How the biodiversity sciences may aid biological tools and ecological engineering to assess the impact of climatic changes

S. Morand [1, 2] & J.-F. Guégan [3]

(1)   Institut des Sciences de l'évolution (ISEM), Centre national de la recherche scientifique (CNRS), Université de Montpellier (UM2) CCO65, 34095 Montpellier, France. E-mail: morand@isem.univ-montp2.fr
(2)   Unité de Recherche 22 (UR22) Animal et Gestion Intégrée des Risques (AGIR). Centre de coopération internationale en recherche agronomique pour le développement (CIRAD), France
(3)   Génétique et Évolution des Maladies Infectieuses (GEMI), Unité mixte de recherche 2724 (UMR 2724), Institut de recherche pour le développement (IRD), Centre national de la recherche scientifique

Summary
This paper addresses how climate changes interact with other global changes caused by humans (habitat fragmentation, changes in land use, bioinvasions) to affect biodiversity. Changes in biodiversity at all levels (genetic, population and community) affect the functioning of ecosystems, in particular host–pathogen interactions, with major consequences in health ecology (emergence and re-emergence; the evolution of virulence and resistance). In this paper, the authors demonstrate that the biodiversity sciences, epidemiological theory and evolutionary ecology are indispensable in assessing the impact of climate changes, and also for modelling the evolution of host–pathogen interactions in a changing environment. The next step is to apply health ecology to the science of ecological engineering.

Keywords
Biodiversity loss – Climate change – Community vulnerability – Ecological engineering – Epidemiological theory – Evolutionary ecology – Health ecology – Host–pathogen interactions.

Introduction

Anthropogenic climate change is now well established, although the local consequences are still difficult to predict (see the Intergovernmental Panel on Climate Change [IPCC] reports at http://www.ipcc.ch/). In addition to temperatures, precipitation patterns and soil humidity will also be affected globally, regionally and locally, causing increases in average values and more frequent extreme events (storms, droughts, etc.). Such changes will, among other things, affect the geographical distributions of animals, plants, pathogens, reservoirs and vectors (33, 67, 76, 79).

Global climate change is altering the ecology of pathogens, and thus the interactions between pathogens and their hosts (18, 21, 44, 48, 49, 61, 65, 69, 70, 71). Temperature is one of the most important abiotic parameters that affects parasites and also vectors, at all their life-cycle stages. Temperature significantly affects the following:

– the release of eggs or larvae by parasites and vectors

– embryonic development and hatching rates

– the longevity of the free-living stage

– infectivity to intermediate hosts

– the development of either micro- or macroparasites in these hosts

– infectivity to definitive hosts

– time until maturation

– life span and mortality (35, 55).

Temperature also plays a key role in host feeding and behaviour, host range size and host resistance to infection.

Recently, Kutz *et al.* (25) have shown how a host–parasite system may respond to climate change. These authors presented an empirical and predictive model to demonstrate the impact of climate warming on development rates of a parasitic nematode of musk oxen in the Canadian Arctic, a region that is particularly vulnerable to climate change. Kutz and colleagues showed that warming in the Arctic may have already radically altered the transmission dynamics of this parasite, with the number of parasite generations increasing due to the lessened detrimental effect of the outside environment. The infection pressure is expected to continue to escalate for musk oxen.

A second example can be given by examining tick-borne diseases in which the tick vector is affected by climate change, such as tick-borne encephalitis (TBE) and Lyme disease. The ticks may live for several years and their survival, reproduction rate and activity are affected by seasonal climate, which indirectly influences the risk of disease. Warmer temperatures increase vector and pathogen reproduction and blood-feeding activity (50). Changes in rainfall and humidity, but also large-scale meteorological phenomena, such as the El Niño-Southern Oscillation (ENSO), may affect the number and quality of vector breeding sites (30). These influences may operate in synergy with other changes in the habitat and biodiversity. Modifications of the habitat and of the occurrence of animals (including small mammals) that carry the different pathogens result in changes in the spread of these tick-borne pathogens. Several studies have shown that, in recent decades, the tick *Ixodes ricinus*, which transmits Lyme borreliosis and TBE, has spread into higher latitudes and altitudes, and become more abundant in many places (8, 37). Climate change in Europe seems likely to facilitate the spread of Lyme borreliosis and TBE and to contribute to the extension and transmission of these diseases into new areas. Modelling suggests that these diseases will no longer persist along the southern edge of their present range but may develop new foci in northern parts of Europe (57).

These two examples and others (Fig. 1 and further articles in this publication) show that global warming has a significant impact on host–parasite interactions. However, scenarios of future interactions are not easy to produce
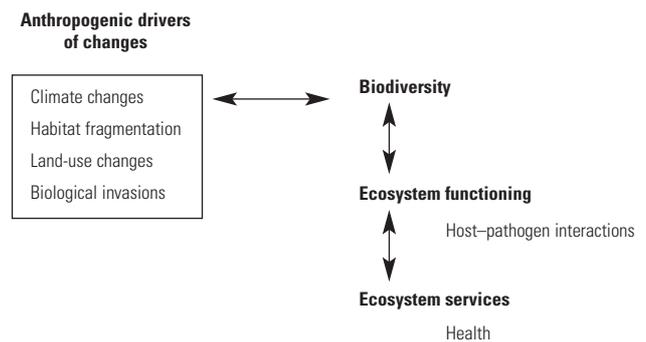

Fig. 1
How anthropogenic drivers of global change may affect biodiversity (climate change, habitat fragmentation, land-use changes and bioinvasions)
Changes in biodiversity at all levels (genetic, population and community) affect ecosystem functioning and, in particular, host–pathogen interactions, with major consequences in health ecology (emergence and re-emergence, the evolution of virulence and resistance)

without the help of modelling because of the non-linear interactions between temperature and various aspects of transmission. Moreover, climate changes can work synergistically with other changes caused by humans.

