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Estimated Effects of Projected Climate Change on the Basic Reproductive Number of the Lyme Disease Vector *Ixodes scapularis*

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Running Title: Climate change and R_0 of arthropod vectors

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Abstract

Background: The extent to which climate change may affect human health by increasing risk from vector-borne diseases has been under considerable debate.

Objectives: We quantified potential effects of future climate change on the basic reproduction number (R_0) of the tick vector of Lyme disease *Ixodes scapularis* and explored their importance for Lyme disease risk, and for vector-borne diseases in general.

Methods: Observed temperature data for North America and projected temperature using regional climate models drove an *I. scapularis* population model to hindcast recent, and project future, effects of climate warming on R_0 . Modelled R_0 increases were compared with R_0 ranges for pathogens and parasites associated with variations in key ecological and epidemiological factors, obtained by literature review, to assess their epidemiological importance.

Results: R_0 for *I. scapularis* in North America increased from 1971 to 2010 in spatio-temporal patterns consistent with observations. Increased temperatures due to projected climate change increased R_0 by factors (2-5 times in Canada and 1.5-2 times in the US) comparable to observed ranges of R_0 for pathogens and parasites due to variations in strains, geographic locations, epidemics, host and vector densities, and control.

Conclusions: Climate warming may have co-driven Lyme disease emergence in northeastern North America, and in the future may drive substantial disease spread into new geographic regions, and increase tick-borne disease risk where climate is currently suitable. This study highlights the potential for climate change to have profound effects on vectors and vector-borne diseases, and the need to refocus efforts to understand these effects.

Introduction

Considerable attention has been devoted to the possibility that climate change will exacerbate the burden of mosquito-borne diseases such as malaria and dengue, with important impacts on public health (Githeko et al. 2000). Early assessments of effects of climate change on malaria and dengue used simplistic models to assess possible effects of climate change on their basic reproductive number (R_0 , the universally recognised metric of the capacity of a parasite or pathogen to reproduce given particular environmental conditions) (Martens et al. 1995; Patz et al. 1998). However these were criticised for giving weight to future increases in R_0 whether or not such increases resulted in R_0 rising above the critical threshold of >1 for disease persistence (Rogers and Randolph 2000), and for being over-simplistic by only accounting for climate effects rather than the full range of non-climatic factors that impact the occurrence of these diseases (Reiter 2001; Rogers and Randolph 2000). Any impact of climate on R_0 of malaria and dengue is limited by effects of variations in human host density, mosquito control, infection prevention and treatment in humans, and human management of the environment (agriculture, forest management, logging etc.) that affect ecology and epidemiology of the vectors, pathogens and diseases (Githeko et al. 2012). Consequently, the strength of evidence for recent climate warming effects on malaria risk has been questioned and much debated (Reiter et al. 2004; Tanser et al. 2003).

Many vector-borne diseases of public health significance (e.g. Lyme disease, West Nile virus) are, however, maintained in transmission cycles involving wild animal hosts. These cycles are independent of human cases, and the spatio-temporal risk of human disease is less dependent on direct effects of human activities than is the risk from malaria and dengue. Nevertheless, despite some assessments (Gubler et al. 2001), effects of climate change on vector-borne zoonoses have

also been down-played mostly on the basis of limited evidence for recent effects of climate change (Kilpatrick and Randolph 2012). Lyme disease emerged (or likely re-emerged) in northeastern USA in the late 1970s due to expansion of tick populations generally thought to have been associated with changes in land use over some decades that resulted in re-forestation and expansion of population of the deer that are key hosts for the ticks (Wood and Lafferty 2013). Lyme disease is now emerging in Canada and some northern US States due to northwards expansion of the geographic range of the tick vector *I. scapularis* (Hamer et al. 2010; Ogden et al. 2009), which is dispersed from source populations by migratory birds and terrestrial hosts (Leighton et al. 2012).

A mechanistic simulation model of the *I. scapularis* life-cycle has identified temperature effects on *I. scapularis* population survival to assist assessment of current and future on-the-ground Lyme disease risk in Canada (Ogden et al. 2005; 2006b). Prospective field studies and retrospective analyses of surveillance data on tick and pathogen emergence in southeastern Canada validated the model findings, and identified temperature as a statistically significant determinant and possible driver of emergence of the tick in Canada (Bouchard et al. 2013ab; Leighton et al. 2012; Ogden et al. 2008; 2010). The *I. scapularis* model was modified to permit direct calculation of R_0 for *I. scapularis* via the next generation operator approach (Wu et al. 2013) which, given the universal use of R_0 and its estimation for a wide range of parasites and pathogens under many different conditions, allowed comparison of R_0 variations in this study with observed variations for other parasites and pathogens.

