Rev. sci. tech. Off. int. Epiz., 2008, **27** (2), 443-452

Effects of climate change on animal and zoonotic helminthiases

S. Mas-Coma, M.A. Valero & M.D. Bargues

Departamento de Parasitología, Facultad de Farmacia, Universidad de Valencia. Av. Vicent Andrés Estellés s/n, 46100 Burjassot, Valencia, Spain

Summary

Current knowledge of animal and zoonotic helminthiases in which effects of climate change have been detected is reviewed. Climate variables are able to affect the prevalence, intensity and geographical distribution of helminths, directly influencing free-living larval stages and indirectly influencing mainly invertebrate, but also vertebrate, hosts. The impact of climate change appears to be more pronounced in trematodes, and is mainly shown by increased cercarial production and emergence associated with global warming. Fascioliasis, schistosomiasis (*S. japonicum*) and cercarial dermatitis caused by avian schistosomes have been the focus of study. Alveolar echinococcosis is currently the only cestode disease that climate change has been found to influence. Nematodiases, including heterakiasis, different trichostrongyliases and protostrongyliases, ancylostomiases and dirofilariases, are the helminth diseases most intensively analysed with regard to climate change. It may be concluded that helminth diseases should be listed among the infectious diseases with which special care should be taken because of climate change in the future, especially in temperate and colder northern latitudes and in areas of high altitude.

Keywords

Acanthocephaliasis – Cestodiasis – Climate change – Domestic animal – Nematodiasis– Trematodiasis – Wild animal – Zoonosis.

Introduction

Many recent studies have emphasised the causal relationship between climate change and parasitic diseases, whether emerging or re-emerging (18, 36, 87).

Among infectious diseases, helminthiases are important because of their large impact on human and animal health and their capacity to regulate the abundance of wild animal populations (1, 41, 95) and communities (73, 80), and hence to affect the functioning of ecosystems.

The different helminth groups present very different transmission patterns, ecological requirements and dispersal strategies. Their dependence on abiotic and biotic factors is related to their free-living stages, and their environment–host population interactions. Among the numerous environmental modifications that give rise to changes in helminth infections, climate variables appear to have a considerable influence (53, 86).

Interactions between climate and helminth development

In general, and according to the life-cycle pattern of each helminth species, climate variables are able to affect the prevalence, intensity and geographical distribution of helminths by directly influencing free-living larval stages as well as indirectly influencing mainly the invertebrate, but also the vertebrate, hosts (Fig. 1).

Trematodes follow a heteroxenous life cycle in which the first intermediate host is a specific mollusc. In trematodes that follow a two-host life cycle, the vertebrate definitive

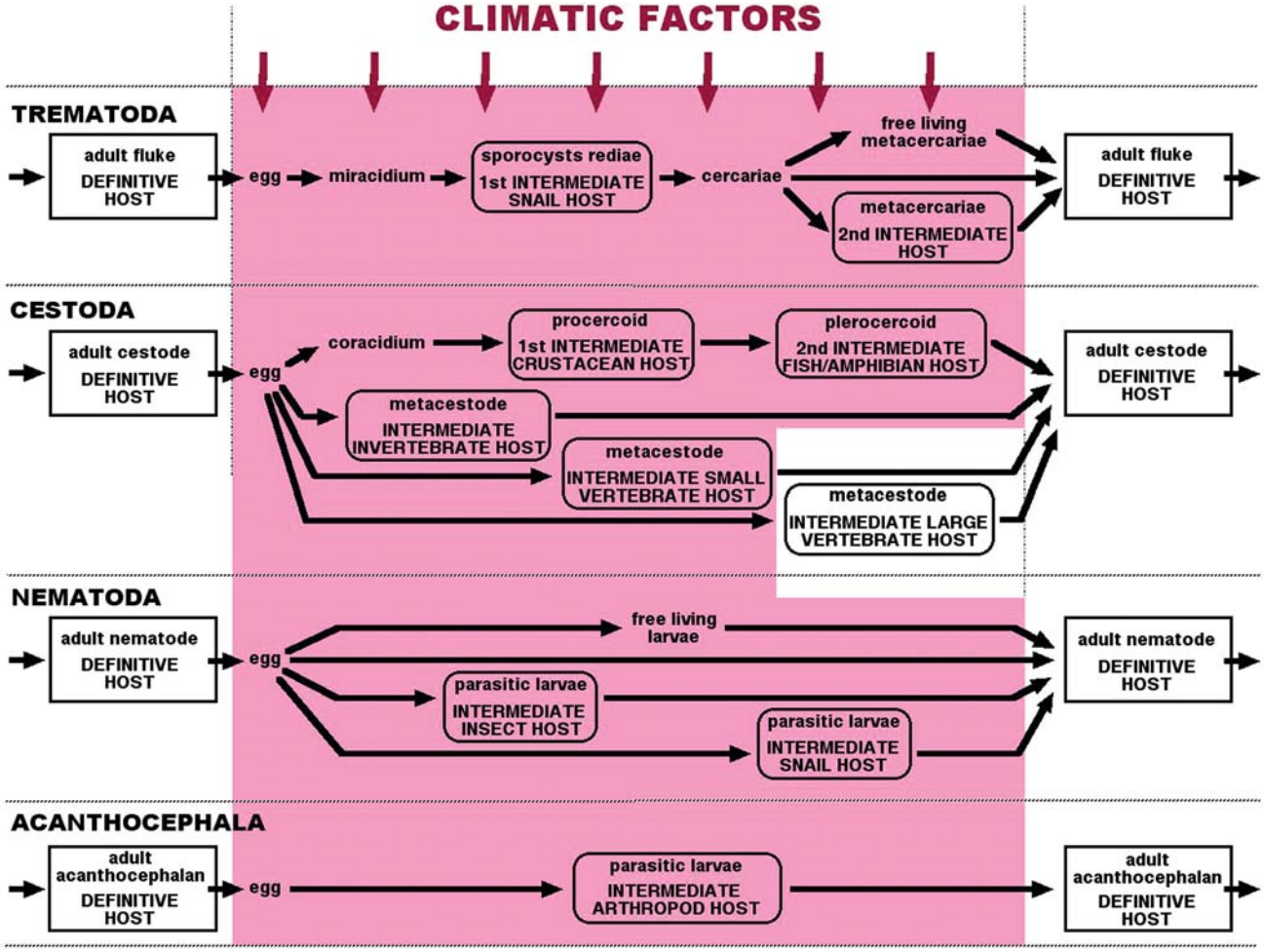

Fig. 1

Life cycles of the main helminth groups of importance to animal and human health

The figure shows the transmission phases of free-living larval stages and parasitic larval stages infecting ectothermic invertebrate hosts or endothermic small vertebrate hosts, which climatic factors influence more profoundly

host becomes infected either via the skin, by the cercarial larva stage released from a snail (in schistosomatids), or by ingestion of the metacercarial larval stage attached to any carrying substratum, e.g. freshwater plants (in fasciolids). In three-host life cycles, the vertebrate host becomes infected by ingesting the second intermediate host, usually an invertebrate (molluscs, insects, crustaceans) or an exothermic vertebrate (fish, frogs, reptiles) (23, 30). Climate conditions either directly influence free larval stages (eggs faecally shed by the vertebrate host, miracidium development inside the egg, snail-released cercariae, or non-parasitic metacercariae) or indirectly affect the parasitic stages (sporocysts, rediae, cercariae in the snail; metacercariae in invertebrates), as further described below.

Cestodes follow life cycles that are less influenced by climatic conditions (3, 46). Triheteroxenous pseudophyllideans (diphyllobothrids) that infect fish-eating carnivores include the free-living egg, the swimming coracidium, the parasitic procercoid in crustaceans and the plerocercoid in fish or amphibians. Cyclophillideans follow two-host predation life cycles in which only the egg stage is free living. Several use invertebrates as intermediate hosts that are highly dependent in terms of physiology and population dynamics on environmental conditions (mites, insects); others use endothermic vertebrates that may be affected by climate (i.e. small mammals), while others (including most taeniids) use large mammals that are scarcely affected by climatic conditions.