Interactions with other drivers of global change

Escalating human activities, the fragmentation and degradation of habitats, and the increasing proximity between wildlife and humans have greatly impaired the health of both humans and wildlife (9). These global changes are occurring at an unprecedented rate, which creates opportunities for parasites to negatively affect their hosts through emergence, outbreak and higher virulence (5).

Diseases and pathogens are now considered important factors in the conservation of biodiversity (9, 10, 38). Several reviews have emphasised the potential importance of parasites and pathogens as mediators of host population dynamics; under changing climatic conditions in general, and global warming in particular. Several studies have also emphasised the need to preserve vertebrate biodiversity and community composition, to significantly reduce the risk of emergence (32).

Parasite extinction is also an issue. For any host extinction, at least one parasite may go with it (56). This may not be considered negative. However, parasites should not be seen only as harmful since they have their own intrinsic value (56, 66). As ecological engineers, parasites

Rev. sci. tech. Off. int. Epiz., **27** (2)

357

contribute to maintaining high diversity (73), they may control biotic invaders and they stabilise ecosystems (3, 36, 45, 68). At the individual host level, parasites may help to equilibrate immune responses and prevent the spread of other parasites. There is, then, a balance between the need to control parasites and the benefits of their presence (51).

Several studies now recognise the need to preserve biodiversity to maintain high ecological health (1). This task must be given higher priority so that more information can be rapidly gathered, particularly through data collection and the organisation of databases on parasites and their hosts (4).

How the biodiversity sciences may help to assess the impact of climate change

Predicting bioinvasions

Biotic invaders are species that establish themselves in a new area in which they proliferate, to the detriment of local species and the environment. Such invaders are the biggest ecological outcomes of the global alterations to the distributions of earth biota, caused by human transport and commerce (77). As emphasised by Mack *et al.* (34), biotic invasions can be compared to epidemics because many important factors in disease epidemiology are also common to invasions. These factors are the minimum population size necessary for successful establishment, population growth and the fate of interacting species in the new range.

The movement of parasites, potential vectors and disease reservoirs is greatly facilitated by modern transport. A recent case of the transfer of a dangerous vector was the introduction from Asia to the United States and several European countries of a mosquito (*Aedes albopictus*) capable of transmitting dengue fever (63).

Identifying future parasite or vector invaders, as well as vulnerable communities, and taking effective measures to prevent their establishment and dispersal are great challenges for conservation biology and ecological health. The features associated with bioinvasions include: the characteristics of the parasite invaders; the characteristics of the invading hosts, vectors and reservoirs; and the vulnerability of the existing community.

Attributes of parasite invaders

Parasitic invaders are generally those parasites that are found in great local abundance or high prevalences, with direct life-cycles and low host specificity. A local abundance of a parasite species is positively correlated with their geographical distribution, with highly abundant parasites found in a larger number of host populations. The distribution of parasites with indirect life-cycles is dependent on the distribution of all hosts in the life-cycle. Any modification of host distribution will determine if parasites can persist and where they can colonise (12). Nevertheless, parasites that have indirect life-cycles, such as *Schistosoma mansoni, Fasciola hepatica* or *Angiostrongylus* spp., can still be invaders. Low host specificity is not always a crucial characteristic of parasite invaders since a more important attribute is the ability of the parasite to switch hosts.

Attributes of invading hosts, vectors and reservoirs

Many morphological and ecological characteristics favour invasion success. The introduced species may have a competitive advantage over local species because, in the new area, they are released from control by their natural enemies. Mitchell and Power (39) and Torchin *et al.* (75) found that parasite diversity is significantly reduced in organisms in their introduced range, compared to their native range, supporting the 'parasite release' hypothesis.

Invasive hosts are also at an advantage during concomitant invasion when they have evolved strong immune defences in their original range. High immune investment confers a better capacity to control the parasites with which these hosts may come into contact, or which they acquire, in the introduced range (29, 41).

Community vulnerability

Vacant niches within communities, i.e. niches which are not rich in host species or their parasites, are widely assumed to provide opportunities for the settlement and spread of biotic invaders (14). The 'vacant niches' hypothesis suggests that species-poor communities cannot offer biological resistance to invasion, particularly to pathogen or vector invasion.

Predicting extinction

Climate changes will extend the geographical ranges of many species along with their parasites, potentially overlapping with those of endangered species and driving them to extinction (12). The seasonal and spatial distributions of parasites and their hosts are often temperature dependent and the synchronicity of their population dynamics is threatened by climatic changes. Many parasites have adapted their life-cycles, reproduction and transmission to overlap with the life-cycles of their definitive and intermediate hosts (55). Any increase in temporal asynchrony between infective parasite stages and hosts has significant effects on the persistence of the

interaction. Some parasites will adapt to these new conditions, whereas others may not. It is expected that parasite extinctions will occur.

The global extinction of parasites may be more pronounced than that of free-living animals. According to Poulin and Morand (56), the 11% of threatened mammalian species implies that a total of 409 helminth species are at risk of extinction. This predicted parasite extinction may have consequences on the epidemiological environment.

