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# Vertebrate reservoirs and secondary epidemiological cycles of vector-borne diseases

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**Summary**
Vector-borne diseases of importance to human and domestic animal health are listed and the increasing emergence of syndromes, new epidemiological cycles and distributions are highlighted. These diseases involve a multitude of vectors and hosts, frequently for the same pathogen, and involve natural enzootic cycles, wild reservoirs and secondary epidemiological cycles, sometimes affecting humans and domestic animals. On occasions the main reservoir is in the domestic environment. Drivers for secondary cycles are mainly related to human impacts and activities and therefore, for purposes of prevention and control, the focus needs to be on the socioecology of the diseases. Technical and therapeutical solutions exist, and for control there needs to be a clear understanding of the main vertebrate hosts or reservoirs and the main vectors. The targets of interventions are usually the vector and/or secondary epidemiological cycles and, in the case of humans and domestic animals, the spillover or incidental hosts are treated. More attention needs to be given to the importance of the political economy in relation to vector-borne diseases, as many key drivers arise from globalisation, climate change and changes in structural ecologies. Attention to reducing the risk of emergence of new infection cycles through better management of the human–animal–environment interface is urgently needed.

**Keywords**
Emerging infectious disease – Epidemiology – Vector-borne disease.

## Introduction

This paper discusses arthropod vectors and vector-borne diseases (VBDs) and their emergence.

A large number of viral, bacterial and protozoan infections carried by blood-sucking arthropod vectors have multiple vertebrate hosts, including humans; most of the arthropod-borne viruses (arboviruses) of animals are zoonotic (1).

Many VBDs have preferential vertebrate host(s), i.e. birds and terrestrial mammals (rarely bats), in which infection can be described as 'natural', as it is enzootic and mainly benign. The dynamic balance that exists between host, vector and pathogen is strongly influenced by their ecology. Deciding on whether a particular species is a maintenance or a reservoir host is challenging and in the case of many animals it is not yet known what type of host they are (2).

Ecosystem change influences the distribution and epidemic cycling of VBD pathogens, resulting in unstable transmission and evolutionary settings, often on the boundaries of their geography (3). Changing ecological conditions can result in the pathogen switching host or vector and leads to emergence of new pathogens in the domestic environment. A good example of host switching is shown by dengue fever virus: it is believed that the pathogen was once isolated to an enzootic lower primate–mosquito cycle in Africa and Asia, but it would appear that it has shifted over the past century to a secondary cycle in humans, where it now persists in the absence of the sylvatic host (4).

The most significant ecological changes with respect to infectious disease emergence have been driven by human activities (5, 6, 7). These factors include: rising global mean ambient temperatures (described as climate change); changed status, genetics and distribution of many vertebrates and invertebrate populations (through animal domestication, habitat destruction, killing, artificial movements and introductions, human population growth). The impact of concern, in these changed structural ecologies, is increased contact rates between domestic host populations, the vectors and novel microorganisms, resulting in secondary epidemiological cycles and disease.