Nematode life cycles vary from very simple to complex (2). Trichurids (*Trichuris*) and ascarids (*Ascaris, Toxocara*) are monoxenous, with a definitive mammalian host and the egg being the only free-living stage dependent on environmental factors (pseudogeohelminths). Ancylostomatids and *Strongyloides* are also monoxenous but present active free-living larval stages that are highly dependent on abiotic factors (geohelminths). Other

nematodes have a two-host life cycle; these include vector-borne parasites transmitted by biting dipteran insects (filarids). Diheteroxenous protostrongylidae are transmitted by strongly climate-dependent snails.

Acanthocephalans are less important in public health, although they may be very pathogenic (82) and even able to regulate their definitive host populations (81). They present a free-living egg stage voided with the faeces of the definitive host, which must be eaten by an intermediate arthropod host (insect, crustacean, myriapod) that is dependent on climatic conditions (96).

Trematodiases

Changes in trematodiases expected from climate change

Global warming has been predicted to alter the geographical distributions of many trematode species (7, 77, 107). Moreover, it may also enhance the local impact of trematodes. Analyses suggest a common scenario in which a temperature increase should lead to marked increases in cercariae, by accelerating their production and triggering their emergence from snails. Any temperature-mediated increase in the extent of trematode infections may have measurable repercussions. The impact may be very important; trematode parasitism is not only a major veterinary and human health problem worldwide, but it also plays a major role in the structuring of animal communities (88).

Fascioliasis

Fascioliasis is caused by *Fasciola hepatica* and *F. gigantica*. Whereas in Europe, the Americas and Oceania only *F. hepatica* is involved, both species overlap in Africa and Asia. The definitive hosts include many herbivorous mammals, such as cattle, sheep, goats, equines, African and South American camelids and marsupials, and also omnivores such as pigs and humans. Intermediate hosts are specific freshwater lymnaeid snail species (68). Fascioliasis shows a surprising spreading power, related to the considerable capacity of the trematode to colonise new environments and adapt to new intermediate and definitive hosts, in addition to the capacity for widespread dispersal of specific lymnaeids (69).

This disease is at present emerging or re-emerging in many regions of Latin America, Africa and Asia (65), and also in Europe (74, 75, 89, 101). The major health problems occur in the Andean countries, Caribbean region, northern Africa, Western Europe, the Caspian area and Southeast Asia (65, 66). The highest prevalence and intensity of human fascioliasis appear in high altitude areas of Andean countries, where the fluke has developed strategies to favour its transmission, including a longer cercarial shedding period, greater cercarial production per snail and longer survival of infected snails (71).

Although liver fluke development is very dependent on environmental characteristics, fascioliasis has become the invertebrate-borne disease with the widest latitudinal, longitudinal and altitudinal distribution known (65, 66). Fascioliasis is unique in having endemic areas that range from below sea level (Caspian Sea) to altitudes of up to 4,000 m (Bolivia, Peru, Ecuador, Venezuela) (70).

Climatic factors are decisive, and increasing drought and floods associated with the El Niño-Southern Oscillation phenomenon (34) may give rise to outbreaks in countries such as Peru and Ecuador.

The incidence of fascioliasis has been related to air temperature, rainfall and/or potential evapotranspiration. Forecast indices taking these climatic factors into account have been successfully applied in different areas of Europe, Africa and the United States of America (USA). However, these climatic factors vary markedly with altitude and latitude. Thus, the very high altitude climatic characteristics of the Bolivian Altiplano differ considerably from those of endemic lowlands in Europe (29).

The most useful climate indices known for fascioliasis (7, 63, 83, 84, 85) indicated, surprisingly, that this disease should not be present on the Altiplano. After introducing modifications for high altitude and low latitude deduced from climate diagrams, the indices indicated that transmission on the Altiplano takes place throughout the year, in contrast to the typical seasonality of fascioliasis in northern hemisphere countries. One of the modified index values distinguished low, moderate and high risk areas, and field validation demonstrated its accuracy (29).

Collection of surface hydrology and vegetation indices and temperature data has proven to be a very useful application of remote sensing (RS) and geographical information systems (GIS) to fascioliasis. A modified forecast system was used to predict fascioliasis transmission in East Africa, using a minimum temperature threshold of 10°C for *F. hepatica* and 16°C for *F. gigantica* (61, 62, 64, 107).

These technologies have also been applied to Andean countries. Based on normalised difference vegetation index (NDVI) classes, a map differentiating different risk levels has been produced for each region of Chile (26).

Large studies were undertaken to determine whether a GIS would be useful in the Bolivian Altiplano hyperendemic zone (67). The prediction capacity of the RS map based on NDVI data (27) appeared to be higher than that derived

Fig. 2A

The Northern Bolivian Altiplano human and animal hyperendemic area at the south-eastern part of Lake Titicaca

Map showing different transmission risk zones according to normalised difference vegetation index (NDVI) values (yellow = no risk; light green = low or moderate risk; dark green = high risk)

from climatic indices (Figs 2A and 2B). The total overlap between the real and predicted ranges of fascioliasis prevalence is worth mentioning. A recent GIS for fascioliasis in Andean countries combines climatic and RS-NDVI data and its accuracy has been verified with field data from Bolivia, Peru and Ecuador (28).

Schistosomiasis

Temperature, water body type, rainfall, water velocity and altitude have a significant effect on schistosomes, their freshwater snail hosts (25) and the geographical distribution of both (79).

In China, the resurgence of the widely zoonotic *Schistosoma japonicum* in recent years has occurred despite a 50-year intensive control programme. Explanations are multifactorial, including the construction of the Three Gorges dam, the recovery of the Dongting Lake, and major flooding events. Additionally, a climate change effect was considered (109). Both the Three Gorges dam and global warming are likely to increase the snail habitat. Data from RS were used to measure the impact of a flood on the dispersal of the snail host in the Yangtze River basin (108). Predictive models of spatial distribution appear to be hampered by the existence of different *Oncomelania hupensis* snail subspecies, whose habitats vary considerably (98). Risk prediction using satellite imagery became difficult because of seasonal flooding, and soil maps are expected to solve the problem (97).

A previously determined lower temperature threshold for intramolluscan larval development of 15.3°C was used to evaluate the effects of climate change (110). A model including a 1972 to 2002 time-series for 39 eastern counties showed that temperatures had increased over the past 30 years, and predicted increases for the entire area studied, including an increase from north to south. The results suggest that temperature changes will alter the frequency and transmission dynamics of schistosomiasis japonica in China (106).

