scaled to mean zero and variance one. Baseline values are year ‘2009’ and season ‘fall’. ‘Red deer density’: red deer density for a municipality. ‘Distance to coast’ was modelled as third-order polynomial using a cubic spline function (bs). The ΔAIC refers to the effect of removing the variable in the given row, and at bottom if excluding, replacing and adding variables from the best model.

Table 5 | Analysis of tick load on red deer.

| Parameter                          | Estimate | S.E. | Z      | P       | ΔAIC |
|-----------------------------------|----------|------|--------|---------|------|
| Intercept                         | 12.3947  | 2.0524 | 6.039  | <0.001  |      |
| Red deer density                  | 1.1918   | 0.3367 | 3.539  | <0.001  | 9.9  |
| Julian date                       | −0.0300  | 0.0055 | −5.460 | <0.001  | 21.4 |
| Carcass mass                      | −0.0396  | 0.0124 | −3.195 | 0.001   | 4.7  |
| Elevational difference between summer and winter range | −0.0032 | 0.0010 | −3.300 | <0.001  | 7.2  |

Excluding all variables (intercept only): 35.4

Parameter estimates and test statistics for the analysis of tick load on ears of red deer (n = 49) in the West region, Norway. Model AIC = 204.1. The ΔAIC refers to the effect of removing a given variable from the model and at bottom excluding all variables.

densities, still the effect of deer density is a weak predictor of LD incidence.

Unravelling the relationship between temporal trajectories of deer populations and the incidence of LD is difficult for several reasons. Time delays may be caused by both the tick life cycle, strong historical momentum in deer population dynamics driven by age structure, and by short-term lags in the quota system. The strong negative effect on tick abundances by presence of other medium-sized mammals when the deer density increased abundance of rodents over time rather than to deer density. Evidence for increasing rodent abundances was based on statistics of declining red fox (Vulpes vulpes) numbers, a small rodent predator, due to expansion of coyote (Canis latrans) populations that predate on red fox, that is, a trophic cascading effect. This increase in rodent populations contrasts with the trend in Europe, where climate change has caused a continental...
The incidence of LD in North America has been important for the development of the biodiversity buffering disease paradigm \(^9,^{24}\) (H\(_2\)). Detailed ecological studies have found correlations between LD risk and both host community composition \(^9,^{23,36}\) and host diversity \(^9,^{23,36}\). Although this paradigm is widely supported across disease systems \(^9\), it is not universal \(^18\). In our study we found no effect of mammal species richness on LD incidence, as predicted by H\(_2\). Although our analysis encompasses a wide range of latitudes, the mammal communities only differed by a small range of latitudes, the mammal communities only differed by a few species (10–17) within LD areas, and this structure was apparently not sufficient to cause any marked dilution effect.

Table 6 | Analysis of pathogen prevalence.

| Parameter                              | Estimate | S.E.  | Z     | P     |
|----------------------------------------|----------|-------|-------|-------|
| S&F—May—using year                     |          |       |       |       |
| Intercept                              | -1.6877  | 0.5237| -3.223| 0.001 |
| Year (2010 versus 2009)                | 1.0016   | 0.4210| 2.379 | 0.017 |
| Year (2011 versus 2009)                | 0.9045   | 0.4074| 2.220 | 0.026 |
| Year (2012 versus 2009)                | 0.6993   | 0.4296| 1.628 | 0.104 |
| Year (2013 versus 2009)                | -1.2884  | 0.6415| -2.009| 0.045 |
| Year (2014 versus 2009)                | 0.7980   | 0.4289| 1.860 | 0.063 |
| Red deer density (lag 1)               | -0.6147  | 0.1931| -3.182| 0.001 |
| S&F—May—using Rodent abundance index   |          |       |       |       |
| Intercept                              | -1.3702  | 0.3149| -4.352| 0.000 |
| Rodent abundance index                 | 2.4879   | 1.0851| 2.293 | 0.022 |
| Red deer density (lag 1)               | -0.4456  | 0.1779| -2.504| 0.012 |
| S&F—Aug—using year                     |          |       |       |       |
| Intercept                              | -2.7669  | 0.5531| -5.003| 0.000 |
| Year (2011 versus 2009)                | -0.1564  | 0.2660| -0.588| 0.557 |
| Year (2012 versus 2009)                | 0.5680   | 0.2984| -1.903| 0.057 |
| Year (2013 versus 2009)                | -0.1203  | 0.2762| -0.435| 0.663 |
| Year (2014 versus 2009)                | 0.1582   | 0.2890| 0.547 | 0.584 |
| Red deer density (lag 1)               | 0.4129   | 0.2734| 1.510 | 0.131 |
| S&F—Aug—using Rodent abundance index   |          |       |       |       |
| Intercept                              | -2.9885  | 0.4695| -6.365| 0.000 |
| Rodent abundance index                 | -1.2321  | 1.5119| -0.815| 0.415 |
| Red deer density (lag 1)               | 0.4711   | 0.2559| 1.841 | 0.066 |
| M&R                                   |          |       |       |       |
| Intercept                              | -1.8763  | 0.2546| -7.371| <0.001|
| Year (2013 versus 2011)                | 0.5620   | 0.1789| 3.141 | 0.002 |
| Red deer density (lag 1)               | -0.5077  | 0.2122| -2.392| 0.017 |

