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REVIEW ARTICLE

Rodent-borne diseases and their risks for public health

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Abstract
Rodents are the most abundant and diversified order of living mammals in the world. Already since the Middle Ages we know that they can contribute to human disease, as black rats were associated with distribution of plague. However, also in modern times rodents form a threat for public health. In this review article a large number of pathogens that are directly or indirectly transmitted by rodents are described. Moreover, a simplified rodent disease model is discussed.

Keywords: *Rodents; rats; mice; disease; health; pathogens*

Introduction

During the last decades we have seen a rise in human diseases that are associated with small-mammal reservoirs. In 1998, Mills and Childs (1998) outlined steps towards understanding the link between vertebrate host ecology and human disease. A decade has passed since their study, and therefore it is now time to look back and see what are the current scientific issues in rodent-borne diseases and their effects on public health.

The intention of this vast review is to contribute to a better understanding of rodents, rodent reservoirs, their contribution to disease and a quantification of the risks per disease. This information is important for clinicians, researchers and professionals in the field. In Table 1, a summary is provided of the contribution of rodents to transmission of different pathogens. In the text, more information can be found about each of these pathogens.

Rodentia is the most abundant and diversified order of living mammals, representing about 43% of the total number of mammalian species (Huchon et al. 2002; Wilson and Reeder 1993). Its species are distributed on every continent except Antarctica and include many of the most abundant and taxonomically diverse mammals. In many places rodents live in close contact with human populations, their farm animals or pets. In other places, peri-urban rodents provide a nexus between wildlife communities and humans, exposing humans to some zoonoses circulating in these natural ecosystems.

Taking food from our table

Rodents are important competitors globally with humans for food, particularly through the pre-harvest damage they cause to cereals (Stenseth et al. 2003). For example, across Asia, pre-harvest losses of rice range from 5% in Malaysia to 17% in Indonesia. To put this into perspective, a loss of 6% in Asia amounts to enough rice to feed 220 million people, roughly the population of Indonesia, for 1 year. Rat damage is often patchy and family rice plots small, so it is not uncommon for farmers or villagers to lose half of their entire rice crop to rats (Singleton 2003). On a global scale, it was recently estimated that almost 280 million undernourished could additionally benefit if more attention were paid to reducing pre- and post-harvest losses by rodents (Meerburg et al. 2009).

In Southeast Asia, rats are the number one pre-harvest pest in Indonesia and are in the top three pests in

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Vietnam. In the uplands of Laos, Myanmar, Vietnam, and parts of India, rat populations occasionally erupt and cause massive problems (Singleton 2003). In 2007, Mizoram, in northeastern India, and Myanmar experienced such an outbreak. In Mizoram, a previous massive plague in the 1950s led to famine conditions and triggered a change of government.

In many areas, farmers actually abstain from planting a second or third rice crop because of the expectation of severe rodent damage. This "forgone" loss in productivity is rarely taken into account.

Rodents are also seen as pests because of their gnawing habit, which can cause economic losses, spoilage of food and lead to structural damages (Brown et al. 2008). However, rodent presence can also have serious implications for public and veterinary health. Rodents are hazardous, as they can amplify pathogens from the environment and form reservoirs of (zoonotic) disease (Gratz 1994; Webster and Macdonald 1995).

Rodents as transmitters of pathogens to humans

Rodent-borne diseases can be spread via two different pathways (Figure 1).

The first pathway is a direct route. Rodents can spread pathogens to humans, e.g., by biting them or because humans consume food products or water that is contaminated with rodent feces. Moreover, humans can come in contact with surface water that is contaminated with rodent urine (e.g., leptospirosis) or we breathe in germs that are present in rodent excrements (e.g., hantaviruses). Also, rodents are sometimes mentioned in relation to horizontal transmission of pathogens that cause animal diseases, thus causing huge economic damages and image losses for animal husbandry. Beside highly contagious viral pathogens such as classical swine fever, and foot and mouth disease also bacterial infections (e.g., *Mycobacterium avium*) need to be addressed in this respect.

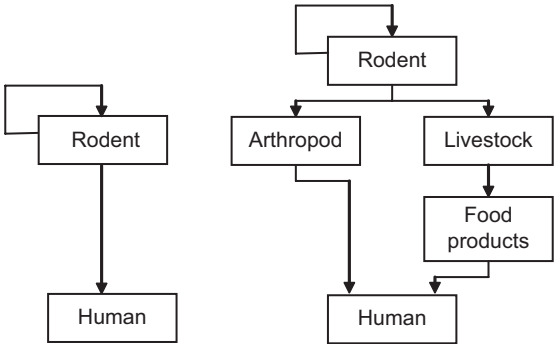

Figure 1. Two different pathogen transmission pathways: on the left the direct route, on the right the indirect route. The pathogen is the arrow in the flowchart.

Rodent-borne pathogens can also be spread indirectly to humans. Then, rodents can serve as amplifying hosts of the pathogens and can bring them into direct contact with humans by mean of ectoparasitic arthropod vectors (ticks, mites, fleas). Rodents that are accidentally or on purpose ingested by livestock can transfer pathogens which can result in human morbidity if these food products are not-thoroughly cooked. Moreover, rodents can help to maintain pathogen transmission cycles in a number of different environments, varying from densely populated urban areas to rural areas and in the wilderness.

The number of different pathogens to whose life cycle rodents contribute in one way or another, is impressive (Singla et al. 2008). The goal of this article is to give a broad overview of the various diseases that humans may acquire from infection with these pathogens, although the authors are aware that the list is non-exhaustive. Table 1 contains an overview of the different diseases in humans, their impact on the human populations, the severity of the disease for human health (measured in its mortality) or the economy (measured in morbidity and production losses). Clearly, rodents can account for huge health and economic losses.

While the numbers of people and animals that are affected are variable in both time and place, the consequences at an individual level can be large. For example, patients hospitalized in the United Kingdom after a rat bite had to stay on average 11.2 days (Hospital Episode Statistics, Department of Health, England, 2002–03).

Moreover, global climate change and changing human settlement patterns (especially in developing countries) could lead to increased problems with rodent-borne pathogens as the distribution of rodent species, arthropods and thus also pathogens linked to these species could be influenced (Githeko et al. 2000).

Below we discuss the different diseases in humans (and sometimes in livestock) that may be the result of pathogen transmission by rodents. An overview of all the diseases that are discussed in this review is provided in Table 1.

Viruses

Hantavirus Pulmonary Syndrome

Hantavirus Pulmonary Syndrome was recognized in 1993 in the South Western parts of the United States as an acute disease caused by several strains of the related strains of viruses in the genus of Hantavirus, *Bunyaviridae* family (Nichol et al. 1993). Most remarkably, the sudden appearance of this rodent-borne virus in the arid US Southwest was accompanied by anomalous weather patterns (Epstein 1995).

Table 1. Overview of different pathogens that may be transmitted by rodents and their consequences.

					Severity	
Disease	Agent	Carrier/Reservoir	Population at-risk	Chance	Human Health	Economy
Hantavirus Pulmonary Syndrome	Virus, Bunyaviridae	Carrier	2	1	3	1
Hemorrhagic Fever with renal syndrome (+ other hemorrhagic fevers)	Virus, Bunyaviridae	Carrier	2	2	2	2
Nephropathia epidemica	Virus, Bunyaviridae	Carrier	1	1	1	1
Crimean-Congo hemorrhagic fever	Virus, Bunyaviridae	Reservoir	1	1	3	1
Borna disease	Virus, Bornaviridae	Reservoir	1	1	1	2
Omsk hemorrhagic fever	Virus, Flaviviridae	Reservoir	1	1	1	1
Kyasanur Forest Disease	Virus, Flaviviridae	Reservoir	1	1	1	1
Apoi Virus Disease	Virus, Flaviviridae	Unknown	Unknown	Unknown	Unknown	Unknown
Tick-borne encephalitis	Virus, Flaviviridae	Reservoir	2	1	3	1
Powassan encephalitis	Virus, Flaviviridae	Reservoir	1	1	1	1
Lymphocytic Choriomeningitis virus (LCMV)	Virus, Arenaviridae	Reservoir	1	1	1	1
Lassa Fever	Virus, Arenaviridae	Carrier	2	2	3	2
South American arenaviruses (Junin, Mapucho etc.)	Virus, Arenaviridae	Carrier	2	2	3	1
North American arenaviruses	Virus, Arenaviridae	Carrier	1	1	Unknown	Unknown
Colorado Tick Fever	Virus, Reoviridae	Reservoir	1	1	1	1
Venezuelan quine encephalitis	Virus, Togaviridae	Reservoir	2	2	2	2
Western equine encephalitis	Virus, Togaviridae	Reservoir	1	1	1	1
Hepatitis E	Virus, Caliciviridae	Reservoir	1	1	1	1
Cowpox	Virus, Poxviridae	Reservoir/ carrier	1	1	1	1
Contagious viral animal diseases (Classical Swine Fever, Foot and Mouth Disease)	Virus, Picornaviridae (FMD); Flaviviridae (CSF)	Reservoir?	0	1	0	3
Leptospirosis (Weils' disease)	Bacteria, Spirochaetes	Carrier	2	2	3	2
Lyme disease	Bacteria, Spirochaetes	Reservoir	3	2	1	2
Tick-borne relapsing fever	Bacteria, Spirochaetes	Reservoir	2	1	1	1
Scrub typhus	Bacteria, Alphaproteobacteria	Reservoir	2	1	3	1
Murine typhus	Bacteria, Alphaproteobacteria	Reservoir	3	1	1	1
Sylvatic epidemic typhus	Bacteria, Alphaproteobacteria	Reservoir	1	1	1	1
Queensland tick typhus or spotted fever	Bacteria, Alphaproteobacteria	Reservoir	1	1	1	1
Rocky Mountain spotted fever	Bacteria, Alphaproteobacteria	Reservoir	1	1	3	1
Rickettsialpox	Bacteria, Alphaproteobacteria	Reservoir	2	1	0	1
Bartonella Illnesses	Bacteria, Alphaproteobacteria	Reservoir	2	2	1	1

Table 1. Continued on next page.

Table 1. Continued.

Disease	Agent	Carrier/Reservoir	Population at-risk	Chance	Severity	
					Human Health	Economy
Human granulocytic anaplasmosis	Bacteria, Alphaproteobacteria	Reservoir	2	1	1	1
Q-fever	Bacteria, Gammaproteobacteria	Reservoir	3	2	3	2
Salmonellosis	Bacteria, Gammaproteobacteria	Carrier	3	1	1	3
Tularemia	Bacteria, Gammaproteobacteria	Carrier	2	1	3	1
E. coli 0157/VTEC	Bacteria, Gammaproteobacteria	Carrier	2	1	3	2
Plague (*Yersina pestis*)	Bacteria, Gammaproteobacteria	Reservoir	2	2	2	2
Campylobacteriosis	Bacteria, Epsilonproteobacteria	Carrier	3	1	1	3
Rat-bite fever and Haverhill fever	Bacteria, Fusobacteria	Reservoir	2	1	3	1
Listeriosis	Bacteria, Bacilli	Carrier	3	1	3	2
Toxoplasmosis	Parasite, Sporozoea	Reservoir	3	2	2	3
Babesiosis	Parasite, Sporozoea	Reservoir	3	2	1	1
Cryptosporidiosis	Parasite, Sporozoea	Reservoir	3	2	1	3
Chagas disease	Parasite, Zoomastigophorea	Reservoir	3	1	3	2
Leishmaniasis	Parasite, Zoomastigophorea	Reservoir	3	2	3	2
Giardiasis	Parasite, Zoomastigophorea	Reservoir	3	2	1	2
Taeniasis	Parasite, Cestoda	Reservoir	1	1	1	1
Rodentolepiasis	Parasite, Cestoda	Reservoir	1	1	1	1
Echinococcosis	Parasite, Cestoda	Reservoir	2	1	3	1
Schistosomiasis	Parasite, Trematoda	Reservoir	3	2	1	3
Human fasciolosis	Parasite, Trematoda	Reservoir	3	1	1	3
Brachylaimiasis	Parasite, Trematoda	Reservoir	1	1	2	1
Alariasis	Parasite, Trematoda	Reservoir	1	1	0	1
Echinostomiasis	Parasite, Trematoda	Reservoir	1	1	0	1
Trichinosis	Parasite, Nematoda	Reservoir	3	2	1	2
Capillariasis	Parasite, Nematoda	Carrier	3	1	1	1
Angiostrongylosis	Parasite, Nematoda	Reservoir	2	1	3	1
Toxascariasis	Parasite, Nematoda	Carrier	1	2	0	2
Baylisascariasis	Parasite, Nematoda	Carrier	1	2	1	2
Aelurostrongylosis	Parasite, Nematoda	Reservoir	0	0	0	0
Amoebic dysentery	Parasite, Lobosea	Reservoir	3	1	3	1
Neosporosis	Parasite, Conoidasida	Reservoir	0	1	0	2

Reservoir: rodents harbor disease-causing organisms and thus serve as potential sources of disease outbreaks, but always via a vector (tick, sand-fly etc.)
Carrier: rodent that shows no or limited symptoms of a disease but harbors the disease-causing agent and is capable of passing it directly onto humans
Population at-risk: focal = 1, regional = 2, more than 2 continents = 3
Chance: chance of contracting the disease (all pathways, not only via rodents): small chance = 1, moderate chance = 2, high chance = 3
Human health: Mortality without treatment <5%=1, 5 to 10% = 2, >10% = 3. No mortality = 0.
Economy: losses in terms of morbidity combined with other losses (e.g. in animal productivity): small losses = 1, moderate losses = 2, huge losses = 3.