In this study we have estimated projected effects of climate change on R_0 of an arthropod vector using a model that has been extensively ground-truthed, and assessed the ecological and

epidemiological significance of the projected changes in R_0 by comparing them with ranges of R_0 values observed for other parasites and pathogens.

Methods

We estimated R_0 under current and future projected climate at 30 sites in Canada that formed two roughly south-north transects in Ontario and Quebec, two Canadian provinces where *I. scapularis* ticks are becoming established. These transects aimed to capture climate variability that exists in the region. For simplicity in data presentation, the sites were grouped into clusters (Southern Ontario, Huron, Upper Southern Ontario, South-Western Quebec and the Boreal region; Supplemental Material, Table S1, Figure S1) according to geographic proximity and similarity in temperature conditions (see Supplemental Material, “Variation in temperature and R_0 amongst sites”; Figures S2-S4), and mean values for clusters are presented. We also estimated R_0 for two sites in the USA where Lyme disease is endemic in the northeast and upper Midwest, respectively, Old Lyme (Connecticut) where the human Lyme disease cases were first recognised (Wood and Lafferty 2013), and Fort McCoy (Wisconsin) (Anderson et al. 1987).

Modelling R_0

The *I. scapularis* model is a deterministic model consisting of 12 ordinary differential equations as described in Wu et al. (2013), based on the mechanistic simulation model described in Ogden et al. (2005). This model captures effects of temperature on host seeking activity and rates of development from one life stage to the next (effects common to, but variable amongst, all arthropod vectors) parameterized from field and laboratory studies on *I. scapularis*. Mortality rates of non-feeding *I. scapularis* in Canada and northeastern USA are similar in summer and winter, presumably due to insulating effects of the litter layer in woodland habitats (Brunner et al. 2012; Lindsay et al. 1995). Our analyses operate on the hypothesis that effects of ambient

temperature on *I. scapularis* population survival are indirect via effects on temperature-dependent rates of development of ticks from one life stage to the next. The lower the temperature, the longer is the tick lifecycle and, due to constant daily per capita mortality, the fewer larval ticks survive to become mated adult female ticks. At a threshold temperature, mortality outstrips reproduction and the tick populations die out (or fail to become established), i.e. at this temperature threshold R_0 falls below unity (Ogden et al. 2005). At the latitudes under study here, effects of climate change on *I. scapularis* are expected to be the effect of climate warming on shortening the lifecycle, resulting in increasing R_0 (Ogden et al. 2006b). Quadratic effects of temperature on arthropod vector life history traits are common (Mordecai et al. 2013) and quadratic effects of temperature on tick activity are included in the model. High temperatures may impact tick survival causing northward contraction of the southern range of *I. scapularis* resulting in a northward shift, rather than overall expansion, of the geographic range of climatic suitability for *I. scapularis* (Brownstein et al. 2005). Here we confine our study to Canada and the main regions of Lyme disease risk in the US north of 40°N (Diuk-Wasser et al. 2012). Impacts of rainfall on off-host tick survival and on host-seeking activity are considered accounted for in the model in assuming i) tick populations only become established in woodlands where the microclimate is suitable for tick survival, and ii) most temperate woodlands types occur where rainfall is sufficient for *I. scapularis* survival, which is supported by studies in Canada (Lindsay et al. 1995). Future projections for increased precipitation across much of Canada with climate warming are already being seen (Environment Canada 2013), so rainfall changes are not expected to limit northward *I. scapularis* spread. For the purposes of the present study, the simulation model of Ogden et al. (2005) was modified to be able to calculate R_0 by the next generation operator as described in Wu et al. (2013). Apart from temperature values used to calculate tick development and host finding rates, values for host numbers (20 deer and 200

rodents) and all other parameter values were those used as starting values in (Wu et al. 2013). R_0 was estimated using mean monthly temperature data for each year and location as described in the following sections. Variations in host abundance affect the final size of the tick population, but not the temperature threshold (Ogden et al. 2005). The temperature threshold would be affected by variations in mortality rates of ticks in the environment, and slight variations in this have been observed in the field (Ogden et al. 2006a). For sensitivity analysis of R_0 to variations in model parameter values see Supplemental Material “Model sensitivity analysis”, Table S2, Figures S5 and S6.