Parameter estimates and test statistics for the analysis of prevalence of *Borrelia burgdorferi* sensu latu in nymphal Ixodes ricinus ticks in two counties in the West region, Norway. Analyses are separate for county Sogn and Fjordane (S&F) in May and August (due to lacking data in 2010 for August) and for county Møre and Romsdal (M&R). In M&R, there was no effect of ‘season’ (AIC = 1.60) or interaction ‘season × year’ (AIC = 2.0) if added to the model. For S&F, due to longer time series, we were able to run models using either year or the rodent abundance index. Random terms were ‘municipality’ (n = 9) for S&F and ‘local management unit’ (n = 21) for M&R.
A dilution effect would require not only that deer feed larvae, which is the case for both roe deer (Capreolus capreolus) and red deer (Cervus elaphus), but also that numbers are quantitatively high enough compared with the number of larvae fed by rodents and birds in a given area. We found that the relationship between prevalence of B. burgdorferi sensu lato in ticks and deer population density varied seasonally at the same location. Our large-scale empirical results showed that a certain pathogen dilution caused by deer density was evident in the main questing period. Furthermore, there was a lack of dilution effect in the fall, which suggests that ticks questing in fall did not come from the same cohort of larvae. Other studies have shown that ticks with nymphs infected with Borrelia have higher energy levels, higher survival and a higher level of questing activity. Over the summer, the changes in pathogen prevalence might be caused by a combination of differences in questing behaviour of infected ticks and by a lower pathogen prevalence might be caused by a combination of changes in the host and the environment. The continuing emergence of LD and other vector-borne diseases in complex systems, drivers of emergence may differ alone are not sufficient to explain the emergence of LD, as LD has emerged also in areas with a stable or declining population density of deer. Our study highlights that for vector-borne diseases in complex systems, drivers of emergence may differ across ecosystems and depend on the range of deer densities observed. The emerging evidence that for vector-borne diseases in the Northern Hemisphere will require detailed epidemiological knowledge for successful forecasting at their northern limits.

Methods

Study areas—regional descriptions. The LD incidence data come from across the whole of Norway. We grouped into four regions based on biogeography: East (Ostfold, Akershus, Oslo, Hedmark, Oppland and Buskerud Counties); South (Vestfold, Telemark, Aust-Agder and Vest-Agder Counties); West (Rogaland, Hordaland, Sogn and Fjordane, and More and Romsdal Counties); and North (Sør-Trøndelag, Nord-Trøndelag, Nordland, Troms and Finnmark Counties). These four regions span contrasting ecosystems. The West region is separated from East by a mountain range, which is a major climatic division with resulting differences in vegetation. Further, topography differs markedly between West and East. The West region has substantial differences from East/South in terms of climate, vegetation composition, intermediate, closer to the East than to West in terms of mammal community and forest composition. The geology in the West region is dominated by Precambrian gneiss, while region South and East is dominated by basement but with region South having some areas with sedimentary rocks in the inner parts of the Oslo fjord. Vegetation in the South includes areas with nemoral forest along the southwest coast, that is, deciduous forest trees such as oak (Quercus spp.) requiring warmer habitat, while the forest is in the boreo-nemoral and boreal zones further inland in the South region and in the East region. Also, region South has a warmer, more humid climate and a much higher LD incidence than East (typically being a dry inland climate). Region North has a colder climate and a very low LD incidence.