The deer mouse (*Peromyscus maniculatus*) is the primary reservoir of the Sin Nombre Virus (SNV) in Canada, the United States of America and Mexico (Abbott et al. 1999; Boone et al. 1998; Childs et al. 1994; Mills et al. 1998; Otteson et al. 1996; Song et al. 1996). In a recent study from the United States, a large number (1700) of individual deer mice were captured and tested for SNV, revealing an average SNV antibody prevalence of

approximately 11% (Lonner et al. 2008). Also other species of rodents, such as the California mouse (*Peromyscus californicus*), cactus mice (*Peromyscus eremicus*), harvest mice (*Reithrodontomys megalotis*) and California voles (*Microtus californicus*), were found positive when checked for SNV-antibodies (Bennett et al. 1999), although cross-reactions with other viruses may have occurred as these can be encountered in this area (Rowe et al. 1995). The cotton rat (*Sigmodon hispidus*), rice rat (*Oryzomys palustris*), the white-footed mouse (*Peromyscus leucopus*), and the Cloudland deer mouse (*Peromyscus maniculatus nubiterrae*) can be competent hosts for hantaviruses that can cause HPS (e.g., Black Creek Canal virus (BCCV), Bayou virus (BAYV), New York virus (NYV), Monongahela virus (MONV) (Peters and Khan 2002; Rhodes III et al. 2000; Schmaljohn and Hjelle 1997; Song et al. 1996) in North America. In South America, HPS can be caused by the long-tailed pygmy rice rat (*Oligoryzomys longicaudatus*) which can carry the Andes virus (ANDV) in Chile, Uruguay and Argentina (Padula et al. 2004; Torres-Perez et al. 2004), and the vesper mouse (*Calomys laucha*) that is also capable to carry an SNV-like virus (Laguna Negra Virus, LNV) in Brazil, Paraguay (Chu et al. 2003; Yahnke et al. 2001), and some parts of Bolivia (Schmaljohn and Hjelle 1997; Williams et al. 1997; Yahnke et al. 2001). In Bolivia and Argentina, the presence of LNV is also reported in the small-eared pigmy rice rat (*Oligoryzomys microtis*) and the large vesper mouse (*Calomys callosus*) (Carroll et al. 2005; Levis et al. 2004). From Honduras it was reported that the Coues' rice rat (*Oryzomys couesi*) (Milazzo et al. 2006) can carry a Bayou-like virus, for which the name Catacamas virus (CATV) was proposed. In Mexico, Playa de Oro virus (OROV) was detected in both Coues' rice rat (*Oryzomys couesi*) and the Jaliscan cotton rat (*Sigmodon mascotensis*) (Chu et al.). Choclo virus (CHOV) was reported in fulvous pygmy rice rats (*Oligoryzomys fulvescens*) in Panama, while Lechiguanuas virus (LECHV) was reported in the same rodent species in Central Argentina (Maes et al. 2004). Moreover, Rio Mamoré Virus (RMV) was reported in small-eared rice rats (*Oligoryzomys microtis*) in Bolivia and Peru, Bermejo Virus (BRMV) in Chacoan pygmy rice rats (*Oligoryzomys chacoensis*), Oran virus (ORNV) in long-tailed rice-rats (*Oligoryzomys longicaudatus*) in North Western Argentina and Maciel virus (MACV) in dark field mice (*Necromys benefactus*) in Central Argentina (Maes et al. 2004). An overview of all these different locations is provided in Table 2.

Hantavirus pulmonary syndrome (HPS) outbreaks in humans are reported in Paraguay (Williams et al. 1997), Argentina (Padula et al. 1998), Chile (Torres-Perez et al. 2004), and Panama (Bayard et al. 2004). Humans can acquire HPS through inhalation of aerosolized virus particles, rodent bites, or direct contact with rodent droppings or urine. Beside the general public, several groups are at higher risks of contracting the disease: mammalogists, public health workers, rodent trappers, farmers, and military personnel (Childs et al. 1995; Jonsson et al. 2008; Zeits et al. 1997). HPS is characterized by bilateral interstitial pulmonary infiltrates, respiratory compromise usually requiring the administration of supplemental oxygen and clinical symptoms resembling those of acute respiratory distress syndrome (ARDS). HPS can be divided into two phases: a prodromal phase, which usually lasts 3–5 days, and a cardiopulmonary stage marked by diffuse pulmonary edema and hypotension within 2–5 days after the onset of pulmonary symptoms. The rapid progression of interstitial pulmonary edema to alveolar edema, with severe bilateral involvement and the accumulation of pleural effusion, accounts for the 30–40% mortality associated with HPS (Peters et al. 1999). Due to the high morbidity and mortality (fatality rate of 30–40%, CDC, Atlanta, http://www.cdc.gov) and the fact that except from supportive care, no treatment exists for hantavirus infection, hantavirus pulmonary syndrome is one of the most serious diseases that rodents can cause in humans, and its impact may even increase in the future as human-to-human transmission of ANDV was reported from Argentina, causing high mortality (Padula et al. 1998). As mentioned before, some authors (Epstein 1995) have claimed that climatic factors have played an important role in the first emergence of the disease in the USA. According to these authors, the likely scenario in the southwestern United States is that a period of 6 years of drought caused pine nuts and grasshoppers to flourish, thereby nourishing deer mice. Then, driven from underground burrows by floodings caused by heavy rains, an increased population of natural hosts enhanced the chance for the virus to thrive and be passed on (Epstein 1995; Kolivras and Comrie 2004).

A variety of vaccines has been developed using both killed virus and recombinant DNA technology (Custer et al. 2003; Hooper et al. 1999; Hooper et al. 2001; Maes et al. 2004). Naked DNA vaccines, usually based on plasmid DNA, have been demonstrated to be promising vaccine approaches for various viral infections (Maes et al. 2004), such as ANDV, HTNV, PUUV, SEOV, and SNV. Currently, there are no therapeutics against HPS, although several approaches are being explored in order to change this (Jonsson et al. 2008; Maes et al. 2004).

Hemorrhagic fever with renal syndrome

The genus Hantavirus, *Bunyaviridae* family, also contains viruses that can cause hemorrhagic fever with renal syndrome or HFRS (Schmaljohn and Hjelle 1997). This denotes a group of similar illnesses throughout Eurasia and adjacent territories (Scandinavia, China, Russia, Korea, Balkans, Western Europe) (Vapalahti et al. 2003).

Table 2. Overview of genus Hantavirus, Bunyaviridae family in New World rodents that are linked to human disease.

Virus	Rodent	Region
Sin Nombre Virus (SNV)	Deer mouse (*Peromyscus maniculatus*) Cactus mouse (*P. californicus*) Harvest mouse (*Rheitrodontomys megalotis*)	North America
Black Creek Canal Virus (BCCV) Bayou Virus (BAYV) New York Virus (NYV) Monongahela Virus (MONV)	Cotton rat (*Sigmidon hispidus*) Rice rat (*Oryzomys palustris*) White-footed mouse (*P. leucopus*) Cloudland deer mouse (*P. maniculatus*)	North America
Andes Virus (ANDV)	Long-tailed pigmy rice rat (*Oligoryzomys longicaudatus*)	Chile, Uruguay, Argentina
Laguna Negra Virus (LNV)	Vesper mouse (*Calomys laucha*) Small-eared pigmy rice rat (*O. microtis*) Large vesper mouse (*Calomys callosus*)	Brazil, Paraguay, Bolivia
Catacamas Virus (CATV)	Coues' rice rat (*O. couesi*)	Honduras
Playa de Oro Virus (OROV)	Coues' rice rat (*O. couesi*) Jaliscan cotton rat (*S. mascotensis*)	Mexico
Choclo Virus (CHOV)	Fulvous pygmy rice rat (*O. fulvescens*)	Panama
Lechiguanuas Virus (LECHV)	Fulvous pygmy rice rat (*O. fulvescens*)	Central America
Rio Mamoré Virus (RMV)	Small-eared pigmy rice rat (*O. microtis*)	Bolivia, Peru
Bermejo Virus (BRMV)	Chacoan pygmy rice rat (*O. chacoensis*)	Argentina
Oran Virus (ORNV)	Long-tailed pigmy rice rat (*O.longicaudatus*)	Argentina
Maciel Virus (MACV)	Dark field mouse (*Necromys benefactus*)	Argentina

HFRS includes diseases that are alternatively known as Korean hemorrhagic fever, epidemic hemorrhagic fever and nephropathia epidemica (Plyusnin et al. 1999; Schmaljohn and Hjelle 1997). The striped field mouse (*Apodemus agrarius*) distributes the Hantaan virus (HTNV) in China, Russia, and Korea (Lee et al. 1981; Lee et al. 1981). In Central and Eastern Europe (Vapalahti et al. 2003), the yellow-necked field mouse (*Apodemus flavicollis*) and the striped field mouse (*Apodemus agrarius*) distribute two closely related viruses: the Dobrava-Belgrade Virus (DOBV, also described as DOBV-Af or Belgrade virus) and Saaremaa Virus (SAAV, previously described as DOBV-aa). DOBV (carried by *A. flavicollis*) has been associated with severe HFRS, especially in the Balkans (Avsic-Zupanc et al. 2000; Brus et al. 2002; Maes et al. 2004; Sibold et al. 1999; Vapalahti et al. 2003). The Norway rat (*Rattus norvegicus*) (Heyman et al. 2004; Reynes et al. 2003) and the black rat (*Rattus rattus*) (Reynes et al. 2003; Wang et al. 2000) are mentioned as worldwide spreaders of the Seoul virus (SEOV). In Korea, the Soochong (SOO) or Amur Virus (Baek et al. 2006) was found in the Korean field mouse (*Apodemus peninsulae*) and was recently also encountered in the North-Eastern part of China (Jiang et al. 2007).

In Europe, the common vole (*Microtus arvalis*) carries the Tula virus (Heyman et al. 2002; Plyusnin et al. 1994; Vapalahti et al. 2003). In the Balkans, the pine vole (*Pitymys subterraneus*) is also linked with Tula virus (Song et al. 2002), although there it is considered to be a spill-over reservoir (which does not contribute to the spreading of the virus). In Germany, a 43-year-old man became ill with fever, renal syndrome, and pneumonia (Klempa et al. 2003). Typing revealed the presence of neutralizing antibodies against TULV, while TULV genetic material was detected in common voles that were trapped in the neighbourhood of the patients' home. This was the first case of hemorrhagic fever with renal syndrome and pulmonary involvement associated with TULV infection (Klempa et al. 2003). Moreover, there has been some evidence of human infection with TULV after a rodent bite (Schultze et al. 2002). However, evidence that TULV can cause symptoms in humans remains far from proven.

HFRS is characterized by systemic involvement of the capillaries and small vessels, which causes capillary leakage and hemorrhagic manifestations. Renal involvement leading to acute renal dysfunction as a result of interstitial hemorrhage and interstitial infiltrates is also common. After the prodromal period, the clinical course of patients with severe disease can be divided into five phases: febrile, hypotensive, oliguric, diuretic, and convalescent (Peters et al. 1999). Approximately 60.000– 150.000 cases of HFRS involve hospitalization, with the majority (90%) in China, Russia, and Korea. The fatality rates range from 5–10% if HFRS is caused by Hantaan virus (Schmaljohn and Hjelle 1997). The mechanism of transmission to man indicates a principal role for respiratory infection from aerosols of infectious virus from rodent urine, feces, and saliva. Interhuman, secondary spread of infection does not occur (Tsai 1987).

Climate influences the emergence and re-emergence of infectious diseases, which is also happening for HFRS. In a particular study in China the impact of climatic, reservoir and occupational variables on the transmission of HFRS in low-lying parts of the country were assessed using empirical data over the period 1980–1996.

The seasonal amount of precipitation, the density of mice and the level of crop production in autumn could be used as predictors of HFRS transmission in the low-lying area of HFRS foci (Bi et al. 2002).

Ribavirin was tested for efficacy in HFRS patients in China and shown to have a statistically significant beneficial effect if initiated early in the disease course (Huggins et al. 1991; Jonsson et al. 2008). Other compounds, such as 1-β-d-ribofuranosyl-3-ethynyl-[1,2,4]triazole (ETAR) seem also promising (Chung et al. 2008).

In North-Western Europe where HFRS is absent, there are hantavirus types that cause a mild variant of this disease. *Nephropathia epidemica* is a virus-infection caused by the Puumala virus (PUUV). The Puumala virus is spread by the bank vole (*Myodes glareolus,* earlier *Clethrionomys*) in Europe (Sibold et al. 1999), Scandinavia (Olsson et al. 2005), and Russia.(Bernshtein et al. 1999; Lundkvist et al. 1997), while the grey-sided vole (*Myodes rufocanus*) was associated with Puumala virus in Japan (Kariwa et al. 1995). For Puumala, there are also signs that the virus can survive for prolonged times outside the host, thus causing indirect transmission via the environment (Kallio et al. 2006). About 80% of the infected individuals are asymptomatic or develop only mild symptoms, and the disease does not spread from human to human. Mortality rate is below 1%. The incubation period of *Nephropathia epidemica* is between two and six weeks. It has a sudden onset with fever, headache, back pain and gastrointestinal symptoms, but sometimes worse symptoms such as internal hemorrhage can occur and it can even lead to death. The bank vole is the reservoir for the virus, which is contracted from aerosolized droppings. Recently, human cases of *Nephropathia epidemica* due to Puumala virus infection in Europe have increased (Tersago et al. 2008). Following the hypothesis that high reservoir host abundance induces higher transmission rates to humans, explanations for this altered epidemiology must be sought in factors that cause bank vole (*Myodes glareolus*) abundance peaks (Tersago et al. 2008), such as the predator-prey cycles in Northern-Europe (Hanski and Henttonen 1996; Hanski et al. 2001) and mast years with heavy seed crops of oak and beech in Central-Europe (Jensen 1982; Jensen 1985).

Other hantaviruses not yet linked with human disease

Rodents also carry a number of other hantaviruses, although these are not (yet) linked to disease in humans. For example, in Africa, the African wood mouse (*Hylomyscus simus*) carries the Sangassou virus (Klempa et al. 2006), the bandicoot rat (*Bandicota indica*) carries the Thailand virus (Hugot et al. 2006; Pattamadilok et al. 2006), the meadow vole (*Microtus pennsylvanicus*) carries Prospect Hill virus, the reed vole (*Microtus fortis*) the Khabarovsk (KBRV) virus in Russia (Horling et al. 1996) and the western harvest mouse (*Reithrodontomys megalotis*) El Moro Canyon (ELMC) virus in the USA and Mexico (Calisher et al. 2005).

The impact of these hantaviruses on human health is yet unknown. Moreover, there will also be other hantaviruses in circulation that are not yet discovered and hantaviruses are constantly coevoluting with their hosts. Also, new hosts are discovered that are also capable to carry hantaviruses: such as happened with insectivores (Okumura et al. 2007; Song et al. 2007). As a consequence, more research is needed on the presence and distribution of hantaviruses in the world and their potential impact on human health.

Crimean-Congo hemorrhagic fever

Crimean-Congo hemorrhagic fever (CCHF) is a tick-borne hemorrhagic fever with documented person-to-person transmission and a case-fatality rate of between 3-30% (Ergonul 2006). Ticks of the genus *Hyalomma* are responsible for the transmission of the *Crimean-Congo hemorrhagic fever virus*, genus *Nairovirus*, family *Bunyaviridae*. This widespread virus has been found in Africa, Asia, the Middle East, and Eastern Europe. Outbreaks have been reported in many countries, including the former Soviet Union, China, Pakistan, Iraq, Iran, South Africa, Mauritania, Uganda, Burkina Faso, the Democratic Republic of Congo, Kosovo, Albania, Bulgaria, and Turkey (Ergonul 2006). According to this author, over 3400 humans were affected during these outbreaks since the 1940s (Ergonul 2006), mainly agricultural workers. The length of the incubation period for the illness depends on the acquisition pathway. It can vary between 1–9 days (usually between 1–3 days) in case of infection via a tick bite to 5–13 days (but usually between 5–6 days) following contact with infected tissues or blood.