Modelling R_0 under current climate

For observed temperatures, we used Australian National University Splines (ANUSPLIN: Hutchinson et al. 2009) of 10 km gridded daily time series data, which are obtained by thin-plate smoothing spline interpolation of daily climate station observations, while accounting for latitude, longitude and elevation. ANUSPLIN data cover 40 years (1971 to 2010) that encompass the period of Lyme disease emergence in North America, have coverage across northern North America and account for missing data by temporal and spatial interpolation. Mean daily near surface temperature was assigned to the 32 study sites, which are weather stations occurring over a wide range of orographic and forest ecosystems of Ontario and Quebec, or interpolations of ANUSPLIN data for locations in the USA. Monthly mean near surface air temperatures were used to parameterize the *I. scapularis* population model for estimating annual values of R_0 for each site, for each year, from 1971 to 2010 (see Supplemental Material, “Variation in temperature and R_0 amongst sites”). Values for annual cumulative degree days above 0°C ($DD > 0^\circ\text{C}$), contemporaneous for each estimated R_0 value, were also computed as the accumulation of daily temperature above 0°C for each year for each site. The tick model calculates R_0 for each

year using temperature data for that year, but in reality R_0 will depend on the temperature conditions over the 2-3 year lifecycle of the tick. Therefore we used the moving average of R_0 over three years (that year, the previous year, and the subsequent year) to describe R_0 for each year for each site, so for each site we obtained a time series for R_0 and $DD > 0^\circ\text{C}$ of 38 years (1972-2009) and $DD > 0^\circ\text{C}$ for 40 years (1971-2010).

Modelling R_0 using projected climate data

An ensemble of modelled temperature data available from three Regional Climate Model (RCM) and two Global Climate Model (GCM) runs were used to estimate future changes in R_0 (see Supplemental Material, “Validation of climate model output”, Table S3). Time series of daily temperature for the 30 Canadian sites were extracted from each model. Simulated temperature at a given site was defined as the mean of the closest grid values to that site which increases confidence in the physical representativeness (Gachon and Dibike 2007).

Bias-corrected output from the fourth version Canadian RCM CRCM4.2.3 (Laprise et al. 1997; Music et al. 2007) was chosen to provide temperature data for the *I. scapularis* model because it relatively accurately and conservatively hindcasted observed ANUSPLIN data in comparison with other climate models, and provided projected temperature at a local scale. For full details justifying the climate model selection see Supplemental Materials, “Validation of climate model output”, Figures S7 and S8. Like other RCMs, CRCM4.2.3 dynamically downscales output from a coarser resolution GCM and produces data at a horizontal resolution of approximately 50 km. CRCM4.2.3 is driven by initial and boundary conditions of the GCM CGCM3.1 T47 (McFarlane et al. 2005; Scinocca et al. 2008). Hindcasting up to 2000 used green-house gas emissions for CGCM3.1 T47 as in the CMIP 20th century experiment (Meehl et al. 2000). For future projections starting in 2001, the A2 scenario (mid-high Green-House-Gas emission scenario) of

the IPCC Special Report on Emission Scenarios (Nakicenovic and Swart 2000) was chosen because of the availability of regional climate model output using this scenario and because current actual trajectory of emissions corresponds best to this emissions scenario.

Mapping R_0 under current and future climate

Thirty year mean values of R_0 for *I. scapularis* were mapped using observed and projected values. Maps of $DD > 0^\circ\text{C}$ for North America north of the 40°N and east of the Rocky Mountains were generated using observed data for 1971 to 2000, and using $DD > 0^\circ\text{C}$ projected by bias-corrected CRCM4.2.3 output for the period 2001 to 2070. R_0 was then computed for each year from the gridded $DD > 0^\circ\text{C}$ data using the formula $R_0 = 1.072 e^{-6} DD > 0^\circ\text{C}^2 - 4.658 e^{-3} DD > 0^\circ\text{C} + 5.556$ (the threshold for $R_0 \geq 1$ was $DD > 0^\circ\text{C} \geq 2859.6^\circ\text{C}$) obtained using observed temperature data as described in Supplemental Material, “Mapping R_0 ”, Figure S9. The mean 30-year values for R_0 were then mapped for the periods 1971 to 2000, 2011 to 2040 and 2041 to 2070 (Figure 2). Regions west of the Rocky Mountains were masked because it was assumed that *I. scapularis* will not cross the Rocky Mountains, west of which Lyme disease risk will continue to depend on transmission of *B. burgdorferi* by the tick *I. pacificus* (Ogden et al. 2009).