Study areas—field sites. In addition to the analysis of LD incidence across the whole of Norway, we conducted detailed field studies, which included a deer exclusion experiment, questing tick surveys and associated B. burgdorferi s.l. prevalence, and tick burdens on red deer ear samples. The purpose of these studies was to determine the mechanisms for how deer can affect LD hazard. The detailed field studies were conducted in the Sogn and Fjordane County and in the More and Romsdal/Sør-Trøndelag County located in the West region of Norway (Supplementary Fig. 2). The climate in these regions is oceanic (www.uklima.no). The precipitation and temperature generally declines from the coast to the inland and from the lowland to higher altitudes. The snow depth and duration of snow cover increases along the same gradients. The topography is highly variable rising from the fjords and valleys to mountain peaks reaching more than 1,500 m above sea level. The study areas are forested and situated in the south to middle boreal zone. Domestic sheep (Ovis aries) are abundant in some areas, but mainly graze in alpine habitats above the main tick population.

Experimental design—deer exclusions. As part of a vegetation–deer study, 10 exclusions with 10 controls each measuring 20 m × 20 m (that is, 0.04 hectares) were established in 2008. These exclusions and controls were located in mature mixed pine-deciduous forests with a closed canopy cover in Tingvoll municipality, Møre and Romsdal County, Norway. Controls and exclusions were separated by 20–60 m to avoid fence-edge effects while minimizing vegetation differences. The placement of pair-wise control and manipulation was randomized at each site. The fence was a woven wire mesh (2-m tall) nailed to stakes that had been sunk into the ground with a top wire at ~2.2 m. With a net width of 15 cm, rodents and other small mammals were unrestricted to enter and exit the exclosures. The fences may behaviourally exclude medium-sized mammals to a certain extent, even though the mesh size was large. The flagging for ticks was performed in the middle of the exclusions and controls, in a plot measuring 2 m × 10 m. The flagging of all sites was performed within a short period of time during the main questing period in the spring (12–17 May 2011). The flagging of each treatment pair was typically initiated within 30–45 min of the previous pair to ensure as similar conditions as possible for each replicate.

Tick load on deer. The tick load data derived from 49 adult red deer (≥2.5 years; 24 males, 25 females) marked with GPS-collars (Telenvilt/Followit, Stockholm, Sweden; Vectronic, Berlin, Germany) during the years 2008–2011 (ref. 42). Individual deer data are derived from one ear from each animal sampled at time of harvest. All tick instar stages were later counted. The analysis was only done on nymphs as they dominated the samples.

Flagging of questing ticks in the landscape. We used the cloth lure (or flagging method) to gather data on the abundance of questing adult and nymphal ticks. The sampling was performed in the spring (May/early June) and fall (August/early September) along 34 transects in Sogn og Fjordane (n = 12,082 nymphs, 2009–2014) and 37 transects in Møre and Romsdal/Sør-Trøndelag (n = 11,216 nymphs, 2011–2013, two missing values for 2011) in areas from coast to inland, from low to high elevation and with variable local densities of red deer. Each transect consisted of 12 survey plots with 20–50 m in between. Each survey plot was flagged with a towel (50 cm × 100 cm) over a 20-m2 plot (~10 m × 2 m). Ticks were counted and removed every 2 m after two drags on each side of the towel. In the field, ticks from each transect were pooled and killed using ethanol and then dried and stored with silica beads at −20 °C.

DNA extraction and pathogen detection. We analysed the prevalence of Borrelia spp. for individual ticks using real-time PCR (n = 5,876). Statistical analysis of variation in prevalence was done on nymphs only (Sogn and Fjordane: n = 3,324, Møre and Romsdal: n = 1,233). On the basis of our previous work, we followed a protocol where DNA extractions were optimized for ticks by modifying the incubation step of the Qiagen DNeasy 96 Blood & Tissue kit. Ticks, 2 mm zirconium oxide beads, 40 μl of proteinase K solution and 4 μl Antifoam-A (Sigma) were incubated at 56 °C overnight followed by 5 min of bead homogenization at 30 cycles per second using a Qiagen TissueLyser II (Redwood City, USA). If the ticks were not broken, they were crushed using wooden toothpicks. The remaining 160 μl of proteinase K solution was added, and the homogenized mixture was incubated for 1 h at 56 °C. Centrifugation was performed before removing seals at any step in the protocol to prevent the cross-contamination of samples. The mixture was then transferred to the DNeasy plates. We used a real-time PCR assay on a Roche LightCycler 480 Real-Time PCR instrument to detect B. burgdorferi s.l. The amplifications were performed in a total reaction volume of 10 μl, including 0.7 μl of each of the primers and 0.4 μl of probe (Forward (5′-CGAGTCTTAAAAGGGCGATTT-3′) and reverse (5′-GGCGCATATATGAAAGTATG-3′) primers and 0.175 μM of the B. burgdorferi specific probe (5′-AGATGGTTAGACCGCCAAGGCGGATTG-3′) in addition to 5 μl LightCycler Probes Mastermix (Roche, Mannheim, Germany), 32 μl μlH2O and 1 μl template DNA. The PCR programme was run as follows: pre-incubation at 95 °C for 600 s; denaturation at 95 °C for 10 s; and elongation at 60 °C for 60 s, repeated for 45 cycles. Positive controls of B. burgdorferi s.l. were used in addition to negative controls from the extraction. The results were scored as positive, negative or unknown (Supplementary Table 4).