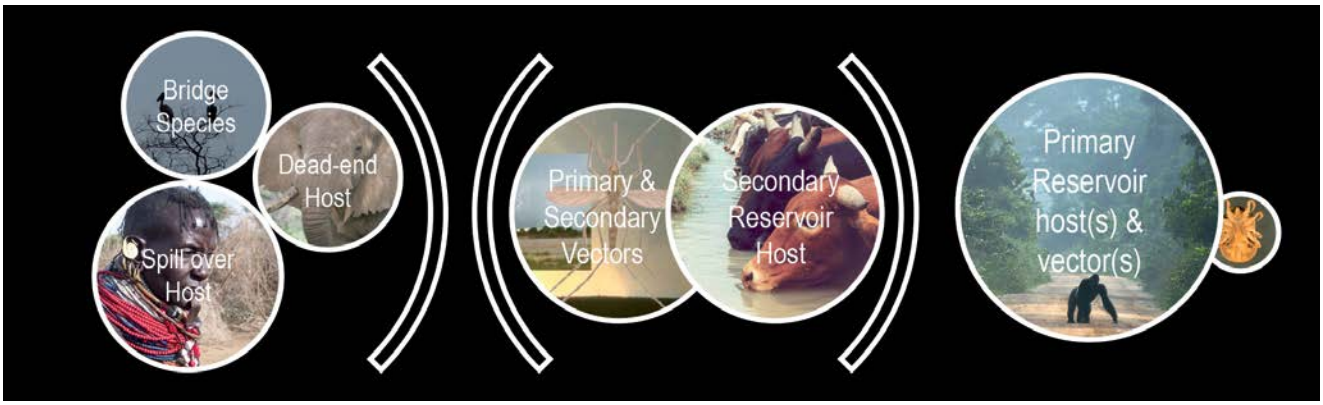


**Fig. 1**
**Illustration of key epidemiological cycles showing ecological boundaries, represented by brackets, which under certain conditions are breached**

A number of species can act as spillover or dead-end hosts with little epidemiological significance or as bridging hosts that can drive pathogen jumps, setting up secondary cycles.

Pathogens in VBDs use a range of strategies in order to survive, with the main reproductive phase usually in the vertebrate host. Unlike the pathogens of directly transmitted infections, vector-borne pathogens are characterised by high reproductive rates, where the vector can transmit many times after feeding on a single infected host, a good example being malaria. These diseases are usually driven by seasonal precipitation, rising temperatures and an associated abundance of arthropod vectors. Transmission and amplification is sometimes facilitated by vectors feeding across a spectrum of species, potentially across whole populations and wide geographical areas. Airborne vectors easily circumvent geographical or artificial physical barriers.

The purpose of this review is to provide:

*i)* lists of the most important diseases, with secondary epidemiological cycles and key references (see Tables I & II)

*ii)* illustrations of common emergence factors for VBDs, using a series of case studies

*iii)* recommendations on priority actions to reduce the risk of secondary epidemiological cycles affecting humans and domestic animals.

# Case studies of emergence factors for vector-borne diseases of humans and domestic animals

The most interesting and arguably most socioeconomically significant emerging VBDs are caused by arboviruses, but many VBDs involving bacteria and parasites have significant secondary epidemiological cycles. In this section, common emergence factors for these diseases are discussed.

## Agriculture and pathogen amplification cycles

Arboviruses such as Japanese encephalitis virus have exploited rural environments, shifting from enzootic wildlife hosts to secondary cycles in domestic animals, notably pigs, where the virus amplifies and spills over into adjacent human populations (37, 38). Irrigation-based agriculture associated with rural development further exacerbates this effect, through expansion of breeding habitats for the *Culex* vectors (39). The rapid development of much of South and South-East Asia has resulted in expansion of the range of this virus into the agricultural areas.

Patterns of infection with West Nile virus, a recently introduced pathogen in the United States, show higher prevalence in agricultural and urban areas (16) and, paradoxically, reduced prevalence of infected mosquitoes in natural wetlands. When modelled, vector density, abundance of amplification hosts and composition of the host community are probable risk factors (40).

Alteration of natural ecologies through the introduction of agriculture and livestock into natural habitat in Africa and South America, over centuries, has probably driven the emergence in cattle and humans in Africa of trypanosomosis, caused by *Trypanosoma brucei* spp. spillover from wildlife natural hosts via tsetse fly (*Glossina* spp.) vectors. In human African trypanosomosis, human–tsetse–human secondary epidemiological cycles are established and some authors consider this to be more significant than transmission of infection from animal reservoirs to humans, although this is disputed in some settings (41, 42). The wildlife–livestock cycle remains significant in most affected regions but secondary cycles in cattle alone occur in the absence