Literature search on R_0 ranges for parasite and pathogen systems

There are to our knowledge, no equivalent estimates of how environmental changes may affect R_0 of vectors. However, to better comprehend the ecological or epidemiological importance of projected changes of R_0 of *I. scapularis*, we performed a literature review to obtain published estimates of how R_0 for parasites and pathogens varies due to changes in factors already recognised as having ecological or epidemiological importance. These include variations in geographic location, host density, strain or genotype, disease control effort and variations amongst different epidemics. Articles were searched in the National Centre for Biotechnology

Information PubMed search site (<http://www.ncbi.nlm.nih.gov/pubmed/>) using search terms i) “basic reproduction number” without specifying pathogens or parasites, and then ii) “basic reproductive number” repeated with one of the following terms: tick, mosquito, chagas, malaria, dengue, nematode, “seasonal influenza”, “pandemic influenza”, pH1N1, “avian influenza”, measles, HIV, and fluke. Abstracts were reviewed and relevant articles were reviewed in full. Relevant articles were those in which R_0 for parasites and pathogens was calculated to explicitly estimate its value under field, rather than theoretical conditions. This meant articles that employed simulation models using field data, fitting of epidemiological data (e.g. age-seroprevalence or age-infection prevalence), or other methods such as estimates from phylogenetic analysis. We did not review and use R_0 ranges obtained in model-based sensitivity analyses, variations in R_0 associated with seasonal variations in mosquito abundance in one location (which may vary from zero to very high values), estimates where control methods effectively eradicated disease resulting in almost infinite values for changes in R_0 , and model-predicted variations across whole potential geographic ranges which range from theoretical high to zero values (e.g. Estrada-Peña et al, 2013). We also did not use some very high modelled ranges of R_0 for malaria (e.g. 1 – 11,000 (Smith et al. 2007) when modelling of empirical age-infection prevalence data produced strongly contrasting single digit estimates of R_0 (Hagmann et al. 2003; Hay et al. 2005). This literature search aimed to provide an illustration of how important projected changes in R_0 of *I. scapularis* could be, compared to R_0 of other parasites and pathogens, but it was not aimed to be an exhaustive cataloguing of all literature in this field, and it was recognised that R_0 estimates are not precise and vary according to the estimation method used (Heffernan et al. 2005; Li et al. 2011).

Results

Using observed (ANUSPLIN) temperature data, R_0 for *I. scapularis* in the late 1970s when Lyme disease emerged in the northeastern USA (Wood and Lafferty 2013), was estimated at approximately 3 and 1.9 in Old Lyme and Fort McCoy respectively, at between 2 and 3 in Southern Ontario, approximately 1.5 in Huron and South-Western Quebec, but mostly below 1 in Upper Southern Ontario and the Boreal region (Figure 1). In Old Lyme R_0 increased to almost linearly to approximately 3.5 by 1999 during the first period of expansion of *I. scapularis* in northeastern US. In Fort McCoy, R_0 increased slightly but this increase was small compared to interannual variations. In Southern Ontario R_0 increased to 4 by the early 2000s during which time *I. scapularis* populations emerged at a number of locations in this region (Point Pelee National Park, Turkey Point, Rondeau Provincial Park: Figure 1 panel 1). In Huron and South-Western Quebec R_0 increased from 1.5 to 2.5 by the early 2000s, and subsequent to this (mostly from 2000 onwards) *I. scapularis* populations began to emerge in South-Western Quebec (Figure 1 panel 4). In Upper Southern Ontario R_0 increased to > 1 in the late 1990s but in the Boreal region R_0 remained below unity for the whole 1971-2010 period (Figure 1).

R_0 values for *I. scapularis* obtained in model simulations using projected climate data were similar for an ensemble of climate models, and bias-corrected output from the regional climate model CRCM4.2.3 was used as a representative of the ensemble because of its spatial resolution and predictive accuracy. R_0 for *I. scapularis* in Canada was projected to increase 1.5 to 2.3 times from the period 1971-2000 to 2001-2050, and 2.2 to 4.6 times from the period 1971-2000 to 2051-2069 depending on location (Figure 1, Table 1), and in the US, R_0 was predicted to approximately double to 7.1 and 5.2 in Old Lyme (Figure 1) and Fort McCoy, respectively by 2051-2069 (Table 1). Increases in R_0 to values > 1 predicted in regions where R_0 was below 1

during the period 1970-2000 would be expected to facilitate range expansion of *I. scapularis* northward and possibly westward (Figure 2).