LD incidence. Statistics on the incidence of LD (borreliosis) in humans (the official MSIS statistics of Norway) is available from 1991 to 2012 (ref. 21). LD is a notifiable disease in Norway, but only including systemic infections (stages 2 and 3) diagnosed from 1993 to 2010 (2 years) (Supplementary Table 1). We included all cases of stage 2 and 3, we excluded data from these years. We only used data for instances when municipality of the tick bite was certain.
Deer density. Data on harvest statistics are available for all municipalities of Norway from Statistics Norway. For More and Romsdal county, we also have data from 15 local management units (higher spatial resolution) with harvest density of red deer varying from 0.16 to 4.65 per km² (in 2010). As a proxy for deer population density, we used the number of harvested deer per km² of deer habitat defined by the management that provides the basis for harvest quotas. This index has been tested thoroughly against independent abundance data and has been widely used in demographic studies of deer. See Supplementary Note 1 for further information regarding this measure.

There were often strong trends in population numbers of deer due to the strong age structure effects. These effects are often referred to as population momentum, and the population from year to the next year can be high (Fig. 2). Furthermore, due to delays in the tick life cycle, we might expect strong time lags and trends over the years. Hence, disentangling the temporal variation in deer density from overall temporal trends is challenging. For questing tick abundance, Borrelia spp. prevalence and tick load, we used deer density of the previous year (lag 1) because most quested larvae will become nymphs the following year. Owing to the short time series, these data will mainly mean the spatial contrasts in deer density. For longer time spans of LD incidence data, we present a final model separating the spatial and temporal component of deer density at the scale of the municipality. The spatial deer density component is the mean deer density of the municipality over the whole period. The temporal component is the deviation of log(density) a given year from log(spatial deer density) of the municipality, that is, the log ratio of density to spatial deer density.

Rodent abundance index. In Norway, the genospecies A. s. f. scotti is dominant in questing ticks (61.6% (ref. 32); 68.4% (ref. 33)), which suggests that small mammals are the most important reservoir hosts rather than birds (R. garini 23.4% (ref. 32); 20.8% (ref. 33)). We retrieved the harvest statistics per county from Statistics Norway to estimate the number of red foxes (V. vulpes). The recruitment of foxes is known to follow rodent numbers (Supplementary Note 1). Hence, we used the number of red fox to test the rodent abundance index [log (Nt/Nt-1)] against independent data on nesting success of birds of prey known to follow the rodent cycle in one county (Supplementary Fig. 3).

Biodiversity. Following Turney et al.23, we used distribution maps of mammal species known to be tick hosts, in total 18 species (Supplementary Note 2). We used mammal species richness at the scale of municipality.