Predicting distribution using environmental niche modelling

Ecological niche modelling is used in biogeography to predict the distributional range of species from existing occurrence data (2). Using appropriate algorithms in a geographical information system containing layers of environmental information (such as topography, climate and vegetation), epidemiological and spatial risk stratification can be derived from data on the location of vectors or pathogens. This approach has been used in the case of Chagas disease and for vectors of leishmaniasis and filovirus infections (52, 53, 54). Moreover, using scenarios of climate change, it is then possible to project scenarios of changes in pathogen and vector distribution.

Biodiversity loss, the diversity-disease hypothesis and the dilution effect

The predicted massive and continuing extinction of populations and species is mostly related to the consequences of land conversion for agricultural purposes (78). Biodiversity loss is usually quantified in terms of the rate of loss of species diversity. A conservative estimate of the global loss rate of eukaryote species is one extinction per hour (78), which exceeds by at least four orders of magnitude the evolution rate of novel species (28). However, biodiversity loss also concerns the reduction of genetic variation among populations, and the modification of food webs linked to the loss of interactions within communities (symbiosis, host-parasite interactions), which may greatly affect the functioning of ecosystems and the 'goods and services' they provide (as defined by the Millennium Ecosystem Assessment).

Recent evidence indicates that high species diversity may reduce exposure to parasites and pathogens (20). Initial evidence came from plant–pathogen interactions. Decreased plant species diversity is thought to increase the severity of diseases caused by specialist pathogens, i.e. the 'diversity-disease' hypothesis (14). The suggested mechanism is that both parasite intensity and spread are inversely related to host species abundance (40). Decreased diversity allows the remaining species to achieve higher abundance, which facilitates the spread of pathogens specific to these hosts.

In the case of vector-borne diseases, it has been suggested that high biodiversity may reduce the risk of disease by a mechanism known as the 'dilution effect'. The dilution effect predicts that infection rates among vectors will be lower in highly diverse host communities, where transmission to and from the competent host reservoir is diluted, due to the presence of unsuitable hosts.

The dilution effect has been demonstrated in several vector-borne diseases (19, 47, 62, 72) and, recently, in the cases of West Nile virus (15) and ectoparasites (24). LoGiudice *et al.* (32) tested the dilution effect hypothesis by showing that a high diversity of host species reduces the infection prevalence of the spirochete *Borrelia burgdorferi*, the agent of Lyme disease, in its tick vector. Higher numbers of host species dilute the effects of the most competent disease reservoir, the white-footed mouse (*Peromyscus leucopus*). Species-poor host communities tend to have many more mice and fewer other hosts, whereas species-rich communities have mice but also many other potential hosts. LoGiudice *et al.* (32) demonstrated that some host species are poor reservoirs of *B. burgdorferi* and thus reduce the prevalence of the disease by providing food for, but rarely infecting, ticks. Important dilution host species include small mammals, such as squirrels, which are characterised by high tick burdens, low reservoir competence and high population density. When such host species are added to a species-poor community, the prevalence of infection in ticks declines. Some other small mammals, such as shrews, are 'rescue hosts', which are capable of maintaining a high disease risk when mouse density is low.

How epidemiological theory may help

Models and the basic reproduction number R_0

The determinants of the reproductive success of a parasite include:

– environmental conditions, such as temperature and moisture

– the life history traits of the parasite, such as fecundity

– the density of the hosts

– the level of parasite virulence

– the response of host immunity

– the resistance of the parasites.

All these parameters should be incorporated and quantified in mathematical epidemiological models.

Rev. sci. tech. Off. int. Epiz., **27** (2)

359

Mouritsen *et al.* (45) have explored the potential consequences of climate change by building a simulation model and, more precisely, by studying the consequences of increasing temperature on a coastal host–parasite system. They set the parameters of the model with experimental and field data, and used a scenario in which the temperature rose by up to 5°C, which corresponds to one of the ranges predicted for the area where the data were obtained by the year 2075. Mouritsen and colleagues showed that an increase in the ambient temperature of 3.8°C would result in a parasite-induced collapse of the second intermediate host population. Owing to the importance of the second intermediate host, this population decline would be expected to have a significant impact on the coastal ecosystem as a whole.

Mathematical models enable the derivation of the basic reproduction number (R_0). A parasite must achieve a basic reproduction greater than the number one to establish and spread in a host population. R_0 is, by definition, the number of secondary cases produced in a population of naïve hosts after the introduction of one primary infected host. There is a critical size for the host population, below which the disease or parasite cannot establish and spread. This threshold host population size is necessary for the perpetuation of most epidemic diseases. Formulae for R_0 can be derived for macroparasites and microparasites transmitted by vectors such as ticks (tick-borne diseases) or fleas. Such formulae enable researchers to investigate theoretically how parasite epidemiology is affected by climate change or other global changes.

The use of R_0 has been illustrated with the case of arthropod-borne diseases, such as TBE, Lyme disease or plague, by Randolph and Rogers (59). For example, a shortening of the generation time at higher temperature has been reported for flea species (22) and ticks (58). The incubation time of the agent within the vector often decreases with increasing temperature. Vector biting rate is positively related to temperature, perhaps in relation to the increase of the metabolic rate. For instance, the metabolic rate of fleas is known to increase as temperature increases (16). However, we should note the highly non-linear interactions among abiotic factors such as temperature and parameters related to the population biology of both hosts and vectors. Moreover, any environmental changes that may affect the population densities of both hosts and vectors are likely to affect R_0.