The projected increases in R_0 are equivalent, for the most part, to ranges of values of R_0 estimated for other globally important parasites and pathogens associated with variations in major determinants of their ecology and epidemiology such as geographic location, pathogen genotype, different epidemics, reservoir host or vector density, and control efforts (Table 1).

Discussion

These findings suggest that increasing temperatures in northern North America that support an R_0 for *I. scapularis* of > 1.5 have been coincident with, or in advance of, but not subsequent to expanding numbers of locations where *I. scapularis* populations have become established. In Canada, where we have tracked *I. scapularis* spread, temperature has remained a statistically significant determinant of *I. scapularis* occurrence in field studies and analyses of surveillance data that accounted for alternative environmental determinants (e.g. host abundance, altitude, rainfall, habitat types, tick immigration rates) (Bouchard et al. 2013ab; Leighton et al. 2012; Ogden et al. 2008; 2010). These observations supported a key role for temperature in *I. scapularis* populations becoming established at the northern edge of the tick's range. Also, *I. scapularis* population expansion in Canada is occurring despite an overall deforestation (Natural Resources Canada 2013) rather than the reforestation thought to have driven the initial re-emergence of Lyme disease in the USA (Barbour and Fish 1993). Forest fragmentation may enhance Lyme disease risk for a variety of reasons, however *I. scapularis* are invading Canada where forest fragmentation occurred over timescales long pre-dating current *I. scapularis* invasion (Elliot 1998). Together, these findings suggest that even if recent warming in the region (5-10% increases in DD $> 0^\circ\text{C}$, Figure 1) was not associated with global warming, a future

warming climate will increase R_0 of *I. scapularis* in northern North America. Increases in R_0 may drive increased Lyme disease risk where it is already endemic (within limits determined by density-dependent regulation of the tick), and range expansion into more northern regions where it is currently absent. Furthermore, they support the hypothesis that climate warming in northeastern North America may have co-driven the emergence of Lyme disease risk, alongside other hypothesised factors such as reforestation and burgeoning deer populations (Wood and Lafferty 2013), by facilitating *I. scapularis* spread from refuges. Expansion of *I. scapularis* in the northern US has then provided the source of ticks to fuel northward expansion into Canada.

The immediate importance of future increased R_0 of *I. scapularis* in the northeast and upper Midwest of North America is that i) regions currently climatically unsuitable become suitable for *I. scapularis* establishment (i.e. R_0 changes from < 1 to >1 : Figure 2), ii) in regions currently suitable for *I. scapularis* (where $R_0 > 1$), tick invasion speed will accelerate as the likelihood of stochastic tick population fade out reduces (May et al. 2001), and tick-borne pathogen invasion speed increases due to increasing tick abundance (Ogden et al. 2007), and iii) risk from *I. scapularis*-transmitted pathogens may increase where the tick and pathogen are already established due to increased tick abundance up to a point at which this is limited by density-dependent regulation (Ogden et al. 2007). To date *I. scapularis* invasion in northern USA and Canada has been followed by invasion of the agent of Lyme disease, *Borrelia burgdorferi* sensu stricto (Hamer et al. 2010; Ogden et al. 2013), hence we assume northward *I. scapularis* range expansion is synonymous with expansion in Lyme disease risk.

The magnitude of projected increases in R_0 of *I. scapularis* in our study is of importance for the ecology and epidemiology of vector-borne diseases in general. This was illustrated by the R_0 ranges estimated for other globally important parasites and pathogens associated with variations

in known key determinants of their ecology and epidemiology (Table 1). These ranges were of a similar magnitude to projected increases in R_0 of *I. scapularis* with climate change. Tick species are likely to respond to the central tendency of increasing temperatures due to i) the long periods of inter-stadial development that take place in the surface layers of the soil where fluctuating air temperature are buffered, ii) latency in responses of development rates to temperature changes minimising effects of very short-term temperature fluctuations (Ogden et al. 2004), iii) their ability to return to soil-level refugia during extremes of heat, cold, drought or rainfall while host seeking, iv) their associations with woodland habitats within which a microclimate is buffered from extremes of temperature occurring in treeless areas (Lindsay et al. 1999a; Morecroft et al. 1998), and v) ticks have no non-parasitic immature feeding stages whose survival is susceptible to short-term changes in weather, as do dipteran vectors such as mosquitoes. Therefore the increases in R_0 projected here represent a possible magnitude of increase in mean R_0 values arthropod vectors may experience with climate change. Around this mean, dipteran population R_0 may fluctuate seasonally and annually over a much wider range due to rapid effects of rainfall and temperature on reproduction and mortality rates. Abundance and geographic distributions of many mosquito-borne diseases are currently driven primarily by control activities that superimpose on any climate effects. Large increases in vector R_0 may, however, render current vector and vector-borne disease control methods ineffective as vector multiplication outstrips control efforts (Massad, 2008; Reithinger et al. 2003; Smith et al. 2007).