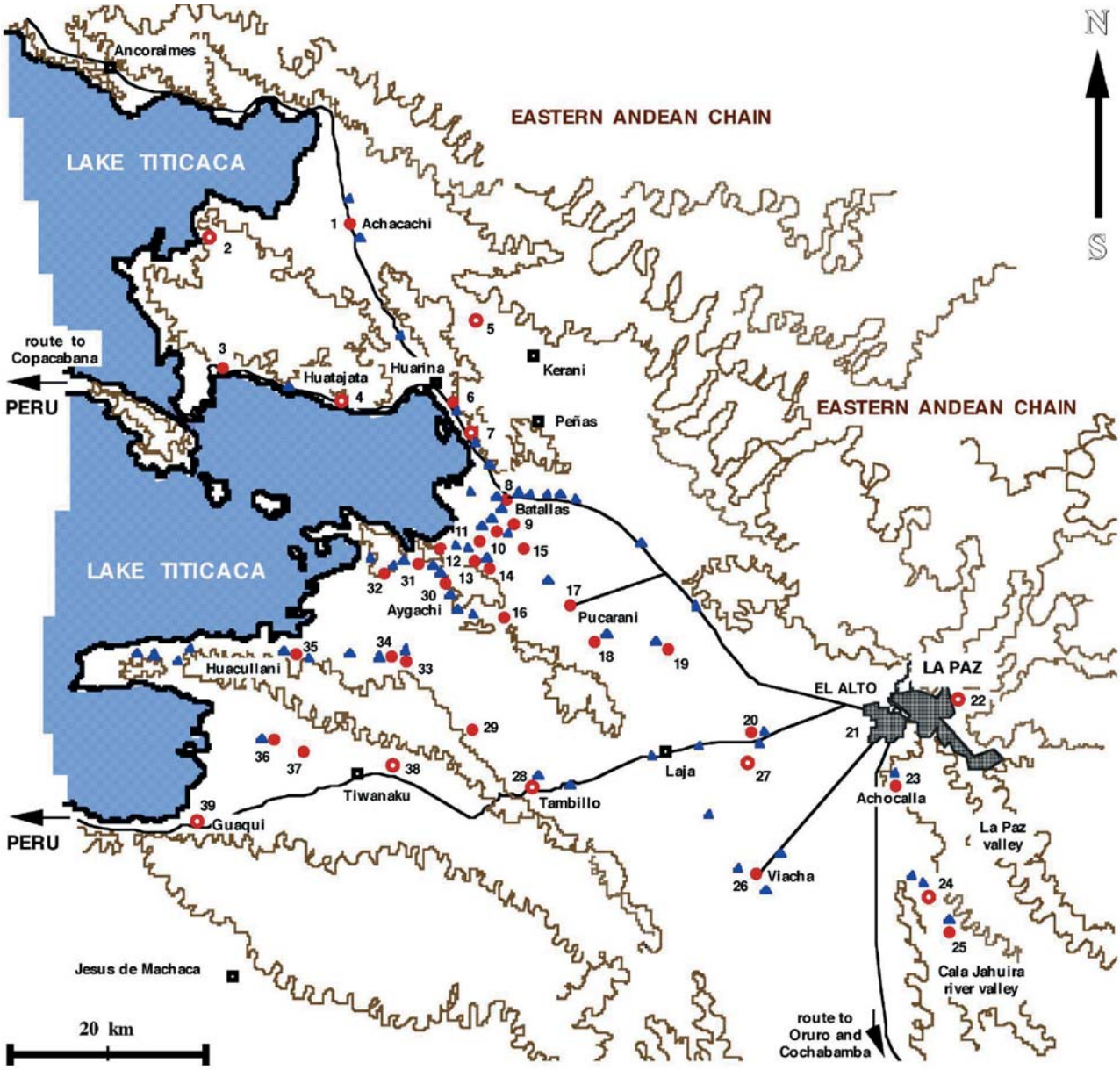

Fig. 2B

The Northern Bolivian Altiplano human and animal hyperendemic area at the south-eastern part of Lake Titicaca

Map showing the distribution of localities presenting infected people (filled red circles), localities with no human infection (empty red circles), and fresh water bodies inhabited by lymnaeid snail vectors (blue triangles)

Consecutive numbers refer to human communities surveyed. Note the relative situation of the Eastern Andean Chain, which includes very high mountains (Huayna Potosi, Chacaltaya) of up to 6,000 m altitude, and the Altiplanic plains and corridors lying at an altitude of between 3,800 m and 4,100 m

Techniques such as RS and GIS can be of value in looking at the distribution of schistosomes at both reduced and large spatial scales (8, 43). A global network (GNOSIS) dedicated to the development of computer-based models for schistosomiasis and other snail-borne diseases of medical and veterinary importance has been created (60, see www.gnosisGIS.org). An agreement has been reached on the use of compatible GIS formats, software, methods and data resources, including the definition of a 'minimum medical database' to enable seamless incorporation of results from each regional GIS project into a global model. Thus, GNOSIS will point users to a toolbox of common resources, provide assistance on routine use of GIS health maps for control programmes and provide models to predict the health impacts of water development projects and climate variation (60).

Cercarial dermatitis

Also called 'swimmer's itch', this disease is an inflammatory skin reaction caused by avian schistosome cercariae that penetrate, develop and die in the skin of humans and other mammals after repeated contact (38). These avian schistosomes show a rather narrow freshwater snail host specificity (6).

This disease occurs globally and has recently appeared as an emerging disease in several lake regions, both in Europe and in North America (16). In Europe, it is caused mainly by cercariae of *Trichobilharzia*, but also by *Bilharziella* and *Gigantobilharzia* (6, 100). Several outbreaks have been reported recently, involving large numbers of swimmers in France (13) and numerous lakes in Sweden (102). The disease is reported even from cold climate areas such as lakes in Iceland (50) and French Alpine lakes (Annecy and Le Bourget) in the Savoy district (19).

Most reports concern outbreaks associated with swimming or wading in recreational lakes (58). The disease is usually seasonal, being most common in the warmer months because both cercarial emergence and the number of people in contact with recreational water areas peak in that period (14). Temperature affects the rate at which schistosomatids develop within snails (59). Higher temperatures accelerate trematode development in snails, resulting in an earlier and more severe disease situation. Additionally, higher populations of snails may correlate with higher levels of algae, leading to higher cercarial density in water (58).

Global warming has caused the climate around lakes used by migratory birds such as anatids to become increasingly mild in recent years. These birds, or at least part of their populations, no longer need to migrate southwards, so they remain on central European lakes and become sedentary (15). This allows the avian schistosomatids to expand their transmission into months in which spread ceases in cold years. This larger seasonal window may be the origin of higher infection prevalence in snails, whose population activity is also seasonally extended. This leads to a larger number of schistosomatid generations per year, and higher prevalences in birds, resulting in a markedly higher density of cercariae able to give rise to a greater incidence of human infection.

Cestodiases

Echinococcosis

Studies on emergence/re-emergence of echinococcoses suggest that climate change may also have an impact on cestodiases (20, 44, 76). *Echinococcus multilocularis*, the causal agent of alveolar echinococcosis, has a vesicular larva adapted to small rodents (usually Arvicolidae), and foxes and coyotes as principal definitive hosts (22). This disease only occurs throughout the northern hemisphere and has received considerable attention because of its severe pathogenicity in humans.

Environmental parameters act on two targets: sufficient ground moisture increasing egg survival in the environment, and certain vegetation types providing the habitat for large densities of suitable rodent species (32, 33, 76, 92). Anthropogenic alterations of landscape and accidental introductions of the parasite or new hosts play a key role (33, 92). The influence of landscape is evident, although the impact of individual landscape parameters appears to vary among regions. Areas with large pastures and meadows show lower prevalence than areas with more fragmented landscapes dominated by crop production (92).

Additional factors are climatic variables that act on the survival of eggs in the environment, host species abundance controlled by factors other than landscape, and human behaviour. Eggs of *E. multilocularis* survive for 478 days at 95% relative humidity (RH) and 4°C, but for only 24 h at 27% RH and 25°C (103). This explains the constrained geographical distribution of this cestode species, in contrast to the worldwide distribution of *E. granulosus*, whose eggs remain viable for months and withstand temperatures of –30°C to +30°C (21, 22).

Climatic change, including changes in temperature, precipitation, rainfall, soil humidity, dryness or desiccation, may thus influence both infection and distribution through modifications of local climatic conditions (e.g. moisture) as well as landscape (i.e. grasslands, pastures).

Nematodiases

~~Heterakiasis~~

Heterakis gallinarum is a caecal pseudogeohelminth parasite of chickens and related bird species. This nematode is transmitted by eggs that become infective for a new definitive host after larval development inside the egg in the external environment. In a study of the egg development rates of this parasite, a temperature increase resulted in a linear increase in the development rate (93). However, when the parasite was subjected to a daily temperature cycle, development started significantly earlier than that expected from the model. Moreover, the development time of eggs placed in stochastic temperature fluctuations with the same thermal energy as the cycle was also significantly reduced, indicating that fluctuations in temperature, and particularly increased variation, can accelerate parasite development rates (94).