Modelling the risks of an outbreak

The possible magnitude of an infectious disease outbreak is related to both the basic reproduction number R_0 and the numbers of introduced infected hosts (or the number of introduced parasites). For pathogens that are minimally transmissible (R_0 close to 0), the size of the outbreak depends largely on the number of introductions of infected hosts or parasites. For highly transmissible pathogens ($R_0 > 1$), outbreak size is determined largely by the size of the susceptible host population. For pathogens that are moderately transmissible ($R_0 \approx 1$), notable outbreaks are possible (especially if multiple introductions occur), but the scale of these outbreaks is very sensitive to small changes in R_0 (80).

Modelling risks of emergence

The epidemiological environment is affected by the way that humans are increasing their potential contacts with parasites and pathogens of wildlife (13, 80). Woolhouse and Gowtage-Sequeria (80) surveyed human pathogen diversity and produced a count of 1,407 human pathogen species. Among all pathogen species, 177 species (13%) were considered as emerging or re-emerging. Microparasites such as viruses and bacteria were the most numerous. Only 287 helminth species were recorded, with 10 emerging or re-emerging (3%).

Dobson and Foufopoulos (13) developed matrix models for quantifying R_0 for a variety of potential types of emergent pathogens that may cause outbreaks. In the models, R_0 was highly sensitive to heterogeneities created by the spatial structure of the host population and the ability of the pathogen to use multiple host species.

How evolutionary biology may help

Immuno-ecology and climate change

A new field of research among the evolutionary sciences has emerged over recent years: immuno-ecology (64). This field hypothesises that immunity has a life trait, whose value should optimally respond to environmental characteristics, including abiotic factors that can create stress (such as temperature) and biotic factors (such as pathogens). The objective, then, is to measure the investment in immunity or immunocompetence (defined as the capability of an organism to cope with pathogen infection) as the result of a trade-off with investment in other tasks. Of course, immunity has a genetic background. Few studies have investigated the role of climate variability on investment or on genetic variability of the immune genes.

In vertebrates, variability of the genes of the major histocompatibility complex (MHC) represents an important adaptation for pathogen resistance. A high genetic diversity of the MHC confers resistance to a greater number of pathogens. Selection pressures on the immune

system of the host are related to pathogen richness and pathogen virulence. Dionne *et al.* (11) tested the hypothesis that the genetic diversity of the MHC of Atlantic salmon (*Salmo salar*) increases with temperature along a latitudinal gradient in response to the selective pressure of pathogens in the wild. They showed that the allelic diversity of MHC genes increases with temperature, thus creating the latitudinal gradient, and proposed that genetic diversity at the MHC level represents a local adaptation to cope with the pathogen diversity associated with different thermal regimes. This study illuminates how selection pressure from the environment may influence host immune adaptation, and questions the future of these local adaptations in the context of climate change (rising water temperatures, distribution shifts in both hosts and their pathogens, changes in pathogen transmission).

Predicting co-adaptation and maladaptation

With global climate change come changes in the distribution and abundance of species and, by logical extension, of their parasites. The habitats of parasites, including their potential host range, are being modified. Parasites are thus experiencing range contractions and extensions, not only geographically, but in terms of the number of host species they may exploit.

The assemblages of parasites at local levels are probably the result of adaptation to regional environmental conditions. Krasnov *et al.* (23) showed that the diversification of flea assemblages on mammals is associated with climatic variables. A greater number of congeneric species per flea assemblage is observed in warm regions than in colder regions, which may be the result of intrahost speciation. When regional temperature increases, intrahost speciation may become a relatively more important mode of diversification than the acquisition of fleas through switching hosts.

There should be intense selection pressure on parasites to adapt to new conditions and new hosts or host populations. Environmental changes imply that parasites will be faced with a new epidemiological environment and new habitats, and will experience contact with new host species.

The continuously changing geographic structure of species and the worldwide redistribution of hosts and parasites will change the global dynamics of co-evolutionary interactions (74). Local adaptations of parasites to their hosts are the result of evolutionary lags between parasite genotypes and host genotypes (27, 43), which depend on both host and parasite migration (17). Local adaptation then depends on the geographical structure of interactive species, and any alteration of this structure may disrupt adaptive processes, leading eventually to maladaptation.

These changes may have consequences for different interactions, and are likely to shift co-evolutionary dynamics back towards earlier stages of non-equilibrium dynamics. However, co-evolutionary dynamics could be changed in a way that makes it very difficult to predict the consequences and outcomes of new interaction networks (74).

Global changes may also affect the evolution of parasite virulence. Models have demonstrated that the evolution of parasite life-history traits is driven by the age-specific mortality rate of the parasite (42), among other factors. The evolution of virulence may depend on many intrinsic factors, e.g. the force of the host immune response, as well as extrinsic ones, such as the survival of free-living infective stages. For example, an acceleration in parasite development in the growing season or lower mortality in certain parasite stages may lead to an increase in virulence. In such a global context, it remains extraordinarily difficult to predict the evolutionary direction of virulence, since it depends on so many factors that act synergistically and in a non-linear fashion.

Integrating tools for developing an ecology of health

Mapping the actual distribution of pathogens, vectors and reservoirs

Initiatives such as the Global Biodiversity Information Facilities aid in placing collected samples, mostly from national museums, on maps. As these samples (holotypes or voucher specimens) have been gathered from various localities in different years, it is then possible to use this information to describe past and present distributions of free-living species. However, information on past and actual distributions of parasites and pathogens is, crucially, missing.

Establishing future distributions of pathogens, vectors and reservoirs

Knowledge of the actual distribution of pathogens, vectors and reservoirs – even incomplete knowledge – allows the projection of future distributions using environmental niche modelling. By envisaging the future distributions of plants, and using scenarios of climate change from the IPCC, it is then possible to produce maps of potential future distributions. Incorporating other change scenarios (habitat fragmentation, changes in land use and biodiversity) permits the production of more accurate projections.