R_0 increases with climate change may be limited in some circumstances. Host population densities and habitat do not seem to be currently limiting on *I. scapularis* range expansion, but they may be in the future. Mosquitoes and other dipteran vectors can be dispersed by wind (Service 1997) but ticks need hosts for their dispersal to effect range expansion. *I. scapularis* are

dispersed over long distances by migratory birds, but ticks that are not carried by migratory animals would be expected to have less capacity to invade climatically suitable environments. Ticks of public health importance such as *I. ricinus* and *I. scapularis* are mostly host and woodland habitat generalists, which facilitates range changes, while the more highly specialised the niche of a species, the less likely it will be to be dispersed and/or capable of becoming established in new locations (Morin and Chuine 2006).

Conclusions

We estimated the degree to which projected climate change may impact the ecology of arthropod vectors and, by inference, vector-borne diseases. The emergence of Lyme disease in North America may itself have been partly driven by recent climate change. Confidence in our projections is increased by observed changes in temperature and estimated R_0 for the vector that are associated with actual emergence of the vector and the vector-borne diseases it transmits. Our findings suggest that effort should be re-focussed on assessing the health risks due to vector-borne disease, particularly vector-borne zoonoses, associated with our changing climate.

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Table 1. R_0 values quantified for infectious diseases, vectors (arthropod) and vector borne diseases.

Pathogen or parasite	R_0 range estimate	Factors associated with variation	Sources
Directly transmitted infectious diseases			
Cholera in Zimbabwe	1 to 2.72 (x2.7)	Environment, socio-economic conditions, and cultural practices	Mukandavire et al. 2011
1918-19 A/H1N1 Pandemic flu	1.5 to 7.5 (x5)	Human population density	Chowell et al 2007; Massad et al. 2007; Mills et al. 2004; Vynnycky et al. 2007
1957–58 A/H2N2 Pandemic flu	1.4 - 1.7	Country	Longini et al. 2004; Nishiura 2010b
2009 A/H1N1	1.3 to 1.7 (x1.4)	Country, community, human population density	Fraser et al. 2009; Pourbohloul et al. 2009; Tuite et al. 2010; White et al. 2009; Yang et al. 2009
Low pathogenic influenza A viruses in turkey flocks	0.6 to 5.5 (x9.2)	Virus strain and farm	Comin A et al. 2011
H5N1 influenza A in poultry	1 to 3 (x3)	Different global epidemics	Zhang et al. 2012
H7N7 influenza A in poultry	1.2 to 6.5 (x5.4)	With and without control	Stegeman et al. 2004
Seasonal influenza	1.6 to 3 (x1.9)	Locations, years and viral strains	Gran et al. 2010; Truscott et al. 2012
HIV	1.1 to 3.7 (x3.4)	Country and sub-epidemic	Nishiura 2010a; Stadler et al. 2012; Xiao et al. 2013
SARS	1.2 to 8 (x6.7)	Modelling methods and human population demography	Bauch et al. 2005
Measles	1.2 to 9.5 (x7.9)	Vaccination, different schools	Mossong and Muller 2000; Plans Rubio 2012
Polio	2 to 14 (x7)	Levels of hygiene	Fine and Carneiro 1999
Canine rabies	1.05 to 2.44 (x2.3)	Location across the world	Fitzpatrick et al, 2012; Kitala et al. 2002
African Swine Fever	2 to 3 (between farms: x1.5), and 8 to 11 (within-farms: x1.4)	Location in Russian federation	Gulenkin et al. 2011
Foot & Mouth disease in cattle	1.6 to 4.5 (x2.8)	With and without control	Ferguson et al. 2001