According to the WHO (WHO, Geneva, http://www.who.int), the onset of symptoms is sudden, with fever, myalgia (aching muscles), dizziness, neck pain and stiffness, backache, headache, sore eyes, and photophobia. There may be nausea, vomiting, and sore throat early on, which may be accompanied by diarrhoea and generalised abdominal pain (Ergonul 2006).

Then, the patient can experience sharp mood swings, and may become confused and aggressive. After two to four days, the agitation may be replaced by sleepiness, depression and lassitude, and the abdominal pain may localize to the right upper quadrant, with detectable hepatomegaly (liver enlargement). Other clinical signs which emerge include tachycardia, lymphadenopathy, and a petechial rash, both on internal mucosal surfaces, such as in the mouth and throat, and on the skin. The petechiae may give way to ecchymoses and other

hemorrhagic phenomena such as melena, hematuria, epistaxis, and bleeding from the gums (Swanepoel et al. 1989). There is usually evidence of hepatitis. The severely ill may develop hepatorenal and pulmonary failure after the fifth day of illness (Swanepoel et al. 1989).

Various rodents have been found to carry antibodies to the virus (Nalca and Whitehouse 2007; Shepherd et al. 1987), and can be a host for the immature stages of the tick vectors (Ergonul 2006). Thus, rodents have an important role in the tick-cycle and the conservation of the pathogen. There is no evidence of direct transmission of the pathogen between rodents and humans.

According the fact sheet of Crimean-Congo hemorrhagic fever on the WHO website (www.who.int), general supportive therapy is the mainstay of patient management. Intensive monitoring to guide volume and blood component replacement is required. The antiviral drug ribavirin has been used in treatment of established CCHF infection with apparent benefit (Fisher-Hoch et al. 1995). Both oral and intravenous formulations seem to be effective (Fisher-Hoch et al. 1995; Saijo et al. 2004).

An inactivated mouse brain-derived vaccine against CCHF has been developed and used on a small scale in Eastern Europe (Papa et al. 2002), but there is no safe and effective vaccine widely available for human use. Control with acaricides (chemicals that kill ticks) is the only realistic preventive option.

Borna disease

Borna disease virus (BDV) is an enveloped virus with a negative-stranded non-segmented RNA genome, which has been classified as the prototype virus of a newly established family, *Bornaviridae*, within the order *Mononegavirales* (Staeheli et al. 2000). BDV infections can result in neurological disease that mainly affects horses and sheep in certain areas of Germany, Switzerland, Austria, and the Principality of Liechtenstein (Staeheli et al. 2000). There are also reports from Finland that the Borna virus is prevalent in wild mammals (Kinnunen et al. 2007). The detection of BDV-specific antibodies in psychiatric patients (Bode et al. 1995; de la Torre et al. 1996; Rott et al. 1985) suggests that Borna disease (BD) may be a zoonosis.

Rodents are mentioned as a possible source of virus transmission to farm animals (Durrwald et al. 2006; Sauder and Staeheli 2003). Experimental infection of rodents has resulted in persistence of BDV and is associated with the presence of viral gene products in rodent saliva, urine, and feces (Sauder et al. 1996). Thus, animal feed contaminated with urine of persistently infected rats or other rodents represent a source of infectious BDV (Sauder and Staeheli 2003; Staeheli et al. 2000). The role of rodents in transmission of BDV has also been mentioned in relation to feline infections in Sweden (Berg et al. 1998). As the pathways of human infection remain unclear, more research is needed on this topic.

Omsk hemorrhagic fever

Omsk hemorrhagic fever is a disease that is endemic in Western-Siberia (Gajdusek 1956; Holbrook et al. 2005). It is caused by Omsk hemorrhagic fever virus (OHFV), a member of the genus *Flavivirus* of the family *Flaviviridae* (Li et al. 2004). The virus is transmitted through direct contact of humans with infected animals and bites from infected ticks (*Dermacentor* spp. (Li et al. 2004)). In humans, OHFV is the only known tick-borne flavivirus that always causes hemorrhagic disease in the absence of encephalitis (Lin et al. 2003), although cases are known where related tick-borne flaviviruses such as Kyasanur Forest Disease and Alkhurma viruses also occur without encephalitis. Clinical signs of OHF are fever, headache, myalgia, dehydration, and hemorrhage (Pavri 1989). OHF has a case fatality rate of 0.5–3% (Li et al. 2004). There are no reported cases of person-to-person transmission or nosocomial spread of OHFV.

OHFV is maintained in nature through circulation among ticks and rodents including water voles (*Arvicola terrestris*) and muskrats (*Ondata zibethica*) (Kharitonova and Leonov 1985; Li et al. 2004). Infection of humans is frequently associated with occupation, as muskrat trappers are common victims of OHF.

Kyasanur Forest Disease

Kyasanur Forest Disease (KFD) is caused by a OHFV related tick-borne *Flavivirus* of the family *Flaviviridae* found in forest rodents in south-western India (Solomon and Mallewa 2001), the Kyasanur Forest Disease Virus (KFDV). Rats (*Rattus rattus*) are known hosts for a species of tick (*Haemaphysalis spinigera*) that is the main vector of the pathogen (Saxena 1997), although other tick species are capable of transmitting the disease (Hoogstraal 1966). In humans, KFD can cause hemorrhagic fever (Hoogstraal 1966). Since its discovery in the 1950s, KFDV has caused epidemic outbreaks of hemorrhagic fever annually, affecting 100–500 people with a case fatality rate of 2–10% (Gould and Solomon). However, the endemic area of KFD is rather limited. Recently, a variant of KFDV, characterized serologically and genetically as Alkhurma hemorrhagic fever virus (AHFV), has been identified in Saudi Arabia (Gould and Solomon; Priyabrata 2006) and very recently in China (Wang et al. 2009). Tick control could be a preventive measure for reducing the transmission of KFD to humans (Ghosh et al. 2007). Moreover, a vaccine candidate against KFD has been developed (Dandawate et al. 1994).

Apoi Virus Disease

A relative of Kyasanur Forest Disease Virus, Apoi Virus (APOIV), was isolated from spleens of healthy *Apodemus* mice in Japan (Karabatsos 1995). Infection causes encephalitis in newborn mice. A case of laboratory infection resulted in CNS signs, fever, headache, myalgia and arthralgia with residual leg paralysis (Karabatsos 1995). No vector could be identified. Exact risk for public health remains unknown.

Tick-borne encephalitis

Tick-borne encephalitis virus (TBEV) is a zoonotic arbovirus infection of the tick-borne flavivirus group, family *Flaviviridae*, genus *Flavivirus*. The TBEV species consists of three sub-types, namely Far Eastern, Siberian (previously west-Siberian) and European (previously Central European Encephalitis, CEE virus) (Gritsun et al. 2003). The European lineage is transmitted by the tick *Ixodes ricinus* and both the Far Eastern and Siberian subtypes by the tick *Ixodes persulcatus* (Han et al. 2005).

TBEV is endemic in Russia and Eastern and Central Europe (Dumpis et al. 1999), China (Lu et al. 2008) and in Western Europe and Scandinavia, resulting in 11,000 annual cases of tick-borne encephalitis (TBE) in Russia and 3,000 cases in the rest of Europe (Gritsun et al. 2003).

Several rodents such as the bank vole (*Myodes glareolus*), field mouse (*Apodemus agrarius Pallas*), and red voles (*Myodes rutilus Schreber*) (Bakhvalova et al. 2006; Weidmann et al. 2006) are known hosts of ticks that can cause TBE. Rodents play an essential role in the transmission of the tick-borne encephalitis virus between ticks (Randolph et al. 1999). These natural hosts have neutralizing antibodies to TBEV and no detectable viremia. However, they can still support virus transmission between infected and uninfected ticks feeding closely together on the same animal (Labuda et al. 1997). TBEV is transmitted to humans usually by the bite of a tick (either *Ixodes persulcatus* or *Ixodes ricinus*); occasionally, cases occur following consumption of infected unpasteurized milk (Dumpis et al. 1999).

Transmission is seasonal and occurs in spring, summer or autumn, particularly in rural areas favored by the tick vector: e.g., in forest foci with enhanced moist vegetation where mammals might provide a blood meal for them. TBEV can cause acute central nervous system disease, which may result in death or in 30% of the cases in long-term neuropsychiatric sequelae. TBEV can produce a variety of clinical symptoms: febrile, meningeal, meningoencephalitic, poliomyelitic, polyradiculoneuritic, and chronic (Gritsun et al. 2003). Usually the febrile form is the first infection phase, after which the second phase with neurological manifestations may or may not occur. Incubation time is between 7 and 14 days (Gritsun et al. 2003). Mortality rates vary from 0.5-2% for the European TBEV-genotype, to mortalities between 20-35% or even higher for Far Eastern TBEV-genotypes (Anonymous 2006; Haglund and Gunther 2003; Lu et al. 2008). Effective vaccines against TBE are reported in Europe (Gritsun et al. 2003), but are also available in China and Russia. Some have mentioned that the risk for travelers of acquiring TBEV has increasing with the recent rise in tourism to areas of endemicity during spring and summer (Dumpis et al. 1999), although in some areas the peak of the amount of cases is in the autumn.

Powassan encephalitis

Powassan virus (POW) is a North American tick-borne flavivirus (Hoogstraal 1966; McLean et al. 1970), related to the TBE virus in Eurasia (Gholam et al. 1999). It was first isolated from a patient with encephalitis in 1958. From 1958–1998, 27 human POW encephalitis cases were reported from Canada and the northeastern United States (McLean et al. 1970; Ralph 1999). From September 1999–July 2001, four Maine and Vermont residents with encephalitis were found to be infected. The incubation period is on average 7–14 days. It is difficult to distinguish the symptoms of Powassan encephalitis from those caused by the herpes simplex virus (Ralph 1999). Moreover, it is likely that not every infection leads to disease: serologic surveillance studies in Canadian communities showed positive tests in up to 3% of the population, suggesting that infection without encephalitis can occur in humans (McLean et al. 1962).

The vector of POW is the woodchuck tick (*Ixodes cookei*), which is hosted by a variety of rodents (marmots, sciurid rodents, groundhogs) (Hardy et al. 1974; Hoogstraal 1966), but also weasels, skunks, raccoons, coyotes, and foxes (Johnson 1987; Main et al. 1979; Ralph 1999). Moreover, the scope of transmission of the virus may be broadened by domestic cats and dogs, which can act as harbingers of infected ticks and thereby expose humans (Ralph 1999).

Transmission of the pathogen to humans can take place by bites from an infected tick or mite (Ralph 1999) or by consumption of food products from infected animals (e.g., raw milk) (Woodall and Roz 1977). As rodents play a role in the life cycle of the tick, Powassan encephalitis was incorporated in this review, although it is difficult to quantify the exact contribution of rodents to human morbidity and mortality.

Lymphocytic choriomeningitis

Lymphocytic choriomeningitis virus (LCMV) is a rodent-borne virus belonging to the family *Arenaviridae*, genus *Arenavirus.* LCMV can cause a variety of human

diseases which are known as lymphocytic choriomeningitis (LCM), ranging in severity from flu-like illness to meningitis and encephalitis (Lourdes Lledó 2003). Moreover, intrauterine infections also occur and can result in chorioretinitis, hydrocephalus, microcephaly or macrocephaly, mental retardation, and fetal death (Barton and Hyndman 2000; Jahrling and Peters 1992; Mets and Chhabra 2008; Rawlinson et al. 2008). Since 1955, 54 cases of congenital LCMV have been reported, with 34 of the cases diagnosed since 1993 (Jamieson et al. 2006).

Infections have been reported in the Americas, Europe, Australia, and Japan. Seroprevalence studies in humans have shown that the prevalence of LCMV in humans lies between 2–5%, which suggest that many cases are clinically inapparent. The disease is contracted by humans through breathing air that is contaminated with rodent excrements, especially from the domestic house mouse *Mus domesticus* (Childs et al. 1991; Childs et al. 1992). A case-report from France in which LCMV was detected in 14 out of 20 mice trapped at a patient's home reports that patient and mouse LCMVs were identical (Emonet et al. 2007). However, this LCMV strain was highly divergent from previously characterized LCMV (Emonet et al. 2007). Also, laboratory animals (Dykewicz et al. 1992) and pet rodents (Amman et al. 2007) can transfer the disease to humans.

Infected wild rodents can be encountered in several areas of the world, including Argentina (Laura Riera 2005), the USA (Childs et al. 1992), and Spain (Lourdes Lledó 2003). Currently, there is no specific treatment available against LCM, which is especially problematic in pregnant women. As ribivarin works against LCMV in cell culture, this is recommended under specific circumstances, such as presentations resembling hemorrhagic fever or in immunosuppressed cancer patients (Peters 1994). The great majority of LCMV-infected patients survive without residua and the mortality rate is about 1% (Jahrling and Peters 1992). Interestingly, there have been recent reports of human to human transmission through organ transplants that have led to severe complications for recipients of organs carrying LCMV (Anonymous 2008; Fischer et al. 2006; Palacios et al. 2008).

Lassa fever

Lassa fever is an acute viral illness that occurs endemically in West Africa and a significant cause of mortality and morbidity. The disease is caused by a single-stranded RNA virus (Lassa Virus, LASV) belonging to the virus family *Arenaviridae*. The disease was isolated in the multimammate rat (*Mastomys natalensis*) (Fichet-Calvet et al. 2008; Green et al. 1978; Johnson et al. 1981; Meulen et al. 1996; Monath et al. 1974), a rodent with a peridomestic dispersal pattern. Humans acquire the infection by breathing air that is contaminated with rodent excrements, by direct contact with rodent droppings or urine, by consumption of food that is contaminated by rodents, by bite wounds, or by close contact with other humans that have contracted the disease.

The number of LASV infections per year in West Africa is crudely estimated at 100,000 to 300,000, with approximately 5,000 deaths (Khan et al. 2008). In some areas of Sierra Leone and Liberia, it is known that 10–16% of people admitted to hospitals have Lassa fever, which indicates the serious impact of the disease on the population of this region (Birmingham and Kenyon 2001). Fatality rate lies between 15–20%. Lassa fever occurs in all age groups and in both men and women, but death rates are highest for pregnant women in the third trimester of their pregnancy and unborn children in uteri of infected mothers. Lassa fever begins after an incubation period of 7–18 days, with fever, weakness, malaise, and severe headache and throat ache (Fisher-Hoch and McCormick 2001). Up to a third of the hospitalized patients progress to a prostrating illness 6–8 days after onset of fever, with persistent vomiting and diarrhea (Fisher-Hoch and McCormick 2001). Bleeding is only seen in 15–20% of the patients, primarily affecting the mucosal surfaces (Fisher-Hoch and McCormick 2001).