Environmental covariates. On the basis of a previous analysis of a subset of the questing data23, we calculated landscape variables such as distance to the coast, distance to the fjord, slope and elevation for all survey plots along the transects. These variables are known to affect the microclimate for ticks in these coastal areas. For the incidence of LD, we calculated several covariates on land use. On the basis of official Norwegian maps available in GIS, we calculated distance to the coast from the centre of each municipality. Likewise, we used Universal Transverse Mercator (UTM) coordinates to measure latitude. From Statistics Norway, we obtained information on several variables that are known to affect the microclimate for ticks in these coastal areas. These variables include municipality, ‘local management unit ID’ and ‘transect’ as random terms. We tested whether using zero-inflated models were necessary to achieve a good fit. Statistical analyses. We analysed data on the variation in questing tick abundances and LD incidence with negative binomial models 43,45 with either count variable or linearize relationship with the response variable. Our response was the number of patients with known systemic infection of LD and a known municipality of the tick emergence by fitting a continuous year term to the LD incidence data46. All models were fitted using logistic regression in the library LME4 (random terms as above). We assessed how well residuals fitted the prediction. To achieve a good fit, several variables were either log-transformed or square root-transformed to linearize the relationship with the response variable. Our response was the number of patients with known systemic infection of LD and a known municipality of the tick bite. We used as an offset the (log) number of people (population) in a municipality so that we in effect are modelling on the LD incidences. We checked for both spatial (using previous year’s presence/absence of LD in neighbouring municipalities) and temporal (LD incidence lagged by 1 year) autocorrelation structure. The model selection (mainly using AIC and checking for consistency with bayesian information criterion (BIC)) was performed for a wide range of environmental covariates in addition to the relevant deer population indices (a summary given in Table 2). Collinearity was assessed by calculating variance inflation factors (VIFs), only variables providing VIFs<4 were kept in the same model (Supplementary Table 2). No patterns were observed on plotting residuals of the final model of LD incidence against all explanatory variables. The final model had no strong remaining spatial pattern in residuals. There was no strong autocorrelation (97.5% of autocorrelation function (lag 1) <0.5) when evaluating the residuals from 92 municipalities with 5 or more years with positive counts of LD. See Supplementary Note 3 for details on modelling and variables included in specific models.

Data availability. LD incidence data derive from the «Norwegian Surveillance System for Communicable Diseases’ (MISS) and are available from the Norwegian Institute of Public Health (http://www.hlno.no/artikler?id=93861). Data on human demography, land use, and host populations are available from Statistics Norway (https://www.ssb.no/statistikkbanken).

References
1. Johnson, P. T. J., Preston, D. L., Hoverman, J. T. & LaFonte, B. E. Host and parasite diversity jointly control disease risk in complex communities. Proc. Natl Acad. Sci. USA 110, 16916–16921 (2013).
2. Johnson, P. T. J., de Rooide, J. C. & Fenton, A. Why infectious disease research needs community ecology. Science 349, 1259590 (2015).
3. Næsset & Gern, L. Lyme borreliosis in Europe and North America. Parasitology 129, S191–S220 (2004).
4. Ruiz-Fons, F., Fernandez-de-Mera, I. G., Acevedo, P., Gortazar, C. & de la Fuente, J. Factors driving the abundances of Ixodes ricinus ticks and the prevalence of Zoonotic I. ricinus-borne pathogens in natural foci. Appl. Environ. Microbiol. 78, 2689–2676 (2012).
5. Estrada-Peña, A., de la Fuente, J., Ostfeld, R. S. & Cabezás-Cruz, A. Interactions between ticks and transmitted pathogens evolved to minimise competition through nested and coherent networks. Sci. Rep. 5, 10361 (2015).
6. Medlock, J. M. et al. Driving forces for changes in geographical distribution of Ixodes ricinus ticks in Europe. Parasit. Vectors 6, 1 (2013).
7. Glass, G. E. et al. Environmental risk factors for Lyme disease identified with geographic information systems. Am. J. Public Health 85, 944–948 (1995).
8. Ostfeld, R. S. & Keeling, F. Biodiversity and disease risk: the case of Lyme disease. Cons. Biol. 14, 722–728 (2001).
9. LoGiudice, K., Ostfeld, R. S., Schmidt, K. A. & Keeling, F. The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. Proc. Natl Acad. Sci. USA 100, 567–571 (2003).
10. Sonenhine, D. E. & Roe, R. M. Biology of Ticks (Oxford Univ. Press, 2014).
11. Laurendon, M. K., Norman, R. A., Gilbert, L., Reid, H. W. & Hudson, P. J. Identifying disease reservoirs in complex systems: mountain hares as reservoirs of ticks and lopping-Ill virus, pathogens of red grouse. J. Anim. Ecol. 72, 177–183 (2003).
12. Ostfeld, R., Canham, C. D., Oggenfuss, K., Winchcombe, R. J. & Keeling, F. Climate, deer, rodents, and coxens as determinants of variation in Lyme-disease risk. PLoS Biol. 4, 1058–1068 (2006).
13. Levi, T., Kilpatrick, A. M., Mangel, M. & Wilmers, C. C. Deer, predators, and the emergence of Lyme disease. Proc. Natl Acad. Sci. USA 109, 10942–10947 (2012).
14. Apollonio, M., Andersen, R. & Putman, R. European Ungulates and Their Management in the 21st Century (Cambridge Univ. Press, 2010).
15. Gilbert, L., Maffey, G. L., Ramsay, S. L. & Hester, A. J. The effect of deer management on the abundance of Ixodes ricinus in Scotland. Ecol. Appl. 22, 658–667 (2012).
16. Brisson, D., Dykhuisen, D. E. & Ostfeld, R. S. Conspicuous impacts of inconspicuous hosts on the Lyme disease epidemic. Proc. R. Soc. Lond. B 275, 227–235 (2008).
17. Randolph, S. E. & Dobson, A. D. M. Pangloss revisisted: a critique of the dilution effect and the biodiversity-buffers-disease paradigm. Parasitology 139, 847–863 (2012).
18. Wood, C. L. et al. Does biodiversity protect humans against infectious disease? Ecology 95, 817–832 (2014).
19. Civitello, D. J. et al. Biodiversity inhibits parasites: broad evidence for the dilution effect. Proc. Natl Acad. Sci. USA 112, 8667–8671 (2015).
20. Gern, L. et al. European reservoir hosts of Borrelia burgdorferi sensu lato. Zentralbl. Bakteriol. 287, 196–204 (1998).
21. Jore, S. et al. Multi-source analysis reveals latitudinal and altitudinal shifts in range of Ixodes ricinus at its northern distribution limit. Parasit. Vectors 4, 8 (2011).
22. Andreasen, A. et al. Prevalence of tick borne encephalitis virus in tick nymphs in relation to climatic factors on the southern coast of Norway. Parasit. Vectors 5, 97 (2012).
23. Turney, S., Gonzalez, A. & Milliken, V. The negative relationship between mammal host diversity and Lyme disease incidence strengthens through time. Ecology 95, 3244–3250 (2014).
24. Gray, J. S., Kahl, O., Janetzki, C. & Stein, J. Studies on the ecology of Lyme disease in a deer forest in County Galway, Ireland. J. Med. Entomol. 29, 915–920 (1992).