Trichostrongyliases

Trichostrongylids are soil-transmitted geohelminths that are excellent models for studies on the effects of climate change. Studies suggest that mean parasite intensities will increase with climate warming for parasites that affect host species lacking a strong acquired immune response, e.g. the rabbit stomach trichostrongylid *Graphidium strigosum* and the red grouse caecal *Trichostrongylus tenuis*, but there will be only minor effects on species that stimulate an acquired response, e.g. the rabbit small intestinal *Trichostrongylus retortaeformis* (40).

Trichostrongylids enter a period of arrested development known as hypobiosis and do not emerge until environmental conditions become favourable (72). This can result in a synchronised emergence of arrested stages at the time when hosts are collecting nutrients to breed, and consequently can have a major impact on host productivity. With warming, selection against parasites entering an arrested stage, and increased rates of parasite turnover, which will increase the basic reproductive number R_0 (the average number of female offspring produced by one adult female parasite that attains reproductive maturity, in the absence of density-dependent constraints), might be expected (40).

Trichostrongylus tenuis reduces the breeding output of the female grouse and plays an important role in generating instability in grouse populations (39, 41, 42). A model has suggested that warming could have a dramatic effect on the R_0 of the nematode and alter the dynamics of the grouse population. Warming would reduce the larval arrest and thus expand the period of the year during which the free-living larval stages would be active. This would, in turn, increase the rates of ingestion of larval stages by the grouse and consequently increase the infection rates of the grouse (17). A reduction in the period of arrest, increases in R_0 or increases in the ingestion rate of the infective stage would increase the instability of the nematode–host system, increasing both the period and the amplitude of the oscillation, and give rise to outbreaks followed by dramatic crashes in abundance of grouse (40).

In northern England, model fitting indicated either accelerated *T. tenuis* transmission, leading to a large-scale disease outbreak and a decline in all grouse populations, or reduced transmission, leading to low parasite intensity and an increase in the host population. Hence, an increase in the frequency of extreme events was shown to lead to spatially synchronised disease outbreaks (12).

In the Netherlands, the summer of 2003 was unusually warm and dry and lamb gastrointestinal nematodes deviated from normal yearly patterns. The proportion of eggs that developed into infective larvae was lower, severe *Haemonchus contortus* infection was observed in lambs grazed at the end of July on pastures contaminated during June and July, and lambs grazing in September still acquired large haemonchosis burdens. Moreover, 60% of *H. contortus* established before mid-July survived until October (24).

Ancylostomiases

Climate change may have a considerable impact on ancylostomid species of cats and dogs that are transmitted by free-living larvae. *Ancylostoma braziliense*, *A. caninum*, *A. ceylanicum* and, more sporadically, *Uncinaria stenocephala* cause cutaneous larva migrans in humans (4). In addition to tropical and subtropical regions, these helminths also inhabit temperate and colder regions, where climate change is expected to be more marked. The ubiquity of *A. caninum* in domestic dog populations is of concern because of its ability to cause not only cutaneous larva migrans but also gastroenteritis in humans (90).

Emergence of human cutaneous larval migrans was detected in Berlin, Germany, in summer 1994 when extraordinary climatic conditions led to 378 sun-hours in July, an extremely high number, the mean surface air temperature was 6°C higher than normal and the absolute air humidity reached 19 g water/m^3 air (average 10 g/m^3). The study concluded that increasing local temperatures and global warming may give rise to the emergence of cutaneous larva migrans due to the ubiquitous presence of these zoonotic hookworms (49).

Protostrongyliases

Protostrongylids have indirect life cycles, in which first-stage larvae are shed in the faeces of the mammalian definitive host, invade an intermediate gastropod host, and develop to infective third-stage larvae. This larval development within the poikilothermic mollusc is temperature dependent. Additionally, alterations in temperature, precipitation or vegetation could modify the geographical distribution, density or survival of mammalian or gastropod hosts, and larval survival, thus amplifying parasite populations and ultimately influencing host population health (55, 57).

Models able to predict larval intramolluscan development have been validated for *Umingmakstrongylus pallikuukensis* and *Parelaphostrongylus odocoilei*, which respectively infect muskoxen and Dall's sheep in different northern habitats (Arctic tundra and Subarctic alpine) (45, 54). These models are tools for determining the potential for range expansion, and predict responses to climate change (56).

In the Mackenzie Mountains of northern Canada, the results indicate that climate warming has already increased the length of the growing season of these helminths and

the amount of heat available for larval development. The extension of gastropod activity earlier and later in the season and the increased number of infective larvae available to infect sheep are the most significant effects. Moreover, climate warming may increase the number of years when development within one summer is possible, facilitating a shift from a multi- to a 1-year cycle (45, 56).

Climate warming may thus result in disease outbreaks (37, 53). Cerebrospinal disease outbreaks caused by *Elaphostrongylus rangiferi* in reindeer in northern Norway have occurred in years when summer temperatures were 1.5°C above normal (35). Climate warming can also have a very important impact on the transmission of *Protostrongylus stilesi*, a ubiquitous lungworm of bighorn and thinhorn sheep that can cause additive or even synergistic pulmonary pathology in conjunction with *P. odocoilei* in thinhorn sheep. Healthy Dall's sheep populations in the Mackenzie Mountains are valuable resources for subsistence and trophy hunting.

Hence, in Subarctic regions, both the length of the season during which temperatures are above the larval development threshold and the amount of warming available for parasite development have increased. Temperature currently limits the northward range expansion of protostrongylids, but climate warming may eliminate such constraints (45).

Dirofilariases

Several filarid species infect dogs and cats worldwide. *Dirofilaria immitis* and *D. repens* are the most prevalent species and are the agents of cardiopulmonary and subcutaneous dirofilariasis, respectively. Furthermore, where this disease is endemic, humans are at risk of developing pulmonary and subcutaneous lesions, mainly caused by *D. immitis* (31). Mosquito species of the genera *Culex, Culiseta, Aedes, Anopheles* and *Coquilletidia* have been incriminated in transmission. These filarids show very low mosquito specificity (11), and very low prevalences at vector level (0.3% to 8.6% in *D. immitis*) (5, 10, 51, 78). Animal dirofilariasis is distributed throughout temperate and tropical areas, with the highest prevalence in the USA, Japan, Australia and Italy (31, 105). In Europe, the prevalence in dogs and cats is usually low (2% to 36%), with high prevalence in Romania (65%) and northern Italy (50% to 80% in dogs; 24% in cats). At present, animal dirofilariasis in Europe is expanding into northern countries such as Switzerland, Austria, Germany, the United Kingdom, the Netherlands, Sweden and Hungary, where an increasing number of canine cases are being diagnosed (99).

Human dirofilariasis has been reported mainly in Southern European countries (Italy, Spain, France and Greece) (104). In parallel with the northward expansion in animals, human dirofilariasis has been reported in Austria, Germany, the United Kingdom and Belgium (99).

The existence of appropriate climatic conditions to support abundant mosquito populations and filarid larval development are key factors in the spread of these organisms (31). *Culex* species are able to adapt to widely differing environments, including urbanisation and polluted habitats. Moreover, the filarids can adapt to species that have been recently introduced, such as *Aedes albopictus* in southern Europe (10). Therefore, the introduction of new competent vector species into an endemic area as a consequence of climatic change could increase the risk of infection (9, 91).

Human dirofilariasis is currently considered an emerging disease in some areas of the world (52, 99). The general climatic trend, mainly of global warming, tends to extend the risk season for infection and to maintain a high incidence level. Infection with *D. immitis* has spread from the hyperendemic area of the Po River Valley to northern Alpine areas, despite temperatures in southern Italy being more favourable for transmission (99).

Acanthocephaliases

Thorny-headed worms may also suffer the consequences of climate change, as seen when analysing the impact of helminth fauna on hosts of commercial interest, such as fish. In a study of the environmental changes occurring off the Labrador coast in Canada from the late 1980s and their effects on the Atlantic cod, *Gadus morhua*, the abundance of an acanthocephalan parasite of the fish, *Echinorhynchus gadi*, declined after 1980 and then further after 1986. This was attributed to a change in fish feeding habits, because crustaceans are the main intermediate host (47). A similar decrease in the prevalence and intensity of *E. gadi* was attributed either to fewer crustaceans or to changes in salinity and pollution levels (48), although subsequent studies showed that salinity was stable and contaminants negligible. Consequently, it was concluded that climatic change was the responsible factor (47).