**Table I**
**Important vector-borne diseases, vertebrate reservoirs and secondary epidemiological cycles: arboviruses**

| Disease, causative agent | Geographical distribution[a] | Vertebrate reservoir[b] | Arthropod vector | Secondary epidemiological cycles – maintenance host(s)[c] | Key references |
|---|---|---|---|---|---|
| **Asfariviridae** | | | | | |
| African swine fever | Africa, Asia | Wild African suids | *Ornithodoros* ticks, *Stomoxys* biting flies | Domestic pigs, wild boar | 8, 9 |
| **Orbiviridae** | | | | | |
| African horse sickness | Africa, Europe, South Asia | Zebra, donkeys | *Culicoides* midges | Horses, mules, dogs | 10 |
| Bluetongue | Global, Middle East, Europe | Wild ruminants | *Culicoides* midges | Domestic ruminants | 11, 12 |
| Epizootic haemorrhagic disease | North America, Australia, Asia, Africa | Wild ungulates | *Culicoides* midges, mosquitoes | Domestic ruminants (rare) | 13 |
| **Flaviviridae** | | | | | |
| Yellow fever | Africa | Primates | *Aedes* mosquitoes | Humans | 14 |
| Tick-borne encephalitis virus | Eurasia, North Africa | Rodents | *Ixodes* ticks | Humans, domestic/wild ruminants, dogs | 15 |
| West Nile virus | Africa, Americas, Eurasia | Wild birds (>600 species) | Mosquitoes (>70 species); *Stomoxys* biting flies | Humans, horses, poultry, other mammals | 16 |
| Japanese encephalitis virus | South-East Asia, South Asia | Wild wading ardeid water birds | *Culex* mosquitoes | Pigs, horses, donkeys | 17 |
| Dengue (sylvatic genotype 1, 2, 4) | Global | Primates | Arboreal *Aedes* mosquitoes | Humans | 4, 18 |
| St Louis encephalitis virus | Americas | Wild birds | *Culex* mosquitoes | Humans | 19 |
| Louping ill | | Sheep | Ticks | Cattle, horses, pigs, dogs, wildlife | 20 |
| **Alphaviridae** | | | | | |
| Equine encephalitis viruses | Americas | Wild birds, rodents | *Culex* & *Aedes* mosquitoes | Humans, horses | 20 |
| Chikungunya virus | Africa, Australia, Caribbean, Eurasia | Primates, birds, rodents | *Aedes* mosquitoes | Humans | 21 |
| Ross River virus | Australia | Marsupials | *Culex* mosquitoes | Humans? | 22 |
| Other rarely reported alphavirus zoonotic infections[d] | | | | | |
| **Bunyaviridae** | | | | | |
| Crimean-Congo haemorrhagic fever | Africa, Eurasia | Camels, cattle, wild ungulates, carnivores, birds | *Hyalomma* ticks | Humans | 23 |
| Schmallenberg virus | Europe (including the United Kingdom) | Wildlife? | *Culicoides* mosquitoes | Sheep, cattle, goats | 24 |
| Akabane virus | Australasia, Africa, Middle East | Wild ruminants | *Culicoides* mosquitoes | Domestic ruminants | 20 |
| Rift Valley fever virus | Africa, Middle East | *Aedes* mosquitoes Vertebrate hosts in inter-epidemic periods? | Mosquitoes | Humans, wild & domestic ruminants | 25 |

a) Including recent and historical distribution
b) Natural host or source ancestral host
c) Clinically affected/mortality reported, maintenance and transmission hosts
d) Barmah Forest, o'nyong-nyong and Semliki Forest viruses (Africa); Mayaro (South America), Sindbis and Sindbis-like viruses (Africa, Asia, Scandinavia and Russia) (20, 22)

**Table II**
**Important vector-borne diseases, vertebrate reservoirs and secondary epidemiological cycles: bacteria and parasites**