Pathogen or parasite	R_0 range estimate	Factors associated with variation	Sources
Arthropod Vectors: <i>Ixodes scapularis</i> in Canada			
Boreal region 1971-2000 versus 2001-2050	0.3 to 0.7 (x2.3)	Climate change	This study
Boreal region 1971-2000 versus 2051-2069	0.3 to 1.4 (x4.6)	Climate change	This study
Huron region 1971-2000 versus 2001-2050	1.8 to 3.0 (x1.6)	Climate change	This study
Huron 1971-2000 versus 2051-2069	1.8 to 5.3 (x2.9)	Climate change	This study
S Ontario 1971-2000 versus 2001-2050	3.0 to 4.5 (x1.5)	Climate change	This study
S Ontario 1971-2000 versus 2051-2069	3.0 to 6.7 (x2.2)	Climate change	This study
Upper S Ontario region 1971-2000 versus 2001-2050	0.9 to 1.7 (x1.9)	Climate change	This study
Upper S Ontario 1971-2000 versus 2051-2069	0.9 to 3.3 (x3.6)	Climate change	This study
SW Quebec region 1971-2000 versus 2001-2050	1.7 to 2.8 (x1.6)	Climate change	This study
SW Quebec 1971-2000 versus 2051-2069	1.7 to 4.3 (x2.5)	Climate change	This study
Old Lyme CT 1971-2000 versus 2001-2050	3.1 to 4.8 (x1.5)	Climate change	This study
Old Lyme CT 1971-2000 versus 2051-2069	3.1 to 7.1 (x2.3)	Climate change	This study
Fort McCoy WI 1971-2000 versus 2001-2050	2.3 to 3.4 (x1.4)	Climate change	This study
Fort McCoy WI 1971-2000 versus 2051-2069	2.1 to 5.2 (x2.2)	Climate change	This study
Vector borne diseases			
Dengue in Columbia	0.88 to 3.87 (x4.4)	Human and mosquito density	Padmanabha H et al. 2012
Dengue in Brazil	1.5 to 2.75 (x1.8)	With and without adult mosquito control	Pinho et al. 2010
Dengue in Brazil	1.6 to 22.9 (x14.3)	City and year	Degallier et al. 2009
Chikungunya in Italy	1.8 to 6.0 (x3.3)	Local variations in mosquito abundance	Poletti et al. 2011

Pathogen or parasite	R_0 range estimate	Factors associated with variation	Sources
Leishmaniasis (<i>L. infantum</i>) in dogs	5.9 to 11 (x1.9)	Countries	Dye et al. 1992; Quinnell et al. 1997
Blue Tongue Virus	1.8 to 11 (x6.1)	Geographic regions of the Netherlands	Santman-Berends et al. 2013
African horse sickness in zebra	10 to 23 (x2.3)	Virus strain	Lord et al. 1997
Endoparasites			
Nematodes of sheep	6 to 16 (x2.7)	Nematode species	Kao et al. 2000
Oncherciasis	5.3 to 7.7 (x1.5)	Countries	Filipe et al. 2005

Figure Legends

Figure 1. Mean values for R_0 of the tick *Ixodes scapularis* obtained in tick model simulations using observed temperature data (ANUSPLIN: 1971-2010), and projected temperature data obtained from the RCM CRCM4.2.3 (according to SRES A2 emissions scenario) for: 1. Southern Ontario; 2. Huron Ontario; 3 Upper Southern Ontario; 4. South-Western Quebec; 5. the Boreal region of central Ontario and Quebec; 6. Old Lyme Connecticut and 7. Fort McCoy, Wisconsin. The black arrows in each panel reference the first identification of Lyme disease in the US (Wood and Lafferty 2012). The green arrows indicate the year of first field detection of *I. scapularis* populations within the Canadian clusters. In panel 1 (Southern Ontario) these dates are 1976 for Long Point (Watson and Anderson 1976), 1996 for Point Pelee (Lindsay et al. 1999b), 1999 for Rondeau Park (Morshed et al. 2003) and 2001 for Turkey Point (Scott et al. 2004) in panel 4, 2007 a number of sites in South-Western Quebec (Ogden et al. 2008). The estimated numbers of Census Subdivisions (CSDs) with established *I. scapularis* populations in South Western Quebec, based on passive surveillance data (Leighton et al. 2012), is also shown as the green dashed line in panel 4. The range of R_0 values produced in simulations for 2020-2069 of CRCM4.2.3 and five other GCMs and RCMs is indicated by the error bar to the right of each panel except for US sites for which only output from CRCM4.2.3 was available. Full details of all simulations are presented in Figure S6.