Establishing R_0 maps for introduction, emergence and outbreak risks

R_0 formulae can be incorporated into maps. Depending on the current body of knowledge, and existing hypotheses on the ecology of transmission, R_0 maps can then be produced. Moreover, such maps can be connected with scenarios of climate change, and other drivers of global change.

Early alert systems that include climatic variables

Over recent years, meteorological satellite data have increasingly been used to help model the spatial and seasonal dynamics of disease transmission and develop early-warning systems. These relatively low-cost and easy-to-use data sources have become familiar to public health services.

One example is the case of *Vibrio cholerae*, a bacterium associated with phytoplankton, which can be transmitted, during the infectious stage, under the same conditions as those that produce algal blooms (6). Copepod species can also be used by this bacterium as reservoirs to ensure persistence and dissemination in estuarine systems. Cholera epidemics are associated with seasonal plankton blooms. Remote-sensing satellite data and estimates of the chlorophyll contained in the phytoplankton show a correlation between the number of cases of cholera and the temperature of the ocean surface. The inter-annual variability of cholera prevalence is also dependent on episodes of ENSO and their intensity (31, 60).

Developing animal sentinel data

Animal sentinels are: 'organisms in which changes in known characteristics can be measured to assess the extent of environmental contamination and its implication for human health and to provide early warning of those implications' (46). Sentinels should be chosen from wildlife or captive species that have continuing exposure to

changing environmental pressures. Measurements should not be limited to seroprevalence but also incorporate other factors, such as stress, endocrine disruption and immunocompetence (26, 81). All of these measurements give an indication of physiological responses to the epidemiological environment. Examples include the surveillance of live birds or captive sentinels to monitor for avian viruses.

Developing ecological engineering for ecosystem management

This is the next step in applying the biodiversity sciences to the ecology of health. It remains to be seen in what ways managing landscapes and biodiversity may help to reduce the impacts and/or emergence of pathogens.

Conclusion

The authors conclude by emphasising that the sciences of biodiversity, ecology and evolution are all central to the study of zoonotic diseases under climate change. It is undeniable that climate and climate variability are key factors in pathogen transmission. However, they also work in synergy with other determinants of global change, such as changes in land use, habitat fragmentation, bioinvasion and loss of biodiversity. All these factors affect the epidemiological environment (7).

The increasing collaboration between experts in the fields of biodiversity and health comes in response to the call for more knowledge from such international agencies as the IPCC; Millennium Ecosystems Assessment; International Mechanism of Scientific Expertise on Biodiversity; Food and Agriculture Organization of the United Nations, World Organisation for Animal Health and the United Nations Environment Programme.

362

Rev. sci. tech. Off. int. Epiz., **27** (2)

Les sciences de la biodiversité au service des approches biologiques et de l'ingénierie écologique pour évaluer l'impact des changements climatiques

S. Morand & J.-F. Guégan

Résumé

Cet article traite des interactions que les changements climatiques entretiennent avec d'autres changements globaux anthropiques (fragmentation de l'habitat, changement de l'affectation des terres, invasions biologiques) et examine les conséquences de ces interactions sur la biodiversité. Toute modification de la biodiversité, à quelque niveau que ce soit (génétique, des populations ou des communautés) altère le fonctionnement des écosystèmes, en particulier les interactions hôte/agent pathogène, ce qui a des conséquences importantes sur l'écologie de la santé (émergence et réémergence de maladies ; évolution de la virulence ; évolution des résistances). Les auteurs démontrent que les sciences de la biodiversité, l'épidémiologie et l'écologie de l'évolution sont indispensables non seulement pour évaluer l'impact du changement climatique, mais aussi pour élaborer des modèles de l'évolution des interactions hôte-pathogène dans un environnement en transformation. La prochaine étape consistera à appliquer l'écologie sanitaire à la science de l'ingénierie écologique.

Mots-clés

Changement climatique – Écologie de l'évolution – Écologie de la santé – Épidémiologie – Ingénierie écologique – Interaction hôte-pathogène – Perte de la biodiversité – Vulnérabilité d'une communauté.

■

De cómo las ciencias de la diversidad biológica pueden ser de ayuda a la ingeniería ecológica y biológica a la hora de evaluar el efecto de los cambios climáticos

S. Morand & J.-F. Guégan

Resumen

Los autores examinan la forma en que los cambios climáticos interactúan con otras transformaciones de alcance mundial causadas por el hombre (fragmentación de los hábitat, cambios en los usos del suelo, invasiones biológicas) para a la postre incidir en la diversidad biológica. Los cambios que ésta sufre en todos los planos (genético, de poblaciones y de comunidades) influyen en el funcionamiento de los ecosistemas, sobre todo en las interacciones entre hospedadores y patógenos, lo que tiene consecuencias de gran calado en el terreno de la ecología sanitaria (emergencia y reemergencia,

evolución de la virulencia y las resistencias, etc.). Los autores demuestran que las ciencias de la diversidad biológica, la teoría epidemiológica y la ecología evolutiva son indispensables para evaluar los efectos de las transformaciones del clima y para elaborar modelos sobre la evolución de las relaciones entre patógeno y hospedador en un medio en constante evolución. El próximo paso consiste en aplicar la ecología sanitaria a la ciencia de la ingeniería ecológica.

Palabras clave
Cambio climático – Ecología evolutiva – Ecología sanitaria – Ingeniería ecológica – Interacción hospedador-patógeno – Pérdida de diversidad biológica – Teoría epidemiológica – Vulnerabilidad de las comunidades.