| Disease, causative agent | Geographical distribution[a] | Vertebrate reservoir[b] | Arthropod vector | Secondary epidemiological cycles – maintenance host(s)[c] | Key references |
|---|---|---|---|---|---|
| **Bacteria** | | | | | |
| Ehrlichiosis (*Ehrlichia ruminantium*) | Africa, Caribbean | Wild ungulates, rodents, carnivores, chelonians, gallinaceous birds | Ticks, *Stomoxys* flies | Humans, dogs, horses, cattle, sheep, goats | 26, 27 |
| Anaplasmosis (*Anaplasma* spp.) | Africa, Eurasia | Rodents, raccoon, artiodactyls | Ticks, *Stomoxys* biting flies | Cattle, horses, dogs | 28 |
| Bovine petechial fever (*Cytocoetes ondiri*) | Africa | Bushbuck *(Tragelaphus scriptus)* | Ticks | Cattle | 29 |
| Plague (*Yersinia pestis*) | Global | Rodents | Fleas | Humans, other mammals | 30 |
| Lyme disease (*Borrelia* spp.) | Global | Rodents, deer, opossum | *Ixodes* ticks | Dogs, horses, cattle | 31 |
| Tularemia (*Francisella tularensis palaearctica*) | Eurasia, North America | Rodents, lagomorphs, rodents | Ticks, biting flies, mosquitoes | Sheep | 32 |
| **Parasites** | | | | | |
| Trypanosomosis (*Trypanosoma* spp.) | Africa, South America | Wild ungulates, small mammals, marsupials | Tsetse fly, triatomine bugs, *Stomoxys* flies | Humans, cattle, horses, pigs, sheep, goats, dogs | 33 |
| Theileriosis (*Theileria parva lawrenci* & *T. equi*) | Africa | Buffalo, zebra | Ticks | Cattle, horses | 34, 27 |
| Leishmaniosis (Visceral *Leishmania infantum,* Cutaneous *Leishmania* spp.) | Global | Wildlife | Sandflies | Humans, primates, dogs, other domestic animals | 35 |
| Dirofiliriosis (*Dirofilaria* spp.) | | Mammals | *Aedes* & *Culex* mosquitoes | Humans | 36 |

a) Including recent and historical distribution
b) Natural host or source ancestral host
c) Clinically affected/mortality reported, maintenance and transmission hosts

of wildlife hosts (43). Re-infection of cattle is a particular problem when cattle encroach on wildlife habitat or protected areas where the disease is enzootic. Chagas disease caused by *T. cruzi* in humans in South America is also sensitive to ecological and environmental change. Destruction of habitat for agriculture and urban development and expansion of peridomestic wildlife species (opossum *Didelphis aurita*) at the expense of small mammal diversity appears to be at the root of disease emergence (44).

## Human population expansion, landscape change, deforestation, urbanisation, opening up of trade, transport systems and the translocation of pathogens

The dominant global socioeconomic model is now based on neoliberalism and free trade. It is driving urbanisation, underpinning the growth of human and domestic animal populations, and increasing global movements of people,

plants, animals, products and machines, with arthropods and microbiota as inadvertent passengers. Development, accelerated by this economic philosophy, has also driven rapid landscape and ecosystem change. Deforestation was recognised early on as a primary driver of VBD emergence (44, 45) and these and other changing structural ecologies have been fundamental to the recent emergence of VBDs (6, 46, 47, 48, 49). Some of these changes are quite subtle; for example, the increase in waste, particularly plastic in the environment, which has provided mosquitoes with novel breeding habitats and resulted in emergence of diseases such as dengue fever (18).

Changing socioeconomic conditions can have dramatic effects on VBD transmission in times of austerity and of relative wealth. For example, tick-borne encephalitis virus and Lyme disease have been shown to increase with increasing wealth associated with increased leisure and back-to-nature activities in tick-infested habitats, exacerbated by increasing secondary hosts as land is set aside for recreation and wildlife conservation. Equally, tick-borne encephalitis has been associated with poorer communities where, as income drops, there is increasing foraging for supplementary food supplies on natural land, thus increasing exposure to ticks (50).

The specific mechanisms for disease introduction are varied, ranging from accidental translocation to the artificial expansion of habitats and breeding sites for vectors. A good example of virus adaptation to changing human ecologies is that of the alphaviruses, which include chikungunya and dengue fever viruses. One of the most important mechanisms of introduction is human population expansion. Where this has occurred adjacent to forest ecosystems where enzootic sylvatic cycles involving non-human primates and other wildlife exist, humans have acted as naïve secondary hosts, often infected through bridge vectors, subsequently amplifying the viruses. The cycle persists while human–mosquito–human transmission is possible but largely burns out after the host population develops immunity or seasonal factors reduce vector abundance. This cycle most probably led to the dispersal of chikungunya virus through human and vector movements on land and sea, a theory now supported by evolving molecular science (51), and apparently driven by virus envelope mutations enabling greater infectivity (21). Nevertheless, the regional picture remained fairly distinct, suggesting locally driven enzootic cycles. Since 2005 this situation has significantly changed and there has been a global spread of Asian strains of virus (52). Two main vectors, *Aedes aegyptii* and *A. albopictus*, have undergone progressive domestication, which has further enabled this expansion (53). The spread of *A. albopictus* out of Asia to the rest of the world was most probably through the timber and rubber (tyre) trade. The relatively sudden geographical expansion of outbreaks of chikungunya was predicated on the introduction of this anthropophilic, highly active, long-lived mosquito