Conclusions

It is evident that helminth diseases may be listed among the infectious diseases to which special attention should be paid because of climate change in the future. As is the case for other infectious diseases, all evidence indicates that the effects of climate change on helminths are more patent in temperate and colder northern latitudes as well as in high altitude areas, where modifications of climate variables

appear to be more pronounced. First temperature and second the water-related variables are the meteorological factors that have been more frequently linked to the impact of climate change on helminths.

Acknowledgements

This article was developed within the framework of Project No. SAF2006-09278 of the Ministry of Education and Science, Madrid, the *Red de Investigación de Centros de Enfermedades Tropicales* – RICET (Project No. RD06/0021/0017 of the Programme of *Redes Temáticas de Investigación Cooperativa*) of the *Fondo de Investigacion Sanitaria* (FIS), Ministry of Health, Madrid, Spain, and Emerging Diseases in a Changing European Environment (EDEN) Project of grant No. GOCE-2003-010284 of the Global Change of Ecosystems Programme, Commission of the European Community (Science, Research and Development), Brussels, Belgium.

Effets du changement climatique sur les helminthiases zoonotiques

S. Mas-Coma, M.A. Valero & M.D. Bargues

Résumé

Les auteurs présentent l'état des connaissances actuelles sur les helminthiases animales et zoonotiques subissant l'influence avérée du changement climatique. Les variables climatiques affectent la prévalence, l'intensité et la distribution géographique des helminthes en exerçant une influence directe sur les formes larvaires qui se trouvent dans le milieu ambiant et une influence indirecte sur les hôtes (principalement invertébrés mais aussi vertébrés). L'impact du changement climatique paraît plus prononcé chez les trématodes, se manifestant principalement par une augmentation de la production et l'émergence de cercaires associées au réchauffement global. La fasciolase, la schistosomiase (*Schistosomia japonica*) et la dermatite cercarienne due aux schistosomes aviaires ont été particulièrement étudiées. L'échinococcose alvéolaire est actuellement la seule cestodose pour laquelle l'influence du climat a été démontrée. Les helminthiases les plus étudiées par rapport au changement climatique sont les nématodoses, y compris les infections à *Heterakis gallinarum*, plusieurs trichostrongyloses et protostrongyloses, ainsi que les ankylostomoses et les dirofilarioses. Ces études permettent de conclure que les helminthiases font partie des maladies infectieuses pour lesquelles l'impact du changement climatique devra être examiné avec une attention particulière, notamment dans les régions tempérées et dans les régions nordiques à climat froid, ainsi qu'en haute altitude.

Mots-clés

Acanthocéphale – Animal domestique – Animal sauvage – Cestode – Changement climatique – Nématode – Trématode – Zoonose.

Efectos del cambio climático en las helmintiasis animales y zoonóticas

S. Mas-Coma, M.A. Valero & M.D. Bargues

Resumen

Los autores pasan revista al conocimiento actual sobre las helmintiasis animales y zoonóticas en las que se ha detectado una influencia del cambio climático. Las variables climáticas pueden afectar la prevalencia e intensidad de estas parasitosis y la distribución geográfica de los helmintos, influyendo directamente en las fases larvarias (de vida libre) e indirectamente en los hospedadores, sobre todo invertebrados, pero también vertebrados. El cambio climático parece tener efectos más pronunciados en los nemátodos, que se manifiestan básicamente por una mayor producción y aparición de cercarias, en vinculación directa con el calentamiento planetario. El estudio que los autores exponen está centrado en la fascioliasis, la esquistosomiasis (*Schistosomia japonica*) y la dermatitis por cercarias causada por esquistosomas aviares. Por ahora la equinococosis alveolar es la única cestodiasis cuya sensibilidad al cambio climático está comprobada. Las nematodosis (comprendidas las heterakiasis, distintas tricostrongiliasis y protostrongiliasis, anquilostomiasis y dirofilariasis) son las enfermedades helmínticas cuya relación con el cambio climático se ha analizado más a fondo. De tales estudios cabe concluir que dichas patologías deberían figurar entre las enfermedades infecciosas con las que en el futuro, ante el cambio climático que se avecina, habría que extremar las precauciones, sobre todo en latitudes templadas y septentrionales y en zonas de gran altitud.

Palabras clave

Acantocefaliasis – Animal doméstico – Animal salvaje – Cambio climático – Cestodiasis – Nematodosis – Trematodiasis – Zoonosis.

References

1. Albon S.D., Stien A., Irvine R.J., Langvatn R., Ropstad E. & Halvorsen O. (2002). – The role of parasites in the dynamics of a reindeer population. *Proc. roy. Soc. Lond., B, biol. Sci.*, **269**, 1625-1632.

2. Anderson R.C. (2000). – Nematode parasites of vertebrates: their development and transmission, 2nd Ed. CAB International, Wallingford, Oxon, United Kingdom.

3. Arme C. & Pappas P.W. (eds) (1983). – The biology of the Eucestoda. Vols. 1-2. Academic Press Ltd, London.

4. Astrup A. (1995). – *Uncinaria stenocephala* as a cause of skin disease in man. *Acta derm. venereol.*, **25**, 389-392.

5. Bargues M.D., Morchon R., Latorre J.M., Cancrini G., Mas-Coma S. & Simon F. (2006). – Ribosomal DNA second intergenic spacer sequence studies of culicid vectors from an endemic area of *Dirofilaria immitis* of western Spain. *Parasitol. Res.*, **99**, 205-213.

6. Bargues M.D., Vigo M., Horak P., Dvorak J., Patzner R.A., Pointier J.P., Jackiewicz M., Meier-Brook C. & Mas-Coma S. (2001). – European Lymnaeidae (Mollusca: Gastropoda), intermediate hosts of trematodiases, based on nuclear ribosomal DNA ITS-2 sequences. *Infect. Genet. Evol.*, **1** (2), 85-107.

7. Bossaert K., Lonneux J.F., Losson B. & Peters J. (1999). – Fascioliasis incidence forecasts in Belgium by means of climatic data. *Ann. Méd. vét.*, **143**, 201-211.

8. Brooker S., Rowlands M., Haller L., Savioli L. & Bundy D.A.P. (2000). – Towards an atlas of human helminth infection in sub-Saharan Africa: the use of geographical information systems (GIS). *Parasitol. Today*, **16**, 303-307.

9. Cancrini G., Allende E., Favia G., Bornay F., Anton F. & Simon F. (2000). – Canine dirofilariosis in two cities of southeastern Spain. *Vet. Parasitol.*, **92**, 81-86.

10. Cancrini G., Frangipane di Regalbono A., Ricci I., Tessarin C., Gabrielli S. & Pietrobelli M. (2003). – *Aedes albopictus* is a natural vector of *Dirofilaria immitis* in Italy. *Vet. Parasitol.*, **118**, 195-200.

11. Cancrini G. & Kramer L.H. (2001). – Insect vectors of *Dirofilaria* spp. *In* Heartworm infection in humans and animals (F. Simon & C. Genchi, eds). Ediciones Universidad de Salamanca, Salamanca, 63-82.

12. Cattadori I.M., Haydon D. & Hudson P.J. (2005). – Parasites and climate synchronize red grouse populations. *Nature*, **43**, 1163-1169.

13. Caumes E., Felder-Moinet S., Couzigou C., Darras-Joly C., Latour P. & Leger N. (2003). – Failure of an ointment based on IR3535 (ethyl butylacetylaminoptopionate) to prevent an outbreak of cercarial dermatitis during swimming races across Lake Annecy, France. *Ann. trop. Med. Parasitol.*, **97** (2), 157-163.