Persons at greatest risk of contracting the virus are those living in rural areas where *Mastomys* are usually encountered, especially in areas of poor sanitation or crowded living conditions. Estimates of antibody prevalence range from 4–6% in Guinea to 15–20% in Nigeria, though in some villages in Sierra Leone as many as 60% of the population have evidence of past infection (Fisher-Hoch and McCormick 2001). Health care workers are at risk if proper barrier nursing and infection control practices are not maintained (Fisher-Hoch et al. 1985).Ribivarin has been successfully used in Lassa Fever patients in combination with supportive care, but also new anti-arenavirus drugs are still being developed as fatalities still occur even while ribivarin is used (Khan et al. 2008). A number of groups are currently developing a vaccine for Lassa fever (Fisher-Hoch et al. 2000; Fisher-Hoch and McCormick 2001; Fisher-Hoch and McCormick 2004; Geisbert et al. 2005).

Other

Beside Lassa virus (LASV) and lymphocytic choriomeningitis virus (LCMV) the lymphocytic choriomeningitis-Lassa (Old World) complex also includes the Ippy virus (IPPV), the Mobala virus (MOBV), and the Mopeia virus (MOPV); viruses that have not yet been associated with human morbidity. IPPV was found in *Arvicanthus spp*. (e.g., Nile grass rats) in Central Africa, MOBV was reported from soft-furred rats (*Praomys spp.*) in the

Central African Republic, while MOPV is present in multimammate mice, *M. natalensis* in South Africa. Recently, also a novel "killer" arenavirus was found in South Africa, which was directly associated with rodent presence (Zeller et al. 2008).

South American hemorrhagic fevers

Of all 15 New World (North and South American) arenaviruses only five South American arenaviruses can cause hemorrhagic fevers. These are Bolivian Hemorrhagic Fever (caused by Machupo virus, MACV), Argentinean Hemorrhagic Fever (caused by Junin virus, JUNV), Venezuelan Hemorrhagic Fever (caused by Guanarito virus, GTOV), Tacaribe fever (caused by Tacaribe virus (Salazar-Bravo et al. 2002), TACV), and Sabia Hemorrhagic Fever (Sabia virus, SABV) in Brazil (Gonzalez et al. 1996). Transmission of four of these South American hemorrhagic fever viruses (MACV, JUNV, GTOV, SABV) are associated with a primary rodent reservoir, and their transmission to humans is believed to involve mechanisms similar to human infection with hantaviruses in North-America (Doyle et al. 1998). Comparisons of arenavirus phylogeny with rodent host phylogeny and taxonomic relationships provide several examples in which virus–host cospeciation is potentially occurring (Bowen et al. 1997): this means that host and pathogen have adapted to each other. Both arena- and hantaviruses have coevolved with their rodent hosts.

In Argentina, JUNV was encountered for the first time in 1958 in the small vesper mouse (*Calomys laucha*) and the corn mouse (*Calomys musculinus*) (Doyle et al. 1998; Mills et al. 1991; Mills et al. 1992). In another study, it was found that seropositive *C. musculinus* were predominantly males in the oldest age and heaviest body mass classes, and that these animals were twice as likely to have body scars as seronegative males. This suggests that most infections were acquired through horizontal transmission among dominant males through scarring and biting (Mills et al. 1994). Other rodent species that can be affected are the grass field mouse (Akodon azarae) and the dark field mouse (Bolomys obscurus). As all of these rodents mainly live in grasslands, cultivated fields and hedgerows, the highest chance for contact of humans with these animals is during field work. Human activity (e.g., changes in land use) can have effects on the number of cases. The expension of maize agriculture has favored the growth of corn mouse populations and there has been a correlated increase in human infections with JUNV (De Villafañe et al. 1977).

In Bolivia, in 1963 the first evidence for existence of a potential non-human reservoir of MACV infection was found (Johnson et al. 1965). Virus strains were recovered from tissue samples of the wild vesper mouse *Calomys callosus* captured in the area of San Joaquín, Bolivia (Johnson et al. 1965). This rodent has a peridomestic lifestyle, with its main habitat in grasslands. Human contact with this animal takes place primarily in houses.

Venezuelan hemorrhagic fever (VHF) was initially recognized as a distinct clinical entity in 1989 and the etiologic agent, GTOV, was isolated and identified as a novel arenavirus in 1991 (Weaver et al. 2000). Epidemiologic field studies in the VHF-endemic region of western Venezuela indicated that two grassland rodent species, the cane mouse (*Zygodontomys brevicauda*) and the cotton rat (*Sigmodon alstoni*), are natural hosts of the virus (Fulhorst et al. 1999; Tesh et al. 1993). The main habitat of this rodent is in brushes and grasslands, and it is thought that most people acquire infection when working there (De Manzione et al. 1997). For SABV that was first isolated in 1990, no wild rodent reservoir is known yet, although the disease was associated with several human cases (including a laboratory worker in Connecticut) (Doyle et al. 1998). For TACV, the wild reservoir consists of bats of the genus *Artibeus*.

After an incubation period of 7–14 days, all South American Arenavirus Hemorrhagic Fevers (AHF) begin insidiously with progressive development of fever, chills, malaise, anorexia, myalgia and sore throat. As the disease progresses, patients develop weakness, arthralgia, back pain, nausea, vomiting, epigastric pain, dizziness, conjunctivitis, flushed face, and bleeding gums. By the 6th or 7th day, the patients are usually acutely ill with dehydration, disorientation and frequently hemorrhagic and/or neurological manifestations (Tesh 2002). If death occurs, it usually results from massive hemorrhage and/or shock. If the patient survives this period, improvement begins about the 10th or 12th day and a slow convalescence ensues The mortality rates in patients with untreated AHF range from about 10 to 33% (Tesh 2002). Mortality rates of AHF decrease with early hospitalization and intensive supportive care, including immune human plasma or the antiviral drug ribavirin (Tesh 2002). Moreover, an effective vaccine against AHF is available (Enria and Maiztegui 1994; Enria and Barrera Oro 2002).

Beside viruses that can cause hemorrhagic fevers, rodents are also linked to a number of arenaviruses that have not (yet) been associated with human disease. Examples are the Pirital virus (PIRV), which was recently isolated from the cane mouse (*Zygodontomys brevicauda*) and the cotton rat (*Sigmodon alstoni*) in Venezuela (Fulhorst et al. 1999; Weaver et al. 2000), the Amapari (AMAV) virus that was encountered in the rice rat (*Oryzomys goeldii*) and the bristly mouse (*Neacomys guianae*) in the tropical forests of Brazil, the Oliveros virus (OLIV) of Northern Argentina that was found in the dark bolo mouse (*Bolomys obscurus*), the Parana virus (PARV) in the Paraguayan rice rat (*Oryzomys buccinatus*), the Latino virus (LATV) in the

large vesper mouse (*Callomys callosus*) in Bolivia, the Flexal virus (FLEV) in Brazil (*Oryzomys spp*.), Pichindé (PICV) virus in the Tomes's rice rat (*Oryzomys albigularis*) in Colombia (Peters 1998) and Allpahuayo virus (ALLV) in arboreal rice rats (*Oecomys bicolor, Oecomys paricola*) in the Peruvian Amazon (Moncayo et al. 2001). It is unknown whether all of these arenaviruses have the potential to cause human disease under natural conditions.

North American arenaviral diseases

Until the mid 1990s the sole arenavirus that was recognized in North America was the Tamiami virus (TAMV), which was observed in the cotton rat *Sigmodon hispidus* in Southern Florida (Calisher et al. 1970). From 1996 onwards the Whitewater Arroyo virus (WWAV) was encountered in the white-throated woodrat (*Neotoma albigula*) in New Mexico (Calisher et al. 2001; Fulhorst et al. 1996). Later, antibodies to the virus were also found in southern plains woodrats (*Neotoma micropus*) in Texas (Fulhorst et al. 2002) and in the Riparian wood rat (*Neotoma fuscipes*), the desert wood rat (*Neotoma lepida*), the brush mouse (*Peromyscus boylii*), the California mouse (*Peromyscus californicus*), the cactus mouse (*Peromyscus eremicus*), the deer mouse (*Peromyscus maniculatus*), and the Western harvest mouse (*Reithrodontomys megalotis*) in Southern California (Bennett et al. 2000).

While TAMV does not seem to have implications for human health, the WWAV was linked to the death of a 14-year-old girl in Northern California and two other fatal cases in the same region (Anonymous 2000; Enserink 2000). These patients presented with febrile illness and respiratory distress, with two developing hemorrhagic symptoms and liver failure (Reignier et al. 2008). It was thought that human infection might occur when humans inhale aerosolized rat urine (Anonymous 2000; Enserink 2000). However, only one victim reported a possible contact with rodent droppings before becoming ill, and no subsequent reports have appeared confirming the association of WWAV with these or any other human infections (Reignier et al. 2008).

In 2002, a new arenavirus in Northern America was discovered: the Bear Canyon virus or BCNV (Fulhorst et al. 2002). This virus was first found in California in the California mouse (*Peromyscus californicus*) (Fulhorst et al. 2002), but the principal host is the large-eared woodrat (*Neotoma macrotis*). This thus represents a successful host-jumping event of the Bear Canyon Virus from the large-eared woodrat to the California mouse (Cajimat et al. 2007). More recently, in Texas, a close relative of Whitewater Arroyo virus was discovered in southern plains woodrats (*Neotoma micropus*): the Catarina virus or CTNV (Cajimat et al. 2007).

In Arizona, an arenavirus was isolated from a captured wild Mexican woodrat (*Neotoma mexicana*) that is considered a strain of a novel species, the Skinner Tank Virus (SKTV) (Cajimat et al. 2008). Very recently, 2 novel virus species were encountered from white-throated woodrats (*Neotoma albigula*) captured in Arizona, Big Brushy Tank virus (BBTV) and Tonto Creek virus (TTCV) (Milazzo et al. 2008).

In general, the implications of these viruses for public health and their association with neotomine or sigmodontine rodents in North America are subject of ongoing research (Cajimat et al. 2007).

Colorado Tick Fever

Colorado Tick Fever Virus (CTFV) is a double-stranded RNA-virus (arbovirus) representing the genus *Coltivirus* of the family *Reoviridae* (Attoui et al. 2005; Leiby and Gill 2004). Field studies in small mammals in the Rocky Mountains, USA established that *Eutamias minimus* and *Spermophilus lateralis* were the most important hosts for CTFV and were the source of virus for immature stages of the tick vector, *Dermacentor andersoni* (Bowen et al. 1981; Carey et al. 1980). Also porcupines (also a member of the *Rodentia*) are an important host (McLean et al. 1993). Beside the main tick vector, also other tick species (*D. occidentalis*, *D. albopictus*, *D. arumapertus*, *Haemaphysalis leporispalustris*, *Otobius lagophilus*, *Ixodes sculptus*, and *I. spinipalpis*) can be infected with the virus (Attoui et al. 2005). Humans can acquire infection by bites from a tick through infected saliva. Seasonal occurrence of infections is correlated with the presence and host-seeking activities of the tick vector. There is evidence of an endemic area where CTFV circulates between ticks and mammalian hosts, namely in the Western United States and in Western Canada (British Columbia). In the past, unconfirmed reports of CTFV presence in natural cycles came from other parts of the USA (Long Island) and eastern Canada (Florio et al. 1950; Newhouse et al. 1964).

In Colorado, at the end of the 1970s about 180 cases of human infection with CTFV were reported each year (op cit. Carey et al. 1980). However, actual incidence is unknown owing to insufficient physician knowledge of the disease, difficulties in establishing the diagnosis, and deficiencies in, or for some diseases absence of, reporting systems (Walker 1998).

Although subclinical infections might occur (Carey et al. 1980; Emmons 1988), most infections result in mild to moderately severe symptoms first appearing several days or 2 weeks post-infection. Onset is typically sudden, with chilly sensations, high fever, severe headache, retrobulbar pain, photophobia, lethargy, myalgia, and arthralgia (Emmons 1988). The spleen and liver are sometimes palpable. There may also be anorexia,

nausea, vomiting, abdominal pains, neurologic or encephalitic signs (disorientation, hallucinations, and stiff neck), and a variety of other rare or unusual complications (Emmons 1988). Mother-to-child transmission was reported in pregnant women (Attoui et al. 2005). In children, severe encephalitis and hemorrhages do occur and viremia can be persistent up to 120 days because of virus survival within maturing erythrocytes (Hughes et al. 1974). Pericarditis and myocarditis have also been reported (Emmons 1988). Fatalities seldom occur, especially since PCR techniques were developed that allow early diagnosis (Klasco 2002). Under specific clinical conditions, provision of ribavirin to patients can be considered (Klasco 2002). Symptomatic treatment includes acetaminophen for relief of fever and pain (Attoui et al. 2005).

Patients infected with CTFV show long-lasting immunity. An experimental vaccine was developed in the 1960s and produced long-lasting immunity, but production was stopped in the 1970s (Attoui et al. 2005).

Venezuelan equine encephalitis (VEE)

Venezuelan equine encephalitis virus (VEEV) is an emerging mosquito-borne pathogen of equids and humans that occurs in the USA (mainly in Texas and Florida), Central America (Mexico, Panama, Guatemala) and parts of South America (Peru, Colombia,Venezuela) (Aguilar et al. 2004). The virus is a member of the *Togaviridae* group, genus *Alphavirus* (Aguilar et al. 2004; Lukaszewski and Brooks 2000), and a number of different variants have been identified (Rico-Hesse et al. 1995). Since its discovery in the 1920s VEEV has caused periodic epidemics among human beings and equines in Latin America from the 1920s to the early 1970s (Weaver et al. 1996). In 1973 there was a large outbreak of VEE in Venezuela (Weaver et al. 1996). Consequences of these outbreaks for both human and domestic animal populations can be high: an outbreak in Colombia in 1995 caused an estimated 75,000 human cases, 3000 with neurologic complications and 300 fatal (Rivas et al. 1997). Of the state's estimated 50,000 equines, 8% may have died (Rivas et al. 1997).

Overall, human death rates have generally been estimated at approximately 0.5% during these epidemics, with most of the neurologic disease and fatal cases reported in children (Aguilar et al. 2004). The worldwide number of human cases of VEE is estimated to exceed 100,000 (Ferro et al. 2003).