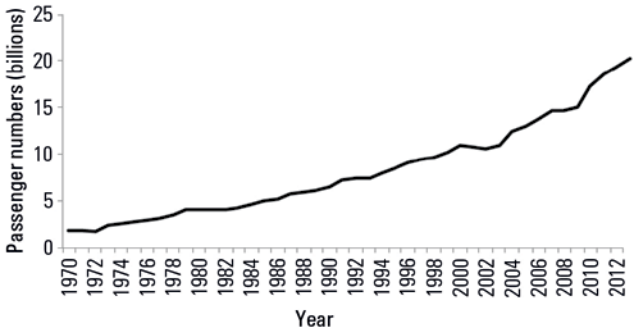


**Fig. 2**
**Global air passenger numbers, 1970–2013**
*Source*: Adapted from World Bank statistics (54)

into urban environments, where water receptacles and waste plastic provided abundant breeding sites. These mosquitoes shifted easily from zoophagy to anthropophagy, which led to persistent cycles in human communities, no longer requiring wildlife reservoirs. The recent trend is for autochthonous outbreaks, and now that suitable vectors in urban environments are abundant the only other factor determining spread is the host. The driver for the recent upsurge in cases has most probably been the dramatic increase in numbers of air passengers and movements over the past decade, with an increase of ten billion (50% of the overall increase over the past 14 years) (Fig. 2). This rapid movement of people is enabling the pathogen infection cycles of VBDs to become transcontinental. A specific example occurred in Europe when a visitor from India who flew in infected with chikungunya virus became a source of virus for an epidemic (55, 56). Optimal conditions for autochthonous outbreaks occur in southern Europe, South and South-East Asia, Oceania, islands in the Indian Ocean and in Central America. The map of chikungunya is constantly being redrawn.

The introduction of West Nile virus to the Americas is now a classic case study in the impact of globalisation on disease (16). This disease was named for its historical geography but now is no longer confined. The spillover did not require introduction of a vector and the virus rapidly adapted to novel hosts and vectors. This event might have been the result of a single transportation of infected animals: the virus is believed to have been introduced through a shipment into New York of animals, perhaps horses, carrying a virus with phylogeny similar to that of viruses from Europe and Israel (57). Once present, the virus adapted to an enzootic bird cycle through local ornithophilic vectors and bridge vectors to initiate secondary epidemiological cycles in a variety of vertebrate hosts in North America. This occurred with remarkable speed, first detected in New York Zoo in 1999 and now widespread across the continent. Over 70 arthropod species belonging to ten different genera are reported as vectors in the United States. The virus is

now well established, maintained and transmitted, with perhaps unexpected dominance of certain hosts such as the American Robin in the transmission cycle. This bird species is highly adapted to the urban landscape and is an effective bridge species that is preferred by *Culex* spp. to the more abundant sparrows and crows (58).

Wildlife trade and translocation or introduction (deliberate or accidental) is another source of pathogen movement (59), and where vectors for particular pathogens are present in the release site a new species introduction can spark secondary epidemiological cycles and disease emergence as the new population establishes. Examples include the introduction of African horse sickness to Spain by zebra (*Equus zebra*) from Africa and the introduction of muskrats (*Ondatra zibethicus*) to Russia in the 1930s, which led to the spread of tularemia among the muskrats, contracted initially from local voles (*Arvicola amphibious*); the presence of the disease in the expanding population of muskrats led to zoonoses among trappers (60).