14. Chamot E., Toscani E. & Rougemont A. (1998). – Public health importance and risk factors for cercarial dermatitis associated with swimming in Lake Leman at Geneva, Switzerland. *Epidemiol. Infect.*, **120**, 305-314.

15. Cotton P.A. (2003). – Avian migration phenology and global climate change. *Proc. natl Acad. Sci. USA*, **100**, 12219-12222.

16. De Gentile L., Picot H., Bourdeau P., Bardet R., Kerjan M., Piriou M., Guennic A.L., Bayssade-Dufour C., Chabasse D. & Mott K.E. (1996). – La dermatite cercarienne en Europe: un problème de santé publique nouveau? *Bull. WHO*, **74**, 159-163.

17. Dobson A.P. & Hudson P.J. (1992). – Regulation and stability of a free-living host-parasite system, *Trichostrongylus tenuis* in red grouse. II: Population models. *J. anim. Ecol.*, **61**, 487-498.

18. Dobson A.P., Kutz S., Pascual M. & Winfree R. (2003). – Pathogens and parasites in a changing climate. *In* Climate change and biodiversity: synergistic impacts (L. Hannah & T.E. Lovejoy, eds). Advances in Applied Biodiversity Science 4. Centre for Applied Biodiversity Science, Conservation International, Washington DC, United States of America, 33-38.

19. Dubois J.P., Buet A., Cusin I., Deloraine A., Gerdil S., Hedreville L. & Maurin G. (2001). – Epidemiological studies related to cercarial dermatitis in lakes of the Savoy District (France). *Helminthologia*, **38**, 244.

20. Eckert J. & Deplazes P. (1999). – Alveolar echinococcosis in humans: the current situation in central Europe and the need for countermeasures. *Parasitol. Today*, **15** (8), 315-319.

21. Eckert J., Gottstein B., Heath D. & Liu F.J. (2001). – Prevention of echinococcosis in humans and safety precautions. *In* WHO/OIE manual on echinococcosis in humans and animals: a public health problem of global concern (J. Eckert, M.A. Gemmell, F.-X. Meslin & Z.S. Pawlowski, eds). World Organisation for Animal Health, Paris, 238-247.

22. Eckert J., Schantz P.M., Gasser R.B., Torgerson P.R., Besonov A.S., Movsessian S.O., Thakur A., Grimm F. & Nikogossian M.A. (2001). – Geographic distribution and prevalence. *In* WHO/OIE manual on echinococcosis in humans and animals: a public health problem of global concern (J. Eckert, M.A. Gemmell, F.-X. Meslin & Z.S. Pawlowski, eds). World Organisation for Animal Health, Paris, 100-142.

23. Erasmus D.A. (1972). – The biology of trematodes. Edward Arnold (Publ.), London.

24. Eysker M., Bakker N., Kooyman F.N.J., Van der Linden D., Schrama C. & Ploeger H.W. (2005). – Consequences of the unusually warm and dry summer of 2003 in the Netherlands: poor development of free living stages, normal survival of infective larvae and long survival of adult gastrointestinal nematodes of sheep. *Vet. Parasitol.*, **133**, 313-321.

25. Fenwick A., Rollinson D. & Southgate V. (2007). – Implementation of human schistosomiasis control: challenges and prospects. *In* Control of human parasitic diseases (D.H. Molyneux, ed.). Elsevier-Academic Press, London, 567-622.

26. Fuentes M.V. & Malone J.B. (1999). – Development of a forecast system for fascioliasis in central Chile using remote sensing and climatic data in a geographic information system. *Res. Rev. Parasitol.*, **59**, 129-134.

27. Fuentes M.V., Malone J.B. & Mas-Coma S. (2001). – Validation of a mapping and predicting model for human fasciolosis transmission in Andean very high altitude endemic areas using remote sensing data. *Acta trop.*, **79**, 87-95.

28. Fuentes M.V., Sainz-Elipe S., Nieto P., Malone J.P. & Mas-Coma S. (2005). – Geographical Information Systems risk assessment models for zoonotic fasciolosis in the South American Andes region. *In* Geographic information systems and remote sensing in parasitology (J.B. Malone, ed.). *Parasitologia (Rome)* (Special Issue), **47**, 151-156.

29. Fuentes M.V., Valero M.A., Bargues M.D., Esteban J.G., Angles R. & Mas-Coma S. (1999). – Analysis of climatic data and forecast indices for human fascioliasis at very high altitude. *Ann. trop. Med. Parasitol.*, **93** (8), 835-850.

30. Galaktionov K.V. & Dovrovolskij A.A. (2003). – The biology and evolution of trematodes. Kluwer Academic Publishers, Dordrecht, the Netherlands.

31. Genchi C., Kramer L.H. & Prieto G. (2001). – Epidemiology of canine and feline dirofilariosis: a global view. *In* Heartworm infection in humans and animals (F. Simon & C. Genchi, eds). Ediciones Universidad de Salamanca, Salamanca, 63-82.

32. Giraudoux P., Craig P., Delattre P., Bao G., Bartholomot B., Harraga S., Quere J.P., Raoul F., Wang Y., Shi D. & Vuitton D.A. (2003). – Interactions between landscape changes and host communities can regulate *Echinococcus multilocularis* transmission. *Parasitology*, **127**, S121-131.

33. Giraudoux P., Delattre P., Takahaski K., Raoul F., Quere J.P., Craig P. & Vuitton D.A. (2002). – Transmission ecology of *Echinococcus multilocularis* in wildlife: what can be learned from comparative studies and multiscale approaches? *In* Cestode zoonoses: echinococcosis and cysticercosis (P. Craig & Z. Pawlowski, eds). IOS Press, Amsterdam, 251-266.

34. Githeko A.K., Lindsay S.W., Confalonieri U.E. & Patz J.A. (2000). – Climate change and vector-borne diseases: a regional analysis. *Bull. WHO*, **78** (9), 1136-1147.

35. Handeland K. & Slettbakk T. (1994). – Outbreaks of clinical cerebrospinal elaphostrongylosis in reindeer (*Rangifer tarandus tarandus*) in Finnmark, Norway, and their relation to climatic conditions. *J. vet. Med.*, B, **41**, 407-410.

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

37. Hoberg E.P., Kutz S.J., Galbreath K. & Cook J. (2001). – Arctic biodiversity: from discovery to faunal baseline – revealing the history of a dynamic ecosystem. *J. Parasitol.*, **89**, S84-S95.

38. Horak P., Kolarova L. & Adema C. (2000). – Biology of the schistosome genus *Trichobilharzia. Adv. Parasitol.*, **52**, 155-233.

39. Hudson P.J. (1986). – The effect of a parasitic nematode on the breeding production of red grouse. *J. anim. Ecol.*, **55**, 85-94.

40. Hudson P.J., Cattadori I.M., Boag B. & Dobson A.P. (2006). – Climate disruption and parasite-host dynamics: patterns and processes associated with warming and the frequency of extreme climatic events. *J. Helminthol.*, **80**, 175-182.

41. Hudson P.J., Dobson A.P. & Newborn D. (1998). – Prevention of population cycles by parasite removal. *Science*, **282**, 2256-2258.

42. Hudson P.J., Newborn D. & Dobson A.P. (1992). – Regulation and stability of a free-living host-parasite system, *Trichostrongylus tenuis* in red grouse. I: Monitoring and parasite reduction experiments. *J. anim. Ecol.*, **61**, 477-486.

43. Huh O.K. & Malone J.B. (2001). – New tools: potential medical applications of data from new and old environmental satellites. *Acta trop.*, **79** (1), 35-47.

44. Jenkins D.J., Romig T. & Thompson R.C.A. (2005). – Emergence/re-emergence of *Echinococcus* spp. – a global update. *Int. J. Parasitol.*, **35**, 1205-1219.

45. Jenkins E.J., Veitch A.M., Kutz S.J., Hoberg E.P. & Polley L. (2006). – Climate change and the epidemiology of protostrongylid nematodes in northern ecosystems: *Parelaphostrongylus odocoilei* and *Protostrongylus stilesi* in Dall's sheep (*Ovis d. dalli*). *Parasitology*, **132**, 387-401.