25. Rand, P. W., Lubelczyk, C., Holman, M. S., Lacombe, E. H. & Smith, R. P. Abundance of Ixodes scapularis (Acari: Ixodidae) after the complete removal of deer from an isolated offshore island, endemic for Lyme disease. J. Med. Entomol. 41, 779–784 (2004).

26. Wilson, M. L., Ducey, A. M., Litwin, T. S., Gavin, T. A. & Spielman, A. Microgeographic distribution of immature Ixodes dammini ticks correlated with that of deer. Med. Vet. Entomol. 4, 151–159 (1990).

27. Deblinger, R. D., Wilson, M. L., Rimmer, D. W. & Spielman, A. Reduced abundance of immature Ixodes dammini (Acari: Ixodidae) following incremental removal of deer. J. Med. Entomol. 30, 144–150 (1993).

28. Coulson, T., Guinness, F., Pemberton, J. & Clutton-Brock, T. The demographic consequences of releasing a population of red deer from culling. Ecology 85, 411–422 (2004).

29. Fryxell, J. M., Packer, C., McGann, K., Solberg, E. J. & Saether, B.-E. Resource management cycles and the sustainability of harvested wildlife populations. Science 328, 903–906 (2010).

30. Cornulier, T., et al. Europe-wide dampening of population cycles in keystone herbivores. Science 340, 63–66 (2013).

31. Lindstrøm, E., et al. Disease reveals the predator: sarcoptic mange, red fox predation, and prey populations. Ecology 75, 1042–1049 (1994).

32. Kjelland, V., Stuen, S., Skarpaas, T. & Slettan, A. Prevalence and genotypes of Borrelia burgdorferi sensu lato infection in Ixodes ricinus ticks in southern Norway. Scand. J. Infect. Dis. 42, 579–585 (2010).

33. Tveten, A. K. Prevalence of Borrelia burgdorferi sensu stricto, Borrelia afzelii, Borrelia garinii, and Borrelia valaisiana in Ixodes ricinus ticks from the northwest of Norway. Scand. J. Infect. Dis. 45, 681–687 (2013).

34. Ostfeld, R. S. & Keeling, P. Straw men don’t get Lyme disease: response to Wood and Lafferty. Trends Ecol. Evol. 28, 502–503 (2013).