◼

References

1. Aguirre A.A., Ostfeld R.S., House C.A., Pearl M.A. & Tabor G.M. (2002). – Conservation medicine: ecological health in practice. Oxford University Press, Oxford.

2. Anderson R.P., Lew D. & Peterson A.T. (2003). – Evaluating predictive models of species' distributions: criteria for selecting optimal models. *Ecol. Modell.*, **162** (3), 211-232.

3. Arias-González J.E. & Morand S. (2006). – Trophic functioning with parasites: a new insight for ecosystem analysis. *Mar. Ecol. Progr. Series*, **320**, 43-53.

4. Brooks D.R. & Hoberg E.P. (2000). – Triage for the biosphere: the need and rationale for taxonomic inventories and phylogenetic studies of parasites. *J. helminthol. Soc. Wash.*, **67** (1), 1-25.

5. Cleaveland S., Laurenson M.K. & Taylor L.H. (2001). – Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. *Philos. Trans. roy. Soc. Lond.*, *B, biol. Sci.*, **356** (1411), 991-999.

6. Colwell R.R. (1996). – Global climate and infectious disease: the cholera paradigm. *Science*, **274** (5295), 2025-2031.

7. Daily G.C. & Ehrlich P.R. (1996). – Global change and human susceptibility to disease. *Annu. Rev. Energy Environ.*, **21**, 125-144.

8. Daniel M., Danielová V., Kriz B., Jirsa A. & Nozicka J. (2003). – Shift of the tick *Ixodes ricinus* and tick-borne encephalitis to higher altitudes in central Europe. *Eur. J. clin. Microbiol. infect. Dis.*, **22** (5), 327-328. Epub.: 8 May 2003.

9. Daszak P., Cunningham A.A. & Hyatt A.D. (2000). – Emerging infectious diseases of wildlife – threats to biodiversity and human health. *Science*, **287** (5452), 433-449. Erratum: **287** (5459), 1756.

10. Deem S.L., Karesh W.B. & Weisman W. (2001). – Putting theory into practice: wildlife health in conservation. *Conserv. Biol.*, **15** (5), 1224-1233.

11. Dionne M., Miller K.M., Dodson J.J., Caron F. & Bernatchez L. (2007). – Clinal variation in MHC diversity with temperature: evidence for the role of host–pathogen interaction on local adaptation in Atlantic salmon. *Evolution*, **61** (9), 2154-2164.

12. Dobson A.P. & Carper R. (1992). – Global warming and potential changes in host-parasite and disease-vector relationships. *In* Global warming and biological diversity (R.L. Peters & T.E. Lovejoy, eds). Yale University Press, New Haven, Connecticut, 201-217.

13. Dobson A.P. & Foufopoulos J. (2001). – Emerging infectious pathogens of wildlife. *Philos. Trans. roy. Soc. Lond., B, biol. Sci.*, **356** (1411), 1001-1012.

14. Elton C.S. (1958). – The ecology of invasions by animals and plants. Methuen, London.

15. Ezenwa V.O., Godsey M.S., King R.J. & Guptill S.C. (2006). – Avian diversity and West Nile virus: testing associations between biodiversity and infectious disease risk. *Proc. biol. Sci.*, **273** (1582), 109-117.

16. Fielden L.J., Krasnov B.R., Khokhlova I.S. & Arakelyan M.S. (2004). – Respiratory gas exchange in the desert flea *Xenopsylla ramesis* (Siphonaptera: Pulicidae): response to temperature and blood-feeding. *Comp. Biochem. Physiol., A, molec. integr. Physiol.*, **137** (3), 557-565.

17. Gandon S., Capowiez Y., Dubois Y., Michalakis Y. & Olivieri I. (1996). – Local adaptation and gene-for-gene coevolution in a metapopulation model. *Proc. biol. Sci.*, **263** (1373), 1003-1009.

18. Harvell C.D., Mitchell C.E., Ward J.R., Altizer S., Dobson A.P., Ostfeld R.S. & Samuel M.D. (2002). – Climate warming and disease risks for terrestrial and marine biota. *Science*, **296** (5576), 2158-2162.

19. Holt R.D., Dobson A.P., Begon M., Bowers R.G. & Schauber E.M. (2003). – Parasite establishment in host communities. *Ecol. Letters*, **6** (9), 837-842.

20. Keesing F., Holt R.D. & Ostfeld R.S. (2006). – Effects of species diversity on disease risk. *Ecol. Letters*, **9** (4), 485-498.

21. Kovats R.S., Campbell-Lendrum D.H., McMichael A.J., Woodward A. & Cox J.S.H. (2001). – Early effects of climate change: do they include changes in vector-borne disease? *Philos. Trans. roy. Soc. Lond., B, biol. Sci.*, **356** (1411), 1057-1068.

22. Krasnov B.R., Khokhlova I.S., Fielden L.J. & Burdelova N.V. (2001). – Development rates of two *Xenopsylla* flea species in relation to air temperature and humidity. *Med. vet. Entomol.*, **15** (3), 249-258.

23. Krasnov B.R., Shenbrot G.I., Khokhlova I.S. & Poulin R. (2005). – Diversification of ectoparasite assemblages and climate: an example with fleas parasitic on small mammals. *Global Ecol. Biogeo.*, **14** (2), 167-175.

24. Krasnov B.R., Stanko M. & Morand S. (2007). – Host community structure and infestation by ixodid ticks: repeatability, dilution effect and ecological specialization. *Oecologia*, **154** (1), 185-194. Epub.: 8 August 2007.