Rodents are the reservoir host for VEEV. In the USA (Chamberlain et al. 1964)and Panama (Grayson and Galindo 1968), wild cotton rats (*Sigmidon hispidus*) are a host species for the mosquito (*Culex* species) that can also transfer the virus to humans. In Venezuela, antibodies were present in the Guaira spiny rat (*Proechimys guairae*), mouse oppossums (*Marmosa spp*), and common oppossums (*Didelphis marsupialis*) (Salas et al. 2001). In Peru, the recently identified VEEV subtype IIID strain was isolated from spiny rats (*Proechimys* spp.), Culex (*Melanoconion*) spp., mosquitoes and from a patient with fever, chills, and malaise (Aguilar et al. 2004). Besides contributing as reservoir hosts to the transmission cycle, the further role of rodents seems limited.

Currently, trials with a vaccine candidate to prevent infection with VEEV in horses look promising (Fine et al. 2007). Moreover, a series of nonclinical studies showed a vaccine candidate to be effective in protecting rodent and nonhuman primates against virulent challenge with several subtypes of VEEV (Fine et al. 2007). Others also report positive results in the battle against VEE (Lukaszewski and Brooks 2000; Riemenschneider et al. 2003). These studies could lead to the development of an effective human vaccine against the disease.

Western equine encephalitis

The Western equine encephalitis virus (WEEV) is an *Alphavirus* in the family of *Togaviridae*. It can cause the relatively rare viral disease Western equine encephalitis (WEE). Transmission patterns are similar to that of VEEV: the virus is transmitted by mosquitoes of the genera *Culex* and *Culiseta*. Especially gray and California ground squirrels were mentioned as host species for these mosquitoes (Hardy et al. 1974). More recently, antibodies were found in the cotton mouse (*Peromyscus gossypinus*), and the cotton rat (*Sigmidon hispidus*) in Florida (Day et al. 1996).

WEEV can cause sporadic and epidemic equine and human CNS infections (Castorena et al. 2008; Earnest et al. 1971). There have been about 700 confirmed human cases in the United States since 1964, but the disease is also present in countries in South America.

Overall mortality is approx. 4% and is associated mostly with infection in the elderly. There is no commercial vaccine against WEEV and there are no licensed therapeutic drugs in the United States for this infection (Wu et al. 2007). Consequently, scientists are working hard to develop a vaccine (Barabé et al. 2007; Wu et al. 2007).

Hepatitis E

Hepatitis E virus (HEV) is a virus in the genus Hepevirus of the family Hepeviridae (*Ahn et al. 2005; Denise Goens and Perdue 2004)*. Recent identification of HEV antibodies in pigs (Kase et al. 2008; Kulkarni and Arankalle 2008; Li et al. 2008; Zhang et al. 2008), horses, ducks (Zhang et al. 2008), sheep, chickens, and cattle suggested that animal reservoirs existed for this virus. In a study that

tried to reveal these reservoirs (Favorov et al. 2000) it was found that there is a widespread HEV or HEV-like infection in rodents, especially for those that live in urban habitat. The highest prevalence of antibody (59.7%) was found in the genus *Rattus* (Favorov et al. 2000); the high prevalence was confirmed in another study (Kabrane-Lazizi et al. 1999). Also, there are indications that rodents form a reservoir for HEV in other parts of the world, such as Nepal (He et al. 2002), where Hodgon's rat (*R. rattus brunneusculus*) and the lesser bandicoot rat (*Bandicota bengalensis*) displayed presence of antibodies.

Humans can acquire infection through direct contact with infected animals or through consumption of meat of animals that is not thoroughly cooked (Li et al. 2005). For example, pigs are frequently mentioned as natural hosts of HEV and are a likely source for human infection (Clayson et al. 1995; Li et al. 2005; Van der Poel et al. 2001). Transfer of the disease from rodents to these food animals (e.g., pigs) is a possibility, but how large risks are remains unknown. In humans, there are reports of vertical transmission (Khuroo et al. 1995; Tsega et al. 1992), but person-to-person transmission of HEV appears to be uncommon (Myint et al. 1985). Transfusion-transmitted infections of HEV due to infected blood do occur (Matsubayashi et al. 2008). Typical clinical signs and symptoms in patients with symptomatic HEV infection are similar to those of other types of viral hepatitis and include malaise, fever, anorexia, abdominal pain, nausea/vomiting, and hepatomegaly (Khuroo 1980; Myint et al. 1985). Hepatitis E is fatal in about 2% of all cases, whilst high case-fatality rates (15–25%) occur among pregnant women (Tsega et al. 1992).

Cowpox

Cowpox virus (CPXV) is a member of the genus *Orthopoxvirus* in the family *Poxviridae*, which is present in Eurasia. Being closely related to smallpox viruses and vaccinia virus (VACV), these viruses all induce cross-protection against each other. Vaccinia virus is one of the consequences of the discovery of vaccination by Edward Jenner, who used CXPV to induce human immunity against smallpox. In his time, poxvirus occurred naturally in livestock (Moss and Flexner 1987). His vaccine was passaged initially in humans and subsequently in cattle and sheep. In some places it was even mixed with smallpox virus, thus probably leading to the emergence of the 'new' vaccinia virus (Moss and Flexner 1987). In some regions of the world such as Brazil, this vaccinia virus has escaped to nature (Da Fonseca et al. 2002), thus circulating in rodents (Fonseca et al. 1998) and causing natural outbreaks.

Natural infection and disease with CXPV occurs primarily in domestic cats (Tryland et al. 1998) (rarely in man and cattle), and wild rodents are generally accepted as reservoir hosts (Boulanger et al. 1996). Antibodies have been detected in wild ground squirrels (*Citellus fulvus*) and gerbils (*Rhombomys opimus*, *Meriones libicus,* and *Meriones meridianus*) in Georgia (Tsanava et al. 1989) and Turkmenistan (Marennikova et al. 1977), in root voles (*M. oeconomus*) in northern Russia (Lvov et al. 1988) and from various rodents in Norway (Tryland et al. 1998). In the United Kingdom, antibodies have been found in house mice, but the highest seroprevalence was encountered in bank voles (*M. glareolus*), wood mice (*A. sylvaticus*), and field voles (*M. agrestis*) (Bennett et al. 1997; Chantrey et al. 1999; Crouch et al. 1995). In a study in bank voles and wood mice it was found that seroprevalences can vary considerably over time at the same site and among the same species (Hazel et al. 2000). Moreover, although the rodents do not show obvious signs of disease, during another study it was demonstrated that cowpox virus can reduce the fecundity of infected bank voles and wood mice by increasing the time to first litter by 20–30 days (Feore et al. 1997). Interestingly, it was found that bank voles that had high probabilities of infection survived better than uninfected individuals, something which was not found in wood mice. This suggests that each species has its own role in the transmission dynamics of cowpox virus (Telfer et al. 2002).

Moreover, in this mixed populations of hosts it was propagated that the best description for the transmission dynamics is frequency dependent, which means that each host makes a fixed number of contacts with other hosts, independent of the population size (Begon et al. 1999). On the contrary, previously it was thought that the best descriptive model was density dependent: susceptible hosts were assumed to contact other hosts throughout the whole of their population at random. The number of these contacts then rose in proportion to the size of the population.

Also, hosts can influence each other: in a study on 14 islands in Northern England (Begon et al. 2003) it was found that in the case of cowpox dynamics, wood mouse density thresholds were influenced at least as much by the bank vole thresholds as they were by the dynamics within the wood mouse populations themselves.

Rodents can also transfer the disease directly to humans, as was reported some years ago when a Norway rat (*R. norvegicus*) transmitted the disease to a woman (Wolfs et al. 2002). Patients show painful, hemorrhagic pustules or black eschars, usually on the hand or face, accompanied by edema, erythema, lymphadenopathy, and systemic involvement (Baxby et al. 1994). Fatalities mainly occur in the immunosuppressed (Baxby et al. 1994).

Via induced cross-immunity the WHO elemination campaign for smallpox with worldwide vaccination also had positive results for the suppression of

cowpox (Pelkonen et al. 2003; Vorou et al. 2008). Since the elimination of smallpox, vaccination practices were abandoned, resulting in decreased immunity of younger unvaccinated age groups against other orthopoxviruses such as monkeypox and cowpox (Pelkonen et al. 2003; Vorou et al. 2008). As effective treatments do not exist, some argue that cowpox can therefore be considered as an emerging zoonotic health threat (Vorou et al. 2008).

Contagious viral animal diseases

Although not important from the perspective of human health, the consequences of an outbreak of contagious viral animal diseases (classical swine fever, foot and mouth disease, avian influenza) for the economy (particularly the livestock sector) are significant. The 2001 outbreak of foot and mouth disease in the United Kingdom is estimated to have caused economic losses in the order of 3.1 billion pounds (Thompson et al. 2002). The effect on the image of the animal husbandry sector are more difficult to measure.

During recent classical swine fever virus (CSFV) outbreaks, rodents were mentioned in direct connection with the large numbers of secondary infections that were observed in the vicinity of primary infected herds (Elbers et al. 1999; Hughes and Gustafson 1960; Mintiens et al. 2003; Terpstra 1988; Westergaard 1996). This was due to the fact that in many of these outbreaks none of the 'traditional' transmission routes for CSFV, for example, direct animal contact, swill feeding, or transport contact, were responsible for the virus spread (Terpstra 1988).

Although often linked, there are only a few experiments that describe the role of rodents in transmission of contagious animal diseases (DeWulf et al. 2001; Hughes and Gustafson 1960; Terpstra 1987). An experiment by Terpstra (Terpstra 1987) revealed that rats which were fed in close contact with CSFV-infected pigs were not able to transmit the infection to susceptible animals. Another study provides evidence that rats are unlikely to represent significant biological reservoirs of CSFV (DeWulf et al. 2001). The same authors suggest that the likelihood of mechanical spread is difficult to assess and claim that the mechanical spread of CSFV by pets and rodents remains a possibility. This opinion is shared by other authors (Elbers et al. 1999), who explain that it is conceivable under field conditions that rodents with a sub-optimal health will have less-efficient grooming of their fur, which may therefore increase the possibility to mechanically transmitted CSFV. On the other hand, in a epidemiological study in which the 1997-1998 outbreak of CSFV in the Netherlands was studied no associations between the presence of rats or mice around the premises and increased risk of infection with CSFV was found (Elbers et al. 2001). This may be because farmers had to respond in a questionnaire whether there were rodents present on their farms. From earlier studies (Meerburg et al. 2004), we know that this formulation generally leads to an underestimation of rodent presence.

Rodents may be involved to some degree in the epidemiology of foot-and-mouth disease because the brown rat *Rattus norvegicus* is susceptible to foot-and-mouth disease and can excrete the virus over long periods. Therefore rodents may play a significant part in the dissemination of the disease (Capel-Edwards 1970).

Moreover, rodents are also mentioned in association with a number of other animal diseases such as porcine parvovirus (Joo et al. 1976; Joo et al. 1976) which can lead to reproductive failure in pigs and Aujeszky's disease virus (Maes et al. 1979), a disease that is also known as pseudorabies or 'mad itch.' Rodents also are associated with the horizontal transmission of clinical encephalomyocarditis fever virus (ECMV) between farms (Knowles et al. 1998; Maurice et al. 2007; Spyrou et al. 2004), thus leading to economic losses.

Bacteria

Leptospirosis

Rodents are carriers of spirochetes of the genus *Leptospira* throughout the world (Boqvist et al. 2002; Bunnell et al. 2000; Thiermann 1977; Webster et al. 1995; Wisseman et al. 1955) and are important reservoirs of infection for man and domestic animals. Several *Leptospira* strains are directly linked to rodents, such as *L. arborea*, *L. copenhagi*, *L. icterohaemorrhagiae, L. bim,* and *L. ballum* (Bharti et al. 2003; Collares-Pereira et al. 1997; Collares-Pereira et al. 2000). The complex taxonomy of different Leptospira species is explained in an excellent review by Barthi and collegues (Bharti et al. 2003).

Humans acquire infection through consumption of food or water that is contaminated by rodents or by contact through skin or mucous membranes with soil or water that is contaminated by rodent urine. Handling of dead infected rodents may also form a source of infection. It can lead to aseptic meningitis or Weil's disease, which is characterized by lymphadenopathy, jaundice, renal failure, and hemorrhages (Arean 1962; Bharti et al. 2003; Plank and Dean 2000; Steele 1958). The mortality rate associated with severe leptospirosis may be as high as 15% (Ko et al. 1999). Although there are some positive reports on the use of antileptospiral vaccines in humans, long-term efficacy studies of vaccines have not been published (Bharti et al. 2003) and many questions remain on virulence factors and pathogenesis (Koizumi and Watanabe 2005).

The number of human cases worldwide is not well-documented and suffers from consequent under-reporting in many areas of the world (WHO, Geneva, http://www.who.int). It probably ranges from 0.1 to 1 per 100 000 per year in temperate climates to 10 or more per 100 000 per year in the humid tropics. During outbreaks (often associated with disasters) and in high-risk groups, 100 or more per 100 000 may be infected (WHO, Geneva, http://www.who.int).

Leptospirosis has a major impact on rural communities in developing countries in Asia. An epizootic of leptospirosis in humans occurred in NE Thailand from 1995 to 2003. In 1996, 398 cases were reported in 4 provinces, with a peak in 2000 of 14,285 cases and 362 deaths, and cases reported across 16 provinces in 2001. The number of human cases remained high until 2003, with 171 deaths in 2001 and 95 in 2002. Most cases (ranging from 72% to 94% of those reported in a year) occurred among rice farmers (Phulsuksombati et al. 2001; Tangkanakul et al. 2005), resulting in a severe impact on rural as well as urban communities in the region (Tangkanakul et al. 2001). Information on leptospirosis in other regions in Asia is extremely limited. The symptoms are flu-like and are often mistakenly diagnosed and neglected in the rural areas until serious clinical damage has occurred.

In the Philippines, >1,000 people are hospitalized annually with leptospirosis. Fatality is high rate ranging from 11% to 20%. This appears to be in part because people go to hospital only if severe symptoms develop because they cannot afford to pay for long stays in a hospital. Therefore, it is the urban and rural poor who are at greatest risk because of higher rates of exposure to infectious bacteria and little available income for early medical intervention.