Figure 2. Maps of values of R_0 estimated from ANUSPLIN observations (1971-2000: upper panel), and projected climate obtained from the CRCM4.2.3 driven by CGCM3.1 T47 and following the SRES A2 GHG emission scenario for 2011 to 2040 (middle panel) and 2041 to 2070 (bottom panel). The colour scale indicates R_0 values. Note that within the zones where R_0 of *I. scapularis* is > 1 , geographic occurrence of Lyme disease risk is also limited by other environmental variables (Diuk-Wasser et al. 2012).

Fig 1

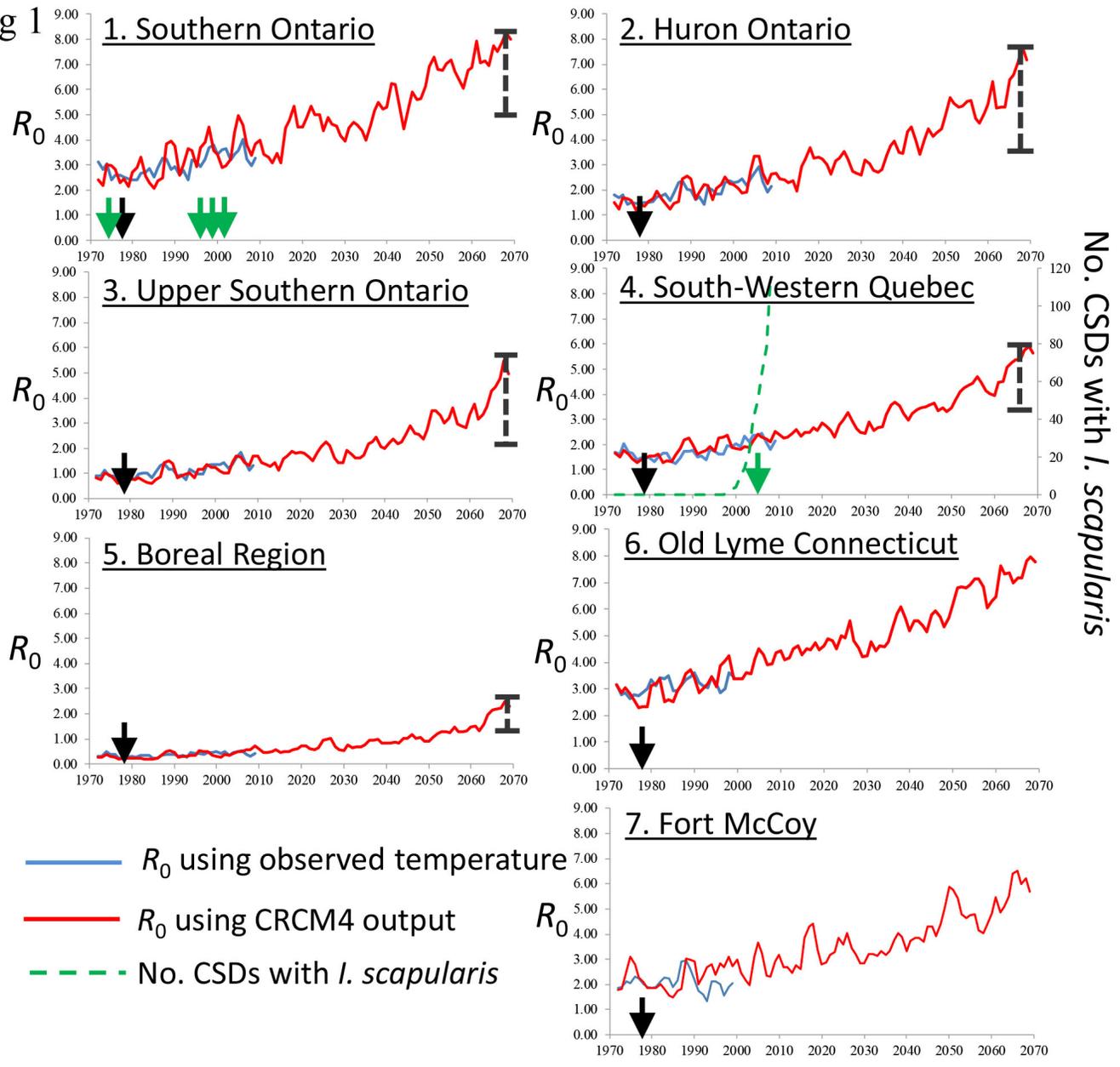


Fig 2