25. Kutz S.J., Hoberg E.P., Polley L. & Jenkins E.J. (2005). – Global warming is changing the dynamics of Arctic host-parasite systems. *Proc. biol. Sci.*, **272** (1581), 2571-2576.

26. Lafferty K.D. & Holt R.D. (2003). – How should environmental stress affect the population dynamics of disease? *Ecol. Letters*, **6** (7), 654-664.

27. Lajeunesse M.J. & Forbes M.R. (2002). – Host range and local parasite adaptation. *Proc. biol. Sci.*, **269** (1492), 703-710.

28. Lawton J.H. & May R.M. (1995). – Extinction rates. Oxford University Press, Oxford.

29. Lee K.A. & Klasing K.C. (2004). – A role for immunology in invasion biology. *Trends Ecol. Evol.*, **19** (10), 523-529.

30. Liang S.Y., Linthicum K.J. & Gaydos J.C. (2002). – Climate change and the monitoring of vector-borne disease. *JAMA*, **287** (17), 2286.

31. Lobitz B., Beck L., Huq A., Wood B., Fuchs G., Faruque A.S. & Colwell R. (2000). – Climate and infectious disease: use of remote sensing for detection of *Vibrio cholerae* by indirect measurement. *Proc. natl Acad. Sci. USA*, **97** (4), 1438-1443.

32. LoGiudice K., Ostfeld R.S., Schmidt K.A. & Keesing F. (2003). – The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proc. natl Acad. Sci. USA*, **100** (2), 567-571. Epub.: 13 January 2003.

33. Lovejoy T.E. & Hannah L. (2005). – Climate change and biodiversity. Yale University Press, New Haven, Connecticut.

34. Mack R.N., Simberloff D., Lonsdale W.M., Evans H., Clout M. & Bazzaz F.A. (2000). – Biotic invasions: causes, epidemiology, global consequences, and control. *Ecol. Applic.*, **10**, 689-710.

35. Marcogliese D.J. (2001). – Implications of climate change for parasitism of animals in the aquatic environment. *Can. J. Zool.*, **79** (8), 1331-1352.

36. Marcogliese D.J. & Cone D.K. (1997). – Food webs: a plea for parasites. *Trends Ecol. Evol.*, **12**, 320-325.

37. Materna J., Daniel M. & Danielová V. (2005). – Altitudinal distribution limit of the tick *Ixodes ricinus* shifted considerably towards higher altitudes in central Europe: results of three years monitoring in the Krkonose Mts. (Czech Republic). *Central Eur. J. public Hlth*, **13** (1), 24-28.

38. Meffe G.K. (1999). – Conservation medicine. *Conserv. Biol.*, **13** (5), 953-954.

39. Mitchell C.E. & Power A.G. (2003). – Release of invasive plants from fungal and viral pathogens. *Nature*, **421** (6923), 625-627.

40. Mitchell C.E., Tilman D. & Groth J.V. (2002). – Effects of grassland species diversity, abundance, and composition on foliar fungal disease. *Ecology*, **83**, 1713-1726.

41. Møller A.P. & Cassey P. (2004). – On the relationship between T-cell mediated immunity in bird species and the establishment success of introduced populations. *J. anim. Ecol.*, **73** (6), 1035-1042.

42. Morand S. & Poulin R. (2000). – Optimal time to patency in parasitic nematodes: host mortality matters. *Ecol. Letters*, **3**, 186-190.

43. Morand S., Manning S.D. & Woolhouse M.E.J. (1996). – Parasite-host coevolution and geographic patterns of parasite infectivity and host susceptibility. *Proc. biol. Sci.*, **263** (1366), 119-128.

44. Mouritsen K.N. & Poulin R. (2002). – Parasitism, climate oscillations and the structure of natural communities. *Oikos*, **97** (3), 462-468.

45. Mouritsen K.N., Tompkins D.M. & Poulin R. (2005). – Climate warming may cause a parasite-induced collapse in coastal amphipod populations. *Oecologia*, **146** (3), 476-483. Epub.: 27 October 2005.

46. O'Brien S.J. & Christie P. (1997). – Do CuSums have a role in routine communicable disease surveillance? *Public Hlth*, **111** (4), 255-258.

47. Ostfeld R.S. & Keesing F. (2000). – Biodiversity and disease risk: the case of Lyme disease. *Conserv. Biol.*, **14**, 722-728.

48. Patz J.A., Epstein P.R., Burke T.A. & Balbus J.M. (1996). – Global climate change and emerging infectious diseases. *JAMA*, **275** (3), 217-223.

Rev. sci. tech. Off. int. Epiz., **27** (2)

365

49. Patz J.A., Graczyk T.K., Geller N. & Vittor A.Y. (2000). – Effects of environmental change on emerging parasitic diseases. *Int. J. Parasitol.*, **30** (12-13), 1395-1405.

50. Patz J.A., Hulme M., Rosenzweig C., Mitchell T.D., Goldberg R.A., Githeko A.K., Lele S., McMichael A.J. & Le Sueur D. (2002). – Climate change: regional warming and malaria resurgence. *Nature*, **420** (6916), 627-628.

51. Pedersen A.B. & Fenton A. (2007). – Emphasizing the ecology in parasite community ecology. *Trends Ecol. Evol.*, **22** (3), 133-139. Epub.: 29 November 2006.

52. Peterson A.T., Bauer J.T. & Mills J.N. (2004). – Ecologic and geographic distribution of filovirus disease. *Emerg. infect. Dis.*, **10** (1), 40-47.