Lyme disease

Rodents play an important role in the spreading of the Lyme disease spirochetes *Borrelia burgdorferi* (Shih and Chao 1998), *B. garinii,* and *B. afzelli* (Parola and Raoult 2001). The enzootic rodent-tick cycle maintains *Borreliae* specifically (Nakao et al. 1994), although the relative potential as reservoir differs between rodent species (Brown and Lane 1996; Burkot et al. 1999; Humair et al. 1999; Mather et al. 1989; Sinski et al. 2006). During a study in Switzerland, *Borrelia* infection was more prevalent in *Myodes* than in *Apodemus* (Humair et al. 1999). Also in an earlier study it was demonstrated that *M. glareolus* plays a different role as reservoir host species compared with two *Apodemus* species (Kurtenbach et al. 1995). These authors also found that prevalence of *Borrelia burgdorferi* in host-seeking ticks (*Ixodes ricinus L.*) was 1% for larvae, 5% for nymphs, and 10–20% for adults. In the USA (California), it was shown that dusky-footed woodrats and California kangaroo rats showed infection prevalences of 85.7% and 78.6%, respectively. In contrast, only 22.2% of brush mice (*Peromyscus boylii*) and 7.1% of pinyon mice (*P. truei*) were infected (Brown and Lane 1996). During another study in the USA (New York), the determinants of Lyme-disease risk (density and Borrelia burgdorferi-infection prevalence of nymphal *Ixodes scapularis* ticks) were assessed (Ostfeld et al. 2006). It was shown that the strongest predictors of a current year's risk were the prior year's abundance of mice and chipmunks and abundance of acorns 2 years previously (Ostfeld et al. 2006).

From a study in Germany we know that immunity to *B. burgdorferi* in natural reservoir hosts is an important regulatory factor in the horizontal transmission of *B. burgdorferi* in nature (Kurtenbach et al. 1994). Voles carry a larger number of ticks than mice, while on the other hand mice are more frequently infected with the pathogen. Thus, voles are high responders to the vector (higher infectivity), while mice are high responders to the microparasite (higher % infected) (Kurtenbach et al. 1994).

This demonstrates that mice and voles play different quantitative roles in the ecology of Lyme borreliosis in Europe. To make things even more complicated, the presence of predatory vertebrates may indirectly protect human health by reducing the population size of rodent reservoirs (Ostfeld and Holt 2004). On the other hand, some researchers describe zooprophylaxis (Matuschka et al. 1991). These researchers think that because rodents are present, the rate at which ticks bite other hosts (including humans) may be reduced, and thus the likelihood of infection is also reduced (Matuschka et al. 1991; Schmidt and Ostfeld 2001). Some say that there is proof that increased biodiversity (i.e., an increasing number of potential tick host species) may lead to a dilution effect (Dobson et al. 2006; Schmidt and Ostfeld 2001).

In the USA, *Ixodes dammini* and pacificus ticks are the most important vectors of the pathogen, while in Europe *Ixodes ricinus* is the responsible vector. In Asia, particularly China, *B. burgdorferi* was encountered in the pygmy wood mouse (*Apodemus uralensis*) and the long-tailed dwarf hamster (*Cricetulus longicaudatus*) and in *I. persulcatus* ticks (Takada et al. 2001).

Humans are infected by the bite of a tick that acquired the infection from an enzootic reservoir. Infection can lead to Lyme's disease, which is characterized by cutaneous manifestations (for example, *Erythema chronicum migrans*, a rash that is an early sign of infection which helps early diagnosis). If the disease remain unnoticed, it can affect the nervous system, heart, eye, and joints in variable combinations. Fatalities due to Lyme's disease seldom occur. However, there are differences in the clinical presentations of Lyme borreliosis between continents, e.g., between the USA and Europe. Patients in Europe may develop certain skin manifestations, such

as borrelial lymphocytoma and acrodermatitis chronica atrophicans) that are either rare of non-existent in the USA (Strle et al. 1999). This might be caused by strain variation: in United States patients, members of the genomic group *Borrelia burgdorferi* sensu stricto were identified, whereas European isolates have included two additional genospecies, *B. garinii* and *B. afzelii* (Picken et al. 1998; Strle et al. 1999).

Although a vaccine based on the recombinant outer-surface protein A (OspA) was available (Wormser et al. 2000), this was withdrawn from the market due to its side effects.

In recent decades the tick that is responsible for spreading of the pathogen in Europe has spread into higher latitudes and has become more abundant, possibly associated with climate change (Lindgren et al. 2000; Randolph 2005). According to the WHO (WHO, Geneva, http://www.who.int), this will also contribute to extended and more intense transmission seasons for all tick-borne diseases, such as Lyme's disease and tick-borne encephalitis (TBE).

Tick-borne relapsing fever

Tick-borne relapsing fever (TBRF), an underrecognized and underreported disease, is caused by numerous species of the spirochete *Borrelia* and occurs throughout the world in many discrete enzootic foci where the spirochetes are maintained primarily in rodents and soft-body ticks of the genus *Ornithodoros* (Schwan and Hinnebusch 1998), with the exception of Australia, New Zealand, and Oceania (Johnson 1977).

In North America, several relapsing fever spirochetes, *Borrelia hermsii*, *Borrelia turicatae*, and possibly, *Borrelia parker* (Fritz et al. 2004), alternate infections between rodents and tick vectors (*Ornithodoros spp.)* (Boyer et al. 1977; Dworkin et al. 2002; Dworkin et al. 2002). Many residents and visitors are exposed in the endemic regions of the western United States to the vectors of TBRF. There are approximately 25 cases of TBRF in the United States each year (CDC, Atlanta, http://www.cdc.gov). The pathogen can be found primarily in chipmunks (*Tamias* spp.) and pine squirrels (*Tamiascuirus* spp.) above elevations of 1,000 m (Fritz et al. 2004). In Israel, cases of TBRF are thought to be the result of the pathogen *B. persica* that is transmitted by the tick *Ornithodoros tholozani* (Sidi et al. 2005). This pathogen is also present in Syria, Egypt, Iran, and Central Asia (Rebaudet and Parola 2006). Recurrent fever due to *B. hispanica* is reported sporadically in Spain, Portugal, Cyprus, Greece and North Africa. A new species pathogenic to humans was isolated and characterized in Spain in 1996 in *Ornithodoros erraticus* (Anda et al. 1996).

West African tick-borne relapsing fever (TBRF) is caused by the microorganism, *Borrelia crocidurae*. If untreated, mortality rates are up to 5% (Southern Jr. and Sanford 1969). A survey in Senegal (Godeluck et al. 1994) demonstrated that the African grassrat (*Arvicanthis niloticus*) and *Mastomys huberti* (Hubert's Mastomys) function as hosts for the pathogen. Besides Senegal, this pathogen also occurs in Morocco, Libya, Egypt, Iran, and Turkey (Rebaudet and Parola 2006).

Borrelia caucasica, present in Iraq and the Caucasus, is transmitted by *Ornithodoros asperus*, another tick of rodents. *Borrelia latyschevii* is transmitted by *Ornithodoros tartakovskyi* in Central Asia, Russia, and Iran (Estrada-Peña and Jongejan 1999).

Humans acquire infection when infected ticks that live in the burrows or nests of rodents within a human dwelling search for an alternative host to feed upon. Such infection can result in recrudescent illness with the characteristic clinical syndrome of periodic fevers. The first attack is generally the most severe in terms of height and duration of fever, while succeeding attacks are usually milder. Severity is highest in (pregnant) women and children (Barbour 1999; Jongen et al. 1997). Incubation time after the tick bite is between 4 and 14 days (Johnson and Golightly 2000).

Scrub typhus

Scrub typhus (also named tsutsugamushi disease) can be the result of infection of humans with the bacterium *Orientia tsutsugamushi*. This pathogen is transmitted by bites from infected trombiculid mites ('chiggers') that are hosted by various rodent species, such as the Norway rat (*R. norvegicus*), the lesser bandicoot (*Bandicoota bengalensis*), the bandicoot rat (*Bandicoota indica*), house mouse (*Mus musculus*), the striped-field mouse (*Apodemus agrarius*), and the Japanese grass vole *Microtus montebelli* (Milne-Edwards) (Kumar et al. 2004; Liu et al. 2003; Takahashi et al. 2004). Cases of scrub typhus have been reported from Southern and Eastern-Asia (Japan, Indonesia, Korea, Thailand, Asiatic Russia, the Indian subcontinent, Pakistan, China), and Australia (Graves et al. 2006; Khuntirat et al. 2003; Kim et al. 2007; Liu et al. 2003; Sin et al. 2000; Takahashi et al. 2004; Traub et al. 1954; Yahnke et al. 2001). In Australia, hosts of chiggers include introduced rodent species, native rodent species and small marsupials, particularly the long-nosed bandicoot, *Perameles nasuta*, and scrub typhus has been reported down the east coast of Australia (Spratt 2005). Similar to murine typhus, people acquire the infection through the bite of an infected mite. The estimated incidence of the disease in endemic areas is one million cases each year (op cit. Fournier et al. 2008). Incubation time varies between 6 and 12 days (Varghese et al. 2006). Symptoms include fever, headache, muscle pain, cough, and gastrointestinal symptoms. More virulent strains can cause hemorrhages and intravascular

coagulation. If untreated, mortality rates can mount up to 40% (Varghese et al. 2006), but with use of antibiotics it is below 5% (Dupon et al. 1992). Though infection can be easily treated with doxycycline/azithromycin, clinical diagnosis is often difficult because the characteristic eschar at the site of the bite is present only in the minority of the patients.

In northern Thailand in 2000, more than 3,900 cases of scrub typhus were reported, with most cases being male rice farmers (in northeast Thailand in 2000, the morbidity rate was 8.7 per 100,000 people) (W. Tangkanakul, personal communication). A 10-year study in northern Thailand identified 9 murid rodents as carriers of scrub typhus, with the main carriers being *R. rattus* (23%, 419 of 1,855), *R. argentiventer* (2%, 5 of 23), *B. berdmorei* (22%, 2 of 9), *R. losea* (13%, 82 of 638), and *B. indica* (9%, 52 of 564) (Coleman et al. 2003).

In Northeast Thailand in 1997, nine of 22 human cases of leptospirosis were diagnosed as positive to scrub typhus. This is the first report of a high incidence of co-infection with leptospirosis and typhus. Twenty of the 22 people screened were rice farmers, a group known to be at high risk in Thailand for both diseases (Watt et al. 2003). The authors highlighted the importance of this finding with the reported death of a patient who was diagnosed with leptospirosis and was treated with the appropriate antibiotic. However, the patient was not diagnosed as a possible typhus victim (combined infection) and scrub typhus was not sensitive to the antibiotic administered (intravenous penicillin). In Asia, doctors who diagnose leptospirosis in agricultural workers are recommended to also consider co-infections with typhus in patients that do not respond to treatment for leptospirosis (Watt et al. 2003).

Murine typhus

Rodents, more specifically rats, are associated with the worldwide distribution of the bacterium *Rickettsia typhi* (Chaniotis et al. 1994; Graves et al. 2006; Gray et al. 2007; Kim et al. 2007; Letaief 2006; Reeves et al. 2006), which can cause murine typhus. The vector is the rat flea, *Xenopsylla cheopis*. The triad of rickettsia-flea-rat seems to be a true commensalism, because the rickettsiae harm neither rat nor flea (Azad 1990). Humans acquire murine typhus from an infected flea: most fleas defecate while biting and their feces can contain the bacteria that cause the disease. Although uncommon, it is also possible for humans to contract murine typhus by inhaling contaminated dried flea feces. The incubation period for the disease lies on average between 6 to 14 days. Symptoms include headache, fever, nausea, and body aches. Five or six days after the initial symptoms, 54% of the patients get a rash that starts on the trunk of their bodies and then spreads to arms and legs (Elston 2005). The full triad of fever, headache, and rash occurs in only 12.5% of the patients (Elston 2005). Respiratory and gastrointestinal symptoms are frequent and may result in confusion with a viral illness. A worse prognosis is noted in those with renal dysfunction, leukocytosis, and hypoalbuminemia (Elston 2005). Adverse outcomes are also associated with advancing age, and therapy with sulfa antibiotics (Dumler et al. 1991). Mortality rate for murine typhus is between 1–4%. No effective vaccine is available (Baxter 1996).

Murine typhus is likely to persist in endemic areas where rat populations remain high (Azad 1990), especially in coastal areas in the vicinity of ports. This was also demonstrated by a survey in Africa, which demonstrated that prevalence of antibodies against *R. typhi* in humans was higher in coastal areas (Tissot-Dupont et al. 1995). In some countries (Pakistan, Thailand, India, Myanmar, and Southern USA) the infection has a wider distribution than only the coastal areas (Azad 1990).

Because the disease is frequently underreported, it is difficult to provide reliable incidence rates. In the United States about 42,000 cases were reported between 1931 and 1946, but incidence rapidly declined due to rat control programs. Since 1961 the number of reported cases per year has remained well under 100 (Azad 1990; Traub et al. 1978).

Recently, the pathogen which causes murine typhus has been shown to have an alternative peridomestic animal cycle that does not involve rats and *X. cheopis*. This new cycle was reported in the USA involving cats, dogs and opossums, and the cat flea *Ctenocephalides felis* (Azad et al. 1997).

Sylvatic epidemic typhus

Sylvatic epidemic typhus, caused by *Rickettsia prowazekii,* is transmitted by ectoparasites (lice and fleas) of rodents to humans. In large parts of the world (South and Central America, Africa, Asia, Mexico), *R. prowazekii* are also transmitted between humans (Yu and Walker 2006). This is done by the body louse *Pediculus humanus humanus*, that lives in clothes and multiplies when such conditions as cold weather, lack of hygiene, or war are present (Raoult and Roux 1999). Its prevalence can be considered as an indicator for the socioeconomic level of the society (Raoult and Roux 1999).

In the wild, the pathogen is maintained in a southern flying squirrel (*Glaucomys volans*) sylvatic cycle in the eastern United States (Foley et al. 2007; Reynolds et al. 2003). Only a few cases have been reported since 1985 (Reynolds et al. 2003). In one third of these cases, contact with flying squirrels or with flying squirrel nests occurred before disease onset (Reynolds et al. 2003). In general, all louse-borne diseases can be transmitted by contact of broken skin or wounds with infected louse,

fleas, mites or their droppings, or by inhalation of their aerosolized feces. However, the transmission mechanism of *R. prowazekii* from flying squirrels to humans is less well understood. Although various transmission modes are mentioned, none has been empirically confirmed. However, one species of fleas (*Orchopeas howardii*) that is hosted by flying squirrels is known to opportunistically bite humans, thereby playing an important role in pathogen transmission (Reynolds et al. 2003).

Queensland tick typhus

Queensland tick typhus or spotted fever has been reported along the eastern coast of Australia. It is caused by *Rickettsia australis* and was first described in soldiers posted to northern Queensland during World War II. The vectors have been described as *Ixodes holocyclus*, *Ixodes tasmani* and, probably, *Ixodes cornuatus*. The reservoir hosts are rodents and marsupial bandicoots (Spratt 2005).