Expansion of host populations is another driver of VBD emergence. Crimean-Congo haemorrhagic fever is an example of a disease that was initially relatively rare and recognised only in Central Asia (Crimea) and Congo, but over time was confirmed throughout Eurasia and Africa (61, 62). The virus co-evolved with the *Hyalomma* tick and the disease appears confined to areas where this genus is present; it has become an increasing problem as more hosts become available through population explosion. The virus has a wide range of mammal hosts and, although birds are generally considered refractory, interesting secondary epidemiological cycles occur, e.g. in the farmed ostrich of South Africa (23). It has been speculated that migratory birds introduce the virus into Europe by serving as mechanical vectors of infected ticks (63, 64). There is increasing evidence to support this view, with bird ticks in Turkey testing positive for some virus genotypes associated with local human infections (65).

## Climate change boosting vector-borne diseases

A range of mechanisms have been considered in relation to the role of climate change in emergence of VBDs (66). The most obvious mechanism is increasing climate variability resulting in changing wet and dry climate cycles. In drought, vectors (and their insect predators) are usually suppressed as breeding sites dwindle, but where flood cycles follow these conditions exacerbate (sometimes preferentially) vector emergence, especially where their life cycles are shorter than those of their predators (1). If these climate cycles are amplified, or become more or less frequent, they can alter the enzootic character of a region and this can lead to more epidemic disease. Epidemics of Rift Valley fever are often cited as an example of climate playing a pivotal role (25). In Africa, a series of dry years followed by extreme wet conditions associated with El Niño oscillations enables an explosive increase in hatching of the highly dry-tolerant eggs of *Aedes* mosquito vectors. The eggs harbour the virus (transovarial transmission cycle), hatch synchronously under optimal conditions, and a myriad of mosquitoes then feed on all available animal life, many of which are naïve hosts, thus ensuring rapid amplification and spread of the virus. These explosive events result in sometimes massive and highly fatal epidemics affecting a range of species, including humans. Where cycles are predictable, early warning is possible, but with climate change, temporal and geographical variability will most probably result in less predictable emergence across wider geographical zones. There is some evidence for this in the apparent increase of outbreaks in southern Africa (67) and data on inter-epidemic cycles in wildlife hosts of the virus are enriching the debate on the epidemiology of Rift Valley fever in a changing African landscape (67, 68, 69). The effects of drought need not be restricted to natural bodies of water: chikungunya outbreaks in East Africa during dry periods have been associated with an increase in the number of water storage containers; secondary breeding sites then establish close to human habitation and lead to greater infection risk (1).

Latitudinal and altitudinal changes in mean annual ambient temperatures are considered to be another driver for VBD emergence. A contemporary example is the spread of bluetongue virus northwards into Europe (70). Although the exact cause for some of the outbreaks is unclear, the primary drivers appear to be a general increase in bluetongue virus types detected in southern Europe (the point source) (71), invasion of northern Europe by the biting midge *Culicoides imicola* and coincident apparent adaptation of the virus to northern European midge vectors (72). Emergence of Schmallenberg virus in Germany from an unknown source is another intriguing example, and the spread of this virus across Europe, including secondary epidemiological cycles in wild cervidae (73), reflects the growing importance of arboviral disease (74).

A number of diseases in temperate climates are showing a trend of increasing prevalence in northern latitudes and higher altitudes, suggesting climate effects or at least a shift in vector distributions, host preference and/or human exposure rates, e.g. tick-borne encephalitis where the vectors are certain species of *Ixodes* ticks (75). In addition to climate, a number of other mechanisms are at play, including changing enzootic host populations (76), as seen with the decline in roe deer and the increasing importance of rodents in Sweden (77). Clearly, in addition to the increasing human population and use of infected habitats, extended vegetation periods are important, with tick vectors questing for hosts earlier and for longer. There is also increasing evidence of the ability of vectors to overwinter in leaf litter (77) and to practise endophagic feeding behaviour in warmer housing, as shown with *Culicoides* and bluetongue/Schmallenberg viruses (78, 79, 80). Further evidence for

this geographical shift in disease prevalence associated with climate is the northward spread of infection with *Dirofilaria*. This mosquito-borne parasite of wild carnivores, which also causes heartworm disease in the domestic dog population and is zoonotic, is apparently spreading from historically endemic regions in southern Europe (81) to more northern latitudes such as France and Germany (82). It is particularly prevalent in central and eastern European countries, where it is now considered an endemic disease (83). In Russia, species of *Dirofilaria* are now present up to 58ºN (84) and are causing zoonotic infection, especially in handlers of working dogs (85). Similarly, the northward spread of *Leishmania infantum* has been documented in Italy (86) and evidenced by increased seroprevalence in dogs in the French Pyrenees and Spain (87, 88).