53. Peterson A.T., Pereira R.S. & Neves V.F. (2004). – Using epidemiological survey data to infer geographic distributions of leishmaniasis vector species. *Rev. Soc. bras. Med. trop.*, **37** (1), 10-14. Epub.: 19 March 2004.

54. Peterson A.T., Sánchez-Cordero V., Beard C.B. & Ramsey J.M. (2002). – Ecologic niche modeling and potential reservoirs for Chagas disease, Mexico. *Emerg. infect. Dis.*, **8** (7), 662-667.

55. Poulin R. (2006). – Global warming and temperature-mediated increases in cercarial emergence in trematode parasites. *Parasitology,* **132** (Pt 1), 143-151.

56. Poulin R. & Morand R. (2004). – Parasite biodiversity. Smithsonian Institution Press, Washington, DC.

57. Randolph S.E. (2001). – The shifting landscape of tick-borne zoonoses: tick-borne encephalitis and Lyme borreliosis in Europe. *Philos. Trans. roy. Soc. Lond., B, biol. Sci.*, **356** (1411), 1045-1056.

58. Randolph S.E. (2003). – Wildlife, climate and politics: possible causes of the recent increases in tick-borne zoonoses. *J. Parasitol.*, **89**, S69-S71.

59. Randolph S.E. & Rogers D.J. (2000). – Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change. *Proc. biol. Sci.*, **267** (1454), 1741-1744.

60. Rodo X., Pascual M., Fuchs G. & Faruque A.S. (2002). – ENSO and cholera: a nonstationary link related to climate change? *Proc. natl Acad. Sci. USA*, **99** (20), 12901-12906. Epub.: 12 September 2002.

61. Rogers D.J. & Randolph S.E. (2000). – The global spread of malaria in a future, warmer world. *Science*, **289** (5485), 1763-1766. Erratum: **289** (5499), 2284.

62. Schmidt K.A. & Ostfeld R.S. (2001). – Biodiversity and the dilution effect in disease ecology. *Ecology*, **82**, 609-619.

63. Scholte E.J. & Schaffner F. (2007). – Waiting for the tiger: establishment and spread of the *Aedes albopictus* mosquitoes in Europe. *In* Emerging pests and vector-borne diseases in Europe (W. Takken & B.G.J. Knols, eds). Wageningen Academic Publisher, Wageningen, the Netherlands, 241-260.

64. Sheldon B.C. & Verhulst S. (1996). – Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. *Trends Ecol. Evol.*, **11** (8), 317-321.

65. Shope R. (1991). – Global climate change and infectious diseases. *Environ. Hlth Perspect.*, **96**, 171-174.

66. Sprent J.F.A. (1992). – Parasites lost? *Int. J. Parasitol.*, **22** (2), 139-151.

67. Stenseth N.C., Mysterud A., Ottersen G., Hurrell J.W., Chan K.-S. & Lima M. (2002). – Ecological effects of climate fluctuations. *Science*, **297** (5585), 1292-1296.

68. Sukhdeo M.V.K. & Hernandez A.D. (2005). – Food web patterns and the parasite's perspective. *In* Parasitism and ecosystems (F. Thomas, F. Renaud & J.-F. Guégan, eds). Oxford University Press, Oxford, 54-67.

69. Sutherst R.W. (1998). – Implications of global change and climate variability for vector-borne diseases: generic approaches to impact assessments. *Int. J. Parasitol.*, **28** (6), 935-945.

70. Sutherst R.W. (2001). – The vulnerability of animal and human health to parasites under global change. *Int. J. Parasitol.*, **31** (9), 933-948.

71. Sutherst R.W. (2004). – Global change and human vulnerability to vector-borne diseases. *Clin. Microbiol. Rev.*, **17** (1), 136-173.

72. Telfer S., Brown K.J., Sekules R., Begon M., Haydon T., Birtles R. (2005). – Disruption of a host-parasite system following the introduction of an exotic host species. *Parasitol.*, **130** (Pt 6), 661-668.

73. Thomas F., Poulin R., De Meeüs T., Guégan J.-F. & Renaud F. (1999). – Parasites and ecosystem engineering: what roles could they play? *Oikos*, **84**, 167-171.

74. Thompson J.N. (2005). – The geographic mosaic of coevolution. University of Chicago Press, Chicago.

75. Torchin M.E., Lafferty K.D., Dobson A.P., McKenzie V.J. & Kuris A.M. (2003). – Introduced species and their missing parasites. *Nature*, **421** (6923), 628-630.

76. Walther G.-R., Post E., Convey P., Menzel A., Parmesan C., Beebee T.J.C., Fromentin J.-M., Hoegh-Guldberg O. & Bairlein F. (2002). – Ecological responses to recent climate change. *Nature*, **416** (6879), 389-395.

77. Williamson M. (1996). – Biological invasions. Chapman & Hall, London.

78. Wilson E.O. (1992). – The diversity of life. Harvard University Press, Cambridge, Massachusetts.

79. Wilson M.E. (2000). – Environmental change and infectious diseases. *Ecosyst. Hlth*, **6**, 7-12.

80. Woolhouse M.E.J. & Gowtage-Sequeria S. (2005). – Host range and emerging and reemerging pathogens. *Emerg. infect. Dis.*, **11** (12), 1842-1847.

81. Yamamoto J.T., Donohoe R.M., Fry R.M., Golub M.S. & Donal J.M. (1996). – Environmental estrogens: implications for reproduction in wildlife. *In* Noninfectious diseases of wildlife (A. Fairbrother, L.N. Locke & G.L. Hoff, eds). Iowa State University Press, Ames, Iowa, 31-51.