Rocky Mountain spotted fever

Rocky Mountain spotted fever (RMSF) is the most severe and most frequently reported rickettsial illness in the United States. Spotted fever also occurs in Mexico and in Central and South America (Fuentes 1986; Hoogstraal 1967), but is sometimes known under different names there (e.g., Brazilian Spotted Fever). The causative agent is *Rickettsia rickettsii*, a bacterium of the *Rickettsiaceae* family, that spreads to humans by ixodid ticks. Rodents can serve as an reservoir of the infectious agent (Atwood et al. 1965), e.g., white-footed mice (*Peromyscus leucopus*) (Magnarelli et al. 1981), the hispid cotton rat (*Sigmodon hispidus*), the cotton mouse (*Peromyscus gossipinus*, Le Conte), the golden mouse (*Ochrotomys nutalli*, Harlan), the woodland vole (*M. pinetorum*), the prairie vole (*Microtus ochrogaster*, Wagner), and the meadow vole (*M. pennsylvaticus*, Ord) are mentioned as possible hosts (Kollars Jr. 1996).

The American dog tick (*Dermacentor variabilis*) and Rocky Mountain wood tick (*Dermacentor andersoni*) are the primary vectors of the pathogen in North-America, while the brown dog tick (*Rhipicephalus sanguineus*) can also be a vector (Demma et al. 2005; Hoogstraal 1967). In Central and South America, the tick *Amblyomma cajennense* can be considered a main vector (Figueiredo et al. 1999).

Initial signs and symptoms of spotted fever include sudden onset of fever, headache, and muscle pain, followed by development of rash. The disease is sometimes difficult to diagnose in the early stages, and without prompt and appropriate treatment with doxycycline fatalities do occur. The incubation period is between 3 and 12 days (Walker 1995).

Rickettsialpox

A disease that is associated with urban environments is rickettsialpox (Comer et al. 2001; Comer et al. 2001). This disease is caused by the worldwide distributed (Comer et al. 2001) pathogen *Rickettsia akari*, which is transmitted to humans by mites of rodents. The pathogen is maintained by vertical transmission in the house mice mite (*Liponyssoides sanguineus*) and by horizontal transmission between the mite and its main host: the house mouse (*M. musculus domesticus*) (Radulovic et al. 1996). The possibility exists, however, that other species of mites are also involved in the cycle (Bennett et al. 2007). Moreover, the pathogen was also isolated from commensal rats in Ukraine, from black rats (*R. rattus*), dusky-footed woodrats (*Neotoma fuscipes*) and deer mice (*P. maniculatus*) in the USA (Bennett et al. 2007) and from Korean reed voles (*Microtus fortis*) in Korea (Jackson et al. 1957). This suggests that *R. akari* can adapt to other rodent hosts. Human infections were reported in the USA (Bennett et al. 2007; Kass et al. 1994; Paddock et al. 2003; Paddock et al. 2006), in South Africa (Gear 1954), Turkey (Ozturk et al. 2003), in Croatia (Radulovic et al. 1996), in Bosnia and Herzegovina (Terzin et al. 1956), and in Ukraine (Eremeeva et al. 1995). Infection of humans occurs only if mice or other preferred hosts are not available (Heymann 1996).

The clinical portrait of rickettsialpox in humans consists of a cutaneous lesion at the site of inoculation by the mite: first a papule appears and later this evolves into an eschar (Yu and Walker 2006). After about 1 week, patients develop fever, chills, malaise, and headache, followed shortly by a secondary papulovesicular cutaneous eruption (Heymann 1996). Although the symptoms look severe, patients will usually recover within 1 to 2 weeks. No fatalities have been reported (Heymann 1996). However, rickettsialpox is infrequently reported and underdiagnosed at present.

Bartonella illnesses

Of the 19 described *Bartonella* species that infect a wide variety of domestic and wild animals such as cats, dogs, mice, rats, squirrels, deer, and moose, 7 have been associated with human disease (Alsmark et al. 2004). Fleas and lice are efficient vectors of rodent bartonellae (Boulouis et al. 2005; Bown et al. 2004) and are involved in the transmission to humans. For this reason, disease often occurs in people with a low-living standard such as the homeless or intravenous drug users (Comer et al. 2001; McGill et al. 2003; McGill et al. 2003; Smith et al. 2002).

Rodents can form a reservoir for several *Bartonella* species such as *B. elizabethae, B. washoensis, B. grahamii, B. taylorii,* and *B. vinsonii.* Evidence of host specificity suggests the possibility of a long-term co-evolution or co-speciation of *Bartonella* with their

rodent hosts (Kosoy et al. 2000). Prevalences in rodent populations can be quite high (11.1–62.2%) with variance between different rodent species as was demonstrated in studies from the United Kingdom, the southeastern USA and Poland (Birtles et al. 1994; Easterbrook et al. 2007; Kosoy et al. 1997; Welc-Faleciak et al. 2008). In a review from some years ago (Boulouis et al. 2005) it is described that Norway rats (*R. norvegicus*) constitute the main reservoir of *B. elizabethae,* while *Bartonella grahamii* has been mainly isolated from bank voles (*M. glareolus*) in the UK and Poland and yellow-necked mice in Sweden. However, this Bartonella species was also isolated in rats and a domestic mouse in the USA. According to these authors, California ground squirrels (*Spermophilus beecheyi*) are the main reservoir of *B. washoensis* (Boulouis et al. 2005). Isolation of *B. henselae* (the causative agent of cat's scratch disease in humans) was recently reported in three long-tailed field mice (*Apodemus sylvaticus*) in Denmark (Engbaek and Lawson 2004). In Spain, two different Bartonella genotypes were detected in *M. spretus*, and one genotype that was corresponding with *B. tribocorum* in the Norway rat (*R. norvegicus*) (Márquez et al. 2008). However, it remains questionable to what extent rodents contribute to *Bartonella* transmission to humans (Anderson and Neuman 1997). The probable mode of transmission is via feces that is deposed by rodent ectoparasites on the broken human skin.

Some years ago, *B. grahamii* was isolated for the first time from the eye of a patient with neuroretinitis (Kerkhoff et al. 1999; Meerburg and Kijlstra 2007) in Europe and possibly linked to rodent presence, although no clear evidence could be provided. But if transmission occurs, consequences may be serious, as in humans *Bartonella* spp. can lead to endocarditis, cardiac disease and febrile illness respectively (Breitschwerdt and Kordick 2000; Iralu et al. 2006; Welc-Faleciak et al. 2008). In the immunodepressed (e.g., AIDS-patients), *B. quintana* has been related with chronic lymphadenopathy and numerous atypical manifestations (English 2006).

Human granulocytic anaplasmosis

Humans can get infected with *Anaplasma phagocytophilum*, the causative agent of human granulocytic anaplasmosis (HGA), previously known as human granulocytic ehrlichiosis, when they infringe on tick-small mammal habitats (Dumler and Bakken 1998). HGA was first recognized in 1994 (Bakken et al. 1994). HGA is characterized by headache, high fever, chills, and myalgias. In the early stage, the disease can be readily treated with antibiotics. However, if untreated, severe illness can occur, including secondary infections due to renal dysfunctions, respiratory failures, and occasionally this may even result in death (Fritz and Glaser 1998). Infected ticks (*Ixodes* spp.) are hosted by rodents (primarily wood rats and white-footed mice, but also other rodents such as squirrels are capable of carrying these ticks (Levin et al. 2002). If these infected ticks then bite a human, they frequently cause HGA-fever in several parts of the world, such as the USA (Upper Midwest, New England, parts of the mid-Atlantic states, northern California), many parts of Europe and sometimes even Asia.

HGA is clinically variable. Recent sero-epidemiological surveys suggested that many infections go unrecognized, since in endemic areas as much as 15–36% of the population has been infected (Bakken et al. 1998; Mattson et al. 2002). Most patients have a moderately severe febrile illness with headache, myalgia, and malaise. The estimated fatality rate is 0.7% as determined from surveillance data, and elderly patients are more prone to severe infections and death (Demma et al. 2005). From the period 1986–2001 in total 1540 cases of HGA were reported in the USA. The average reported annual incidences for HGA in the USA during 2001–2002 was 1.4 cases per million people. However, because of the nonspecific nature of clinical signs, human granulocytic anaplasmosis is most likely underrecognized and under reported (Demma et al. 2005).

Q-fever

Q-fever is caused by the intracellular gram-negative bacterium *Coxiella burnetii*. Morphologically, the genus *Coxiella* is similar to the genus *Rickettsia*, but with a variety of genetic and physiological differences.

Coxiella bacteria are present throughout the world, except for Antarctica and probably New Zealand (Fournier et al. 1998). Cattle, sheep and goats are the most common animal reservoir of this zoonotic disease.

Rodents also are suspected as a reservoir, but their role in transmission to humans might be limited compared to other pathways. Nevertheless, rodents can be infected. In the United Kingdom, wild Norway rats (*R. norvegicus*) displayed seroprevalences between 7-53% (Webster et al. 1995), while in the USA low seroprevalences were detected in muskrats (*Ondatra zebethica*), rats (*Rattus* spp.), Beechey ground squirrels, (*Otospermophilus beecheyi*), wood rats (*Neotoma fuscipes*), and deer mice (*Peromyscus* spp) (Riemann et al. 1979). In Japan, another rodent species, *Myocastor coypus,* has shown moderate (13%) seroprevalence for *C. burnetii*, (Ejercito et al. 1993).

People acquire infection through inhalation of contaminated dust, contact with contaminated animal products (milk, meat, wool) and particularly birthing products (amniotic fluid etc.). Moreover, ticks can transfer the pathogenic agent to other animals, but contrary to other tick-borne pathogens, not to humans (op cit. Fournier et al. 1998).

If untreated, the disease is usually deadly, but with appropriate treatment the death rate is around 10%. During its course, the disease can progress to an atypical pneumonia, which can result in a life threatening acute respiratory distress syndrome (ARDS), which is frequently found in North America. Symptoms usually occur during the first 4–5 days post infection. However, at other locations in the world (e.g. Europe) Q-fever causes (granulomatous) hepatitis which becomes symptomatic with malaise, fever, liver enlargement (hepatomegaly), pain in the right upper quadrant of the abdomen and jaundice (icterus). If a person is chronically infected, the disease can lead to endocarditis. Q-fever also causes abortions and reproductive problems in livestock, and thus economic losses for farmers. In livestock, a natural cycle of transmission without involvement of ticks has been postulated (Krauss 1989).

Salmonellosis and campylobacteriosis

Salmonella and Campylobacter are generally regarded as the most important food-borne pathogens in the world. Reduction or elimination of these pathogens in the first part of the food chain (on the farm) is important to prevent disease among consumers of animal products. Previous research has proven that wild rodents and house mice are able to amplify these pathogens in the environment and are probably capable of transmitting them to food animals (Davies and Wray 1995; Evans and Sayers 2000; Garber et al. 2003; Henzler and Opitz 1992; Meerburg et al. 2006; Meerburg 2007). If products of these animals (e.g., their meat) are then improperly cooked, they could lead to human infection. It is known also that rodents can be long-term sources of infection: e.g., a study demonstrated rodents are still capable to transfer *Salmonella enteriditis* to chicks after two and five months post infection (Davies and Wray 1996). Unfortunately, it is not yet known to what extent human cases of salmonellosis or campylobacteriosis can be related to rodents, but other sources are presumably more important. The consequences of infection with these pathogens can be severe: diarrhea, headache, vomiting, and sometimes even death.

Tularemia

Tularemia is caused by the intracellular gram-negative bacterium *Francisella tularensis*, a member of the Francisellaceae family. Humans can acquire infection through direct contact with infected animal carcasses, consumption of food or water that is contaminated by rodents, after a bite of an infected mammal (Friedl et al. 2005), tick, deerfly, or another insect or by breathing aerosols containing the bacteria (Christova and Gladnishka 2005; Hörnfeldt 1978; Pape et al. 2005; Petersen and Schriefer 2005; Wobeser et al. 2007). Tularemia mainly occurs in the northern hemisphere and most frequently in Scandinavia, Central Europe, Northern America, Japan, and Russia, although *F. tularensis* subsp. *novicida* has been reported in Australia (Hollis et al. 1989).

Until now, four subspecies of the pathogen have been discovered, which each exhibit distinct biochemical and viral profiles (Farlow et al. 2005). Human disease is primarily associated with two of these subspecies: the highly virulent *F. tularensis* subsp. *tularensis* (type A), which can only be encountered in North America and the moderately virulent *F. tularensis* subsp. *holarctica* (type B), which is endemic throughout the Northern Hemisphere (Farlow et al. 2005). While type A is reported to have a terrestrial cycle with the main reservoirs being cottontail rabbits (*Sylvilagus* spp.) and ticks (Mörner 1992). Recently, molecular subtyping has further divided type A into 2 subpopulations, A1 and A2 (Kugeler et al. 2009). Type B is reported to have a mainly water-borne cycle with aquatic rodents as reservoirs, e.g., muskrats (*Ondatra zibethicus*) and beaver (*Castor canadensis*) in North America, and ground voles (*Arvicola terrestris*) in the former Soviet Union (Mörner 1992). In Europe, tularaemia is most frequently seen in hares (*Lepus* spp.) (Mörner 1992).

Humans contract tularaemia mostly through mosquito bites or by handling infected animals (Ikaheimo et al. 2000). The disease occurs in several forms in humans, depending to a large extent on the bacterial entry route into the body. Most common is ulceroglandular tularemia, which usually occurs after a bite from an arthropod vector which has previously fed on an infected animal (Ellis et al. 2002). Sometimes cases of ulceroglandular tularemia occur in hunters and trappers as a consequence of the handling of infected meat, with infection via cuts or abrasions. Although the ulceroglandular form of tularemia even without treatment is rarely fatal (mortality rate less than 3%), patients may take a significant time (2–3 months) to heal.

Mortality ranges depend strongly on the subspecies of the infection (Kugeler et al. 2009). Very recently, pulsed-field gel electrophoresis typing identified 4 distinct type A genotypes, A1a, A1b, A2a, and A2b, as well as type B (Kugeler et al. 2009). Human infections due to A1b resulted in significantly higher mortality (24%) than those caused by A1a (4%), A2 (0%), and type B (7%).

The worldwide incidence of this disease is unknown, (WHO, Geneva, http://www.who.int), but several thousands of cases each year have been estimated. Nevertheless, some data on the incidence of disease are available. In Japan 1355 cases were reported between 1924 and 1987 (Ohara et al. 1991), while in Sweden, the annual number of reported case from 1973 to 1985 ranged from less than 5 cases to over 500 (Mörner 1992). In 2003, 823 human cases of tularemia were reported in Finland, 698 in Sweden, and 22 in Norway (Bystrom et al. 2005).

In Turkey, 205 cases were reported over the period 1988 to 1998 (Helvaci et al. 2000), while 126 cases of disease were reported in Slovakia during the period 1985 to 1994 (Gurycová 1997). In Bulgaria, a tularemia outbreak affected 285 people from 1997 to 2005 (Kantardjiev et al. 2006). In the USA, 316 isolates from human cases were collected in the period 1964-2004 (Staples et al. 2006).