### Vector-borne diseases spreading independently of vectors

Although considered a vector-borne disease in Africa, African swine fever was transported in recent years to Eurasia, most probably via food waste or pigs and pig products, and has survived and spread through direct transmission between domestic pigs or via fomites (48). The disease has persisted without relying on an enzootic sylvatic cycle or apparently the tick vector (8) and is now reported within the European Union (Poland, Sardinia) (89). On occasions, there is spillover infection in wild boar, which results in a secondary epidemiological cycle; however, in this case, it appears limited in extent and duration, apparently because of the high virulence in this species (90).

### Domestic animals as reservoirs for vector-borne diseases

Perhaps the best illustration of the importance of a domestic reservoir is with zoonotic visceral leishmaniosis in South America. Here, the domestic dog is now considered the main reservoir host for *Leishmania infantum*, the primary causative agent of zoonotic visceral leishmaniosis in domestic landscapes (2). A variety of natural hosts exist and some of these are synanthropic in domestic landscapes, but it is the domestic dog that has provided the main transmission bridge to humans and appears to be the only true reservoir.

## Discussion: priority actions for reducing the risk of secondary epidemiological cycles and emergence of vector-borne diseases?

Prevention, control or elimination strategies for VBDs, many of which are arising as the result of secondary epidemiological cycles, are urgently needed. Such diseases, affecting humans, domestic animals and the livestock economy, require a thorough understanding of the disease ecology in each case. In particular, there is a need for a comprehensive understanding of the enzootic cycles, reservoir host(s), the vectors involved and the drivers of transmission in the domestic landscape. Currently, the biology is perhaps better understood than the socioecology of these diseases, given that their emergence appears to be largely driven by human activities and impacts on the landscape and ecosystems. Some examples have been described above to give a more ecological than biological focus to this review.

Technical and therapeutical solutions exist for many VBDs, but it is the implementation of these solutions in a global context that has proven most challenging. In many cases the most progress has been made when there is a focus on the vector or where the reservoir host or bridge species for human infection is unique and controllable (domestic). For example, for malaria, the use of insecticides and insecticidal treatment of nets, supported by massive funding, has reduced the burden of disease significantly in Africa and Asia. The control of leishmaniosis in dogs in South America has dramatically reduced *L. infantum* infection rates in humans. On the other hand, decades of effort to control trypanosomosis have seen little reduction in overall tsetse occupancy or disease risk, although it is recognised that total elimination of tsetse is possible in some situations. Recently, targeted chemical control of the tsetse fly, together with appropriate prophylaxis and treatment regimens for livestock and people, has reduced the burden considerably but at a high cost. Resources remain limiting and whether malaria and trypanosomosis will be resolved in any permanent sense will depend on global public good priorities. The resource issue is further complicated by the emergence of new and equally significant VBDs such as dengue fever, chikungunya, West Nile fever and others described above.

It appears unlikely that technical or biological solutions are the answer to the current challenges, although these interventions will remain important for control and mitigation. Long-term solutions lie in modifications to human behaviour, social and political economics and more careful attention to the landscape and human–domestic animal–environment interactions or socioecology. The descriptions given above show how, for example, preventing climate change and rising global temperatures would significantly reduce expansion of VBD distribution into temperate zones, thereby mitigating the risk to millions of humans and domestic animals. Prevention of deforestation and better management of agricultural development and the agriculture–human–environment interface could also dramatically alter the current emergence trajectory. Lastly, urgent consideration of the

effects of globalisation, currently facilitating translocation of vectors, hosts, pathogens and disease is warranted. Increasing VBDs might be an acceptable trade-off for the economic benefits of open markets and resultant monetary wealth, but more accounting is needed to evaluate the risks and costs to human society while alternative, more resilient and sustainable systems are explored. Solutions might well now lie in health professionals stepping outside their domains and engaging with the wider scientific and political communities to find answers to these seemingly intractable and emerging disease challenges.