35. LoGiudice, K., et al. Impact of host community composition on Lyme disease risk. Ecology 89, 2841–2849 (2008).

36. Werden, L. et al. Geography, deer, and host biodiversity shape the pattern of Lyme disease emergence in the Thousand Islands Archipelago of Ontario, Canada. PLoS ONE 9, e85640 (2014).

37. Telford, S. R., Matther, T. N., Moore, S. I., Wilson, M. L. & Spielman, A. Incopetence of deer as reservoirs of the Lyme disease spirochete. Am. J. Trop. Med. Hyg. 39, 105–109 (1988).

38. Kiffner, C., Lode, C., Alings, M. & Rühe, F. Attachment site selection of ticks on roe deer, Capreolus capreolus. Exp. Appl. Acarol. 53, 79–94 (2011).

39. Mysterud, A., Hatlegjerde, J. L. & Sørensen, O. J. Attachment site selection of life stages of Ixodes ricinus ticks on a main large host in Europe, the red deer (Cervus elaphus). Parasit. Vectors 7, 510 (2014).

40. Herrmann, C. & Gern, L. Do the level of energy reserves, hydration status and Borrelia infection influence walking by Ixodes ricinus (Acari: Ixodidae) ticks? Parasitology 139, 330–337 (2012).

41. Abrahamson, J. et al. Naturgeografisk region-inndeling av Norden. Nordiske Utredn. Ser. B 34, 1–135 (1977).

42. Mysterud, A., Qviller, L., Meisingset, E. L. & Viljugrein, H. Parasite load and seasonal migration in red deer. Oecologia 180, 401–407 (2016).

43. Qviller, L. et al. Landscape level variation in tick abundance relative to seasonal migration pattern of red deer. Plos One 8, e71299 (2013).

44. Mysterud, A., Easterday, W. R., Qviller, L., Viljugrein, H. & Ytrehus, B. Spatial and seasonal variation in prevalence of Anaplasma phagocytophilum and Borrelia burgdorferi in Ixodes ricinus ticks in Norway. Parasit. Vectors 6, 187 (2013).

45. James, M. C. et al. Environmental determinants of Ixodes ricinus ticks and the incidence of Borrelia burgdorferi sensu lato, the agent of Lyme borreliosis, in Scotland. Parasitology 140, 237–246 (2013).

46. Funk, S., Bogich, T. L., Jones, K. E., Kilpatrick, A. M. & Daszak, P. Quantifying trends in disease impact to produce a consistent and reproducible definition of an emerging infectious disease. PLoS ONE 8, e69951 (2013).

47. James, G., Witten, D., Hastie, T. & Tibshirani, R. An Introduction to Statistical Learning with Applications in R (Springer, 2013).

Acknowledgements

This study was funded by the Research Council of Norway and the Norwegian Environmental Agency (TickDeer project; Pr. No. 203786 and #EcoTick project; Pr. No. 254694). We are grateful to Martin Bless, Kim Magnus Børrum, Nina Risnes-Olsen and Lars Qviller for their help with collecting questing ticks in the field, to Leif Egil Loe and Michael Angeloff for their help with GIS, to Rolf Anker Ims, Kjell Ibsen, Lars Løfaldli, Reidar Mehl, Jan Erik Roer and Trond Amund Steenset for access to or advice regarding data, host competence or species distribution, and to Lene Martinsen for linguistic editing.

Author contributions

A.M. and H.V. initiated the study; A.M. and E.L.M. organized the fieldwork; W.R.E. implemented the rtPCR; W.R.E., V.M.S. and A.B.A. performed the lab work; H.V. and A.M. and E.L.M. approved the final manuscript. All authors edited and approved the final manuscript.

Additional information

Supplementary Information accompanies this paper at http://www.nature.com/naturecommunications

Competing financial interests: The authors declare no competing financial interests.

Reprints and permission information is available online at http://npg.nature.com/reprintsandpermissions/

How to cite this article: Mysterud, A. et al. Contrasting emergence of Lyme disease across ecosystems. Nat. Commun. 7:11882 doi: 10.1038/ncomms11882 (2016).

This work is licensed under a Creative Commons Attribution 4.0 International License. The images or other third party material in this article are included in the article’s Creative Commons license, unless indicated otherwise in the credit line; if the material is not included under the Creative Commons license, users will need to obtain permission from the license holder to reproduce the material. To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/