46. Joyeux Ch. & Baer J.G. (1961). – Classe des cestodes. Cestoidea Rudolphi. *In* Traité de zoologie, Vol. 4, Part 1 (P.P. Grassé, ed.). Masson et Cie, Paris, 347-560.

47. Khan R.A. & Chandra C.V. (2006). – Influence of climatic changes on the parasites of Atlantic cod *Gadus morhua* off coastal Labrador, Canada. *J. Helminthol.*, **80**, 193-197.

48. Khan R.A. & Thulin J. (1991). – Influence of pollution on parasites of aquatic animals. *Adv. Parasitol.*, **30**, 201-238.

49. Klose C., Mravak S., Geb M., Bienzle U. & Meyer C.G. (1996). – Autochthonous cutaneous larva migrans in Germany. *Trop. Med. int. Hlth*, **1** (4), 503-504.

50. Kolarova L., Skirnisson K. & Horak P. (1999). – Schistosome cercariae as the causative agent of swimmer's itch in Iceland. *J. Helminthol.*, **73**, 215-220.

51. Konishi E. (1989). – Enzyme-linked immunosorbent assay to detect antigens of *Dirofilaria immitis* (Spirurida: Filariidae) larvae in *Aedes albopictus* and *Culex tritaeniorhynchus* (Diptera: Culicidae). *J. med. Entomol.*, **26**, 113-117.

52. Kramer L.H., Kartashev V.V., Grandi G., Morchon R., Nagornii S.A. & Karanis P. (2007). – Human subcutaneous dirofilariosis in Russia. *Emerg. infect. Dis.*, **13**, 150-152.

53. Kutz S.J., Hoberg E.P., Nagy J., Polley L. & Elkin B. (2004). – 'Emerging' parasitic infections in arctic ungulates. *Integr. comp. Biol.*, **44**, 109-118.

54. Kutz S.J., Hoberg E.P., Nishi J. & Polley L. (2002). – Development of the muskox lungworm, *Umingmakstrongylus pallikuukensis* (Protostrongylidae), in gastropods in the Arctic. *Can. J. Zool.*, **80**, 1977-1985.

55. Kutz S.J., Hoberg E.P. & Polley L. (2001). – A new lungworm in muskoxen: an exploration in Arctic parasitology. *Trends Parasitol.*, **17** (6), 276-280.

56. Kutz S.J., Hoberg E.P., Polley L. & Jenkins E.J. (2005). – Global warming is changing the dynamics of Arctic host-parasite systems. *Proc. roy. Soc. Lond., B, biol. Sci.*, **272** (1581), 2571-2576. DOI: 10.1098/rspb.2005.3285.

57. Kutz S.J., Hoberg E.P. & Polley L. (2001). – *Umingmakstrongylus pallikuukensis* (Nematoda: Protostrongylidae) in gastropods: larval morphology, morphometrics, and development rates. *J. Parasitol.*, **87**, 527-535.

58. Lindblade K.A. (1998). – The epidemiology of cercarial dermatitis and its association with limnological characteristics of a northern Michigan lake. *J. Parasitol.*, **84** (1), 19-23.

59. Lodge D.M., Brown K.M., Klosiewski S.P., Stein R.A., Covich A.P., Leathers B.K. & Bronmark C. (1987). – Distribution of freshwater snails: spatial scale and the relative importance of physicochemical and biotic factors. *Am. malacol. Bull.*, **5**, 73-84.

60. Malone J.B., Bergquist N.R., Huh O.K., Bavia M.E., Bernardi M., El Bahy M.M., Fuentes M.V., Kristensen T.K., McCarroll J.C., Yilma J.M. & Zhou X.N. (2001). – A global network for the control of snail-borne disease using satellite surveillance and geographic information systems. *Acta trop.*, **79** (1), 7-12.

61. Malone J.B., Fehler D.P., Loyacano A.F. & Zukowski S.H. (1992). – Use of LANDSAT MSS Imagery and soil type in a geographic information system to assess site-specific risk of fascioliasis on Red River Basin farms in Louisiana. *In* Tropical veterinary medicine: current issues and perspectives (J.C. Williams, K.M. Kocan & P.J. Gibbs, eds). *Ann. N.Y. Acad. Sci.*, **653**, 389-397.

62. Malone J.B., Gommes R., Hansen J., Yilma J.M., Slingenberg J., Snijders F., Nachtergaele F. & Ataman E. (1998). – A geographic information system on the potential distribution and abundance of *Fasciola hepatica* and *F. gigantica* in east Africa based on Food and Agriculture Organization databases. *Vet. Parasitol.*, **78**, 87-101.

63. Malone J.B., Williams T.E., Muller R.A., Geaghan J.P. & Loyacano A.F. (1987). – Fascioliasis in cattle in Louisiana: development of a system to predict disease risk by climate, using the Thornthwaite water budget. *Am. J. vet. Res.*, **48**, 1167-1170.

64. Malone J.B. & Yilma J.M. (1999). – Predicting outbreaks of fasciolosis: from Ollerenshaw to satellites. *In* Fasciolosis (J.P. Dalton, ed.), CAB International Publishing, Wallingford, Oxon, United Kingdom, 151-183.

65. Mas-Coma S. (2004). – Human fascioliasis. *In* Waterborne zoonoses: identification, causes and control (J.A. Cotruvo, A. Dufour, G. Rees, J. Bartram, R. Carr, D.O. Cliver, G.F. Craun, R. Fayer & V.P.J. Gannon, eds). Published on behalf of the World Health Organization by IWA Publishing, London, 305-322.

66. Mas-Coma S. (2005). – Epidemiology of fascioliasis in human endemic areas. *J. Helminthol.*, **79** (3), 207-216.

67. Mas-Coma S., Angles R., Esteban J.G., Bargues M.D., Buchon P., Franken M. & Strausss W. (1999). – The Northern Bolivian Altiplano: a region highly endemic for human fascioliasis. *Trop. Med. int. Hlth*, **4** (6), 454-467.

68. Mas-Coma S. & Bargues M.D. (1997). – Human liver flukes: a review. *Res. Rev. Parasitol.*, **57** (3-4), 145-218.

69. Mas-Coma S., Bargues M.D. & Valero M.A. (2005). – Fascioliasis and other plant-borne trematode zoonoses. *Int. J. Parasitol.*, **35**, 1255-1278.

70. Mas-Coma S., Bargues M.D., Valero M.A. & Fuentes M.V. (2003). – Adaptation capacities of *Fasciola hepatica* and their relationships with human fascioliasis: from below sea level up to the very high altitude. *In* Taxonomy, ecology and evolution of metazoan parasites (C. Combes & J. Jourdane, eds). Presses Universitaires de Perpignan, Perpignan, France, 2, 81-123.

71. Mas-Coma S., Funatsu I.R. & Bargues M.D. (2001). – *Fasciola hepatica* and lymnaeid snails occurring at very high altitude in South America. *Parasitology*, **123**, S115-S127.

72. Michel J.F. (1974). – Arrested development of nematodes and some related phenomena. *Adv. Parasitol.*, **12**, 279-366.

73. Minchella D.J. & Scott M.E. (1991). – Parasitism: a cryptic determinant of animal community structure. *Trends Ecol. Evol.*, **6**, 250-254.

74. Mitchell G.B. (2002). – Update on fascioliasis in cattle and sheep. *In Practice*, **24**, 378-385.

75. Mitchell G.B. & Somerville D.K. (2005). – Effects of climate change on helminth diseases in Scotland. SAC Veterinary Centre Auchincruive, Ayr, United Kingdom, 1-11.

76. Miterparkova M., Dubinsky P., Reiterova K., Stanko M., Stefanik P., Rybanska L., Cervenska M., Kuzma L. & Kasencak M. (2006). – The effect of climatic and ecological factors on spreading of tapeworm *Echinococcus multilocularis* in the territory of Slovakia – results of long-term monitoring. *Slov. vet. Casopis*, **31** (4), 232-235.