## Les réservoirs vertébrés et les cycles épidémiologiques secondaires des maladies à transmission vectorielle

R.A. Kock

**Résumé**
L'auteur fait l'inventaire des maladies à transmission vectorielle importantes pour la santé humaine et la santé des animaux domestiques et attire l'attention sur les syndromes émergents, de plus en plus nombreux, ainsi que sur les nouveaux cycles épidémiologiques et les changements de distribution. Ces maladies sollicitent un grand nombre de vecteurs et d'hôtes qui souvent interagissent avec un seul agent pathogène ; elles font aussi intervenir des cycles naturels d'enzootie, des réservoirs sauvages et des cycles épidémiologiques secondaires, qui parfois affectent l'être humain et les animaux domestiques. Dans certains cas, le principal réservoir se trouve dans l'environnement domestique. Les cycles secondaires sont principalement liés aux activités humaines et à leur impact, de sorte qu'il convient de prêter une grande attention aux aspects socio-écologiques des maladies lors de la conception des mesures de prévention et de contrôle. Il existe des solutions techniques et thérapeutiques, mais la réussite des stratégies de contrôle passe par une bonne connaissance des principaux vertébrés jouant le rôle d'hôtes et de réservoirs ainsi que des vecteurs eux-mêmes. Les interventions ont généralement pour cible les vecteurs et/ou les cycles épidémiologiques secondaires, mais peuvent aussi viser, dans le cas de l'homme et des animaux domestiques, les hôtes accidentels ou incidents. Il convient de prêter une grande attention à l'influence exercée par l'économie politique sur ces maladies à transmission vectorielle dans la mesure où de nombreux facteurs sont directement liés à la mondialisation, au changement climatique et aux modifications structurelles des écosystèmes. Il est désormais impératif de réduire les risques d'émergence de nouveaux cycles infectieux en améliorant la gestion de l'interface humain–animaux–environnement.

**Mots-clés**
Épidémiologie – Maladie à transmission vectorielle – Maladie infectieuse émergente.

## Reservorios vertebrados y ciclos epidemiológicos secundarios de las enfermedades transmitidas por vectores

R.A. Kock

**Resumen**
Tras ofrecer una relación de las enfermedades transmitidas por vectores que revisten importancia sanitaria y zoosanitaria, el autor destaca la creciente

aparición de síndromes, nuevos ciclos epidemiológicos y áreas de distribución. En este tipo de enfermedades intervienen multitud de vectores y anfitriones, con frecuencia para un mismo patógeno, así como ciclos enzoóticos naturales, reservorios salvajes y ciclos epidemiológicos secundarios que a veces afectan a personas y animales domésticos. En ocasiones el reservorio principal se encuentra en el entorno doméstico. Los factores que desencadenan los ciclos secundarios guardan relación sobre todo con la actividad humana y sus efectos, por lo que el trabajo de prevención y control debe girar básicamente en torno a la socioecología de las enfermedades. Existen soluciones técnicas y terapéuticas, siempre y cuando se tenga un cabal conocimiento de los principales anfitriones o reservorios vertebrados y los vectores más importantes de la enfermedad que se trata de combatir. En general las intervenciones van dirigidas contra el vector y/o el ciclo epidemiológico secundario y, cuando la patología afecta al ser humano o la fauna doméstica, se administra tratamiento a los anfitriones no preferentes (o episódicos). Conviene asimismo tener más en cuenta la importancia de la economía política en relación con estas enfermedades, pues muchos de los principales factores que las propician son resultado de la mundialización, el cambio climático y alteraciones ecológicas estructurales. Por último, urge ocuparse de reducir el riesgo de que surjan nuevos ciclos infecciosos, lo que pasa por una mejor gestión de la interfaz entre personas, animales y medio ambiente.

**Palabras clave**
Enfermedad infecciosa emergente – Enfermedad transmitida por vectores – Epidemiología.

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