77. Moodley I., Kleinschmidt I., Sharp B., Craig M. & Appleton C. (2003). – Temperature-suitability maps for schistosomiasis in South Africa. *Ann. trop. Med. Parasitol.*, **97**, 617-627.

78. Morchon R., Bargues M.D., Latorre J.M., Melero-Alcibar R., Pou-Barreto C., Mas-Coma S. & Simon F. (2007). – Haplotype H1 of *Culex pipiens* implicated as natural vector of *Dirofilaria immitis* in an endemic area of Western Spain. *Vector-borne zoon. Dis.*, **7** (4), 653-658.

79. Morgan J.A.T., De Jong R.J., Snyder S.D., Mkoji G.M. & Loker E.S. (2001). – *Schistosoma mansoni* and *Biomphalaria*: past history and future trends. *Parasitology*, **123**, S211-S228.

80. Mouritsen K.N. & Poulin R. (2002). – Parasitism, community structure and biodiversity in intertidal ecosystems. *Parasitology*, **124**, S101-S117.

81. Mowlavi G.R., Massoud J., Mobedi I., Solaymani-Mohammadi S., Gharagozlou M.J. & Mas-Coma S. (2006). – Very highly prevalent *Macracanthorhynchus hirudinaceus* infection of wild boar *Sus scrofa* in Khuzestan province, south-western Iran. *Helminthologia*, **43** (2), 86-91.

82. Neafie R.C. & Marty A.M. (2000). – Acanthocephaliasis. *In* Pathology of infectious diseases, Vol. 1. Helminthiases (W.M. Meyers, R.C. Neafie, A.M. Marty & D.J. Wear, eds). Armed Forces Institute of Pathology and American Registry of Pathology, Washington, DC, 519-529.

83. Ollerenshaw C.B. (1971). – The influence of climate on the life cycle of *Fasciola hepatica* in Britain with some observations on the relationship between climate and the incidence of fasciolasis in the Netherlands. *In* Facts and reflections, Symposium proceedings (3 June 1971). Parasitology Department of the Centraal Diergeneeskundig Institut, Lelystad, the Netherlands, 41-63.

84. Ollerenshaw C.B. (1973). – A comment on the epidemiology of *Fasciola hepatica* in Italy. *Ann. Fac. Med. vet. (Tor.)*, **20**, 83-121.

85. Ollerenshaw C.B. & Rowlands W.T. (1959). – A method of forecasting the incidence of fascioliasis in Anglesey. *Vet. Rec.*, **71**, 591-598.

86. Ollerenshaw C.B. & Smith L.P. (1969). – Meteorological factors and forecast of helminthic diseases. *Adv. Parasitol.*, **7**, 283-232.

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

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

89. Pritchbard G.C., Forbes A.B., Williams D.J.L., Salimi-Bejestami M.R. & Daniel R.G. (2005). – Emergence of fasciolosis in cattle in East Anglia. *Vet. Rec.*, **157**, 578-582.

90. Prociv P. & Croese J. (1990). – Human eosinophilic enteritis caused by dog hookworm *Ancylostoma caninum*. *Lancet*, **335**, 1299-1302.

91. Romi R. (2001). – *Aedes albopictus* in Italy: an underestimated health problem. *Ann. Ist. sup. Sanità*, **37**, 241-247.

92. Romig T., Thoma D. & Weible A.K. (2006). – *Echinococcus multilocularis* – a zoonosis of anthropogenic environments? *J. Helminthol.*, **80**, 207-212.

93. Saunders L.M., Tompkins D.M. & Hudson P.J. (2000). – The role of oxygen availability in the embryonation of *Heterakis gallinarum* eggs. *Int. J. Parasitol.*, **30**, 1481-1485.

94. Saunders L.M., Tompkins D.M. & Hudson P.J. (2002). – Stochasticity accelerates nematode egg development? *J. Parasitol.*, **88**, 1271-1272.

95. Scott M.E. (1987). – Regulation of mouse colony abundance by *Heligmosomoides polygyrus*. *Parasitology*, **95**, 111-124.

96. Schmidt G.D. (1985). – Development and life cycles. *In* Biology of the Acanthocephala (D.W.T. Crompton & B.B. Nickol, eds). Cambridge University Press, Cambridge, 273-286.

97. Seto E.Y.W., Wu W.P., Qiu D.C., Liu H.Y., Gu X.G., Chen H.G., Spear R.C. & Davis G.M. (2002). – Impact of soil chemistry on the distribution of *Oncomelania hupensis* (Gastropoda: Pomatiopsidae) in China. *Malacologia*, **44**, 259-272.

98. Seto E., Xu B., Liang S., Gong P., Wu W.P., Davis G., Qiu D.C., Gu X.G. & Spear R. (2002). – The use of remote sensing for predictive modeling of schistosomiasis in China. *Photogr. Engin. remote Sens.*, **68**, 167-174.

99. Simon F., Lopez-Belmonte J., Marcos-Atxutegi C., Morchon R. & Martin-Pacho J.R. (2005). – What is happening outside North America regarding human dirofilariasis? *Vet. Parasitol.*, **133**, 181-189.

100. Simon-Martin F. & Simon-Vicente F. (1999). – The life cycle of *Trichobilharzia salmanticensis* n. sp. (Digenea: Schistosomatidae), related to cases of human dermatitis. *Res. Rev. Parasitol.*, **59**, 13-18.

101. Thomas C., Jacquiet P. & Dorchies P. (2007). – La prévalence des helminthoses bovines a-t-elle été modifiée par la canicule de l'été 2003 dans le Sud-Ouest de la France? *Parasite*, **14**, 265-268.

102. Thors C. & Linder E. (2001). – Swimmer's itch in Sweden. *Helminthologia*, **38**, 244.

103. Veit P., Bilger B., Schad V., Schafer J., Frank W. & Lucius R. (1995). – Influence of environmental factors on the infectivity of *Echinococcus multilocularis* eggs. *Parasitology*, **110** (1), 79-86.

104. Velez I.D., Montoya M.N., Prieto G., Morchon R. & Simon F. (2001). – Epidemiology of human dirofilariosis. *In* Heartworm infection in humans and animals (F. Simon & C. Genchi, eds). Ediciones Universidad de Salamanca, Salamanca, 135-146.

105. Venco L. & Vezzoni A. (2001). – Heartworm (*Dirofilaria immitis*) disease in dogs and cats. *In* Heartworm infection in humans and animals (F. Simon & C. Genchi, eds). Ediciones Universidad de Salamanca, Salamanca, 161-177.

106. Yang G.J., Gemperli A., Vounatsou P., Tanner M., Zhou X.N. & Utzinger J. (2006). – A growing degree-days based time series analysis for prediction of *Schistosoma japonicum* transmission in Jiangsu Province, China. *Am. J. trop. Med. Hyg.*, **75** (3), 549-555.

107. Yilma J.M. & Malone J.B. (1998). – A geographic information system forecast model for strategic control of fascioliasis in Ethiopia. *Vet. Parasitol.*, **78**, 103-127.

108. Zhou X.N., Lin D.D., Yang H.M., Chen H.G., Sun L.P., Yang G.J., Hong Q.B., Brown L. & Malone J.B. (2002). – Use of Landsat TM satellite surveillance data to measure the impact of the 1998 flood on snail intermediate host dispersal in the lower Yangtze River basin. *Acta trop.*, **82**, 199-205.

109. Zhou X.N., Wang L.Y., Chen M.G., Wu X.H., Jiang Q.W., Chen X.Y., Zheng J. & Utzinger J. (2005). – The public health significance and control of schistosomiasis in China – then and now. *Acta trop.*, **96**, 97-105.

110. Zhou X.N., Yang G.J., Sun L.P., Hong Q.B., Yang K., Wang R.B. & Hua Z.H. (2002). – Potential impact of global warming on the transmission of schistosomiasis. *Chin. J. Epidemiol.*, **23**, 83-86.