E. coli 0157/VTEC

The Shiga toxin-producing *Escherichia coli* (STEC/VTEC) of the O157 serotype can cause a serious human food-borne disease, which can lead to hemorrhagic or watery diarrhea. Particularly in children, this can be accompanied by the life-threatening hemolytic uremic syndrome. The mortality rate lies between 3–17% and up to 30% during outbreaks (Todd and Dundas 2001).

Most human cases are caused by the consumption of raw cow milk or undercooked meat, while also food products or drinks that are contaminated with cow manure can be contaminated by the pathogen. Sporadic cases have been shown to originate from direct contact with cattle or the contaminated environment (Lahti et al. 2002; Moller Nielsen et al. 2005). In an on-farm trial, the persistence of E. coli 0157/H7 in cattle and the farm environment was investigated on eight Ontario dairy farms positive for E. coli O157 during the previous year (Rahm et al. 1997). VTEC was found in composite samples from calf feeders, calf barn surfaces, cow feeders, flies, cow barn surfaces, and individual milk filters (Rahm et al. 1997). These authors did not encounter the pathogen in other environmental samples, including rodent feces. Nevertheless, it is thought that rodents can form a pathogen reservoir (Cizek et al. 2000; Van Donkersgoed et al. 2001). In an on-farm study from the Czech Republic, fresh droppings of wood mice (*A. sylvaticus*), house mice (*Mus musculus*), and Norway rats (*R. norvegicus*) were examined for VTEC. In 40% of the rats, *E. coli* was demonstrated (Cizek et al. 1999). In the USA however, samples from 300 rodents (species not specified) from 12 cattle farms were tested and no *E. coli* was found among them, while herd prevalences varied from 1.1 to 6.1% (Hancock et al. 1998).

In Denmark, wild animals living close to cattle and pig farms (four each) were examined for VTEC (Nielsen et al. 2004). Among the 260 samples from wild animals (including birds, rodents and insects) the prevalence was generally low. However, rodents carried VTEC and VTEC isolates from a Norway rat (*R. norvegicus*) were identical to cattle isolates(Nielsen et al. 2004). This suggest that rodents can carry VTEC, but it remains unclear which role they play in the transmission cycle of VTEC.

Plague

The most famous disease associated with rodent presence is probably the infection of rodent fleas with bubonic plague (caused by *Yersinia pestis*, a member of the family *Enterobacteriaceae*), resulting in many millions of casualties during its first (6th and 7th century AD), second (14th to 17th century AD) and third (late 19th and early 20th century AD) pandemics (Perry and Fetherston 1997). Natural transmission of plague to humans remains a possibility in many regions of the world, where foci exist in sylvatic rodent populations. Even today, there are an estimated 1000–3000 cases of the bubonic plague each year worldwide, mainly in Africa, the Americas, and Asia (Keeling and Gilligan 2000). Between 1967 and 1993 the fatality rate was around 10% (Perry and Fetherston 1997). A recent review highlights that although in a historical sense the number of plague cases is relatively low, the disease is still widely distributed globally, has an innate ability to spread rapidly and clinical symptoms can unfold quickly (Stenseth et al. 2008). Moreover, the effects of the previous pandemics are strongly etched in our memories and therefore the disease still generates a high fear factor. In 1994, a localized outbreak of plague in Surat, India, led to 50 deaths (Ganapati 1995) and national and international fear resulting in a collapse of tourism and trade in at a national level at an estimated cost of US$600 million (Fritz et al. 1996). Therefore the authors of this review conclude that "it would be a mistake to overlook its threat to humanity" (Stenseth et al. 2008).

The oriental rat flea (*Xenopsylla cheopis*) is the classic vector for plague. However, also other fleas are sometimes mentioned in connection with plague (Adjemian et al. 2007). Most rodents show moderate resistance to infection (as a result of previous exposure or possibly an inherent characteristic of the species or subspecies), relatively mild signs, and low mortality rate (Perry and Fetherston 1997). Some species of *Microtus* and *Peromyscus* have been suggested as maintenance hosts in western North America, while some types of mice (in Africa and Russia), gerbils (in Russia, India, Iran, South Africa, Syria, and Turkey), and voles (in Russia and Mongolia) are relatively resistant to plague and are suspected enzootic hosts (Perry and Fetherston 1997). One type of highly resistant rat (*Dipodomys* spp.) seroconverts, with few animals becoming ill and rarely dying, despite evidence of bacterial presence (Perry and Fetherston 1997). Field data from Kazakhstan showed that the incidence of clinical cases correlated with fluctuations in the abundance of its main reservoir host, the great gerbil (*Rhombomys opimus*) (Davis et al. 2004).

Clinical symptoms of bubonic plague are the following (Hoffman 1980): an abrupt onset, where a chill is followed by a temperature rise, often to 40^{o}C, and accompanied by headache, backache, restlessness, and

rapid pulse. This is followed by extreme postration and central nervous system manifestations such as anxiety, delirium, and coma or convulsions. By this time, 75–90% of patients have an enlarged, tender, nonfluctuant, hard lymph node, the bubo (Hoffman 1980). Patients should be isolated and treated with gentamicin or doxycycline (Mwengee et al. 2006), as streptomycin, tetracycline, and chloramphenicol have mostly become outdated or unavailable.

Rat-bite fever and Haverhill fever

Both rat-bite fever and Haverhill fever are caused by *Streptobacillus moniliformis.* In Asia, also *Spirillum minus* (or: sodoku) can cause rat-bite fever. Haverhill fever was originally recognized in 1926 and is an infection transmitted to humans through the consumption of contaminated water, milk, or food that has previously been in contact with rats. This disease is characterized by a high incidence of pharyngitis and pronounced vomiting. More common is rat-bite fever, which is characterized by abrupt high fevers, headaches, migratory ancylosis, vomiting and skin rash (2–4 days post infection) (Elliott 2007). Despite its worldwide distribution, rat-bite fever is rarely diagnosed by physicians. Estimates of the mortality rate of untreated cases approach 10–13% (Elliott 2007; Graves and Janda 2001) and fatality is often associated with cases in infants and patients with endocarditis. In previous studies (Graves and Janda 2001) it was demonstrated that not only rat bites, but also owners of pet rats are vulnerable of contracting the disease. The pathogen can be transferred via scratches or kissing of the animal. Most reports of *S. moniliformis* originate from the United States, although reports have also come from Brazil, Canada, Mexico, Paraguay, the United Kingdom and France, but sporadic reports from Norway, Finland, Germany, Spain, Italy, Greece, Poland, Denmark, and The Netherlands also exist. Australia has also demonstrated some cases where it has been reported in field populations of house mice (*Mus muculus domesticus*) (Taylor et al. 1994). Only few cases have been reported in Africa (Elliott 2007).

Listeriosis

Listeria are gram-positive bacteria and are named after their discoverer, the English surgeon Joseph Lister. The most renown species is *L. monocytogenes*, the causative agent of listeriosis. Indeed, this pathogen can frequently be encountered among rodents. A recent study from eastern Russia (Zaytseva et al. 2007) revealed that 1.1% of rodents screened carried this pathogen. Unfortunately, the rodent species were not specified in this study. In an earlier (military) study from the USA (Olsufev and Emelyanova 1968), it was found that the pathogen affected wild voles (*Microtus arvalis* Pall.) and water rats (*Arvicola terrestris* L.). In Japan, wild black rats (*Rattus rattus*) were collected in the city centre of Tokyo and it was demonstrated that 6.5% were infected with *L. monocytogenes* (Iida et al. 1998).

Food contaminated with *L. monocytogenes* is a significant cause of human illness and death worldwide (Mead et al. 1999). The pathogen can cause granulomatous septicemia in newborns and fatal meningitis in adults (Farber and Peterkin 1991), but is opportunistic and frequently encountered in the elderly, pregnant mothers, and AIDS patients. The case-fatality rate in recent outbreaks and sporadic cases is around 20%-30% (WHO, Geneva, http://www.who.int). Antibiotics effective against *Listeria* species include vancomycin, ampicillin, linezolid, azithromycin, and ciprofloxacin.

Parasites

Toxoplasmosis

Toxoplasma gondii is a protozoan parasite with a complex life cycle. Cats play a key-role as definitive hosts: they acquire infection from their prey species, such as rodents (intermediate hosts). Infected cats shed oocysts with their feces. Livestock animals that take up oocysts from their environment can form tissue-cysts in their meat and organs (Van Knapen 1989). If their meat is consumed without proper cooking, the parasite may still be viable and is transferred to humans (Kijlstra and Jongert 2008). This process is called acute infection and most cases via this route are asymptomatic (although the immunosuppressed form an exception). Congenital infection is another route of transmission of the disease and may have serious and sometimes even fatal consequences. This occurs when a woman is infected for the first time during pregnancy and the parasite crosses the placenta and invades the tissues of the fetus (Gilbert 2000). The severity of infection depends on the time of infection during the pregnancy: severity is the greatest during the early stage of pregnancy and infection may then result in hydrocephalus, mental retardation or even spontaneous abortion. Infection at a later stage during gestation may lead to milder symptoms such as, e.g., ocular toxoplasmosis. The risk of transmission from mother to fetus is highest during the third trimester of the gestation, when contact of maternal and fetal circulation is more likely to occur (Rothova and Kijlstra 1989). Of these infected children, only a part will perceive clinical symptoms at birth. At a later age however, the disease may manifest itself and result in eye lesions.

Rodents have been shown to carry *Toxoplasma gondii* (Dubey et al. 1995; Hejlicek et al. 1997; Kijlstra et al. 2008; Smith and Frenkel 1995; Tizard et al. 1978), and they are

therefore associated with transmission of this parasite to pigs (Kijlstra et al. 2004; Meerburg 2006; Meerburg and Kijlstra 2006; Weigel et al. 1995). It remains unclear whether the role of rodents is to provide a constant reservoir of infection for cats or whether they directly transmit the disease to pigs, if the latter consume dead rodent carcasses or even live rodents (Kijlstra et al. 2008).

Babesiosis

Babesiosis is an uncommon parasitic disease caused by piroplasms, protozoan parasites of the *Babesia* genus (Homer et al. 2000). About 70 different types of *Babesia* protozoa exist. The pathogen is usually transmitted by ticks, sometimes in conjunction with Lyme disease as is the case with *Babesia microti* that uses the same tick vector, *Ixodes scapularis* (Benson et al. 2004). Moreover, in babesia-endemic areas, the organism can also be transmitted by blood transfusion (Leiby 2006; Wei et al. 2001). Depending on the Babesia strain, infection may vary from aymptomatic (as is often the case with *B. microti* in the USA and Japan), or cause a mild non-specific illness (*B. divergens* in Europe). Sometimes, infection results in severe disease, especially in young (neonatals), the elder and immunosuppressed people, and fatalities do occur (Hatcher et al. 2001). In regions where malaria is present, symptoms are often confused with that of *Plasmodium* infection. Due to its clinical appearance, the disease is often underdiagnosed and underreported.

Rodents can be reservoirs of the infection, especially for infection with *B. microti*. This species is commonly distributed throughout North America (Goethert et al. 2003), Eurasia (Zamoto et al. 2004), Europe (Casati et al. 2006; Duh et al. 2003), and Japan (Tsuji et al. 2001), and can cause human disease. After six human cases of clinical babesiosis were diagnosed, examination of rodents on Nantucket Island, USA disclosed infections with *Babesia microti* in field mice (*Microtus pensylvanicus*) and in white-footed mice (*Peromyscus leucopus*) (Healy et al. 1976). After a human case of babesiosis in Taiwan, three species of rats (black rats, *R. rattus*, Norway rats, *R. norvegicus,* and spiny rats, *R. coxinga*) from areas around the patient's neighborhood were trapped and examined for the prevalence of *B. microti* infection by routine techniques of examining blood smears and inoculating blood into hamsters. The infection rate was about 67% and 83% as determined by examining blood smears and inoculating hamsters, respectively (Shih et al. 1997). In Japan (Shiota et al. 1984), parasite presence was demonstrated in Japanese field rodents, (*A. speciosus*) and (*Apodemus argenteus*). In a study from Poland (Karbowiak 2004), prevalence of infection for *B. microti* in *Microtus arvalis* varied from 9 to 33%, in *M. agrestis* it was almost 50% and in *M. oeconomus* between 7 and 50%. *Myodes spp.* voles and *Apodemus spp.* mice played an inferior role as zoonotic reservoir for *Babesia microti*. In Slovakia (Duh et al. 2003), prevalence among yellow-necked mice (*A. flavicollis*) was 12% and prevalence among bank voles (*M. glareolus*) 16%. Recently, in Thailand a new type of rodent babesia (Dantrakool et al. 2004) that seems to be phylogenetically closest to the canine *B. canis*, was found in bandicoot rats (*Bandicota indica*). Whether this species can also infect humans, as is the case with *B. microti* is not known yet.

Cryptosporidiosis

Cryptosporidiosis is a diarrheal disease that is caused by microscopic parasites of the genus *Cryptosporidium*. *Cryptosporidium* thrives in the intestinal tracts of infected humans or animals. As a consequence, the parasite is found in soil, food, water, or surfaces that have been contaminated with infected human or animal feces. Major outbreaks are often associated with drinking of infected water.

Symptoms of cryptosporidiosis generally begin 2–10 days (average 7 days) after becoming infected with the parasite. In persons with a healthy immune system, symptoms (which could include watery diarrhea, stomach cramps, dehydration, nausea, vomiting, fever, weight loss) usually last about 1–2 weeks (WHO, Geneva, http://www.who.int). However, in the immunosuppressed the disease can lead to more severe problems, which is a major treatment problem in AIDS patients.

Moreover, cryptosporidiosis has emerged as a major cause of neonatal mortality in livestock, principally in lambs and calves, thus causing economic losses for farmers.

Rodents seem an important source of cryptosporidial oocysts, especially in sylvatic and bushy areas. There, they impose the highest risk for infection in humans. A survey in Spain showed that *Cryptosporidium* is present in various rodents, such as the wood mouse (*A. sylvaticus*), the yellow-necked mouse (*A. flavicollis*), the Algerian mouse (*Mus spretus*), the black rat (*R. rattus*), and the bank vole (*Myodes glareolus*) (Torres et al. 2000). Studies in the USA (Klesius et al. 1986) and the United Kingdom (Chalmers et al. 1997) reported that the house mouse (*M. musculus*) can be infected by *Cryptosporidium* and that prevalences vary between 22–30%. In the UK, prevalence in wood mice (*Apodemus sylvaticus*) was 22% (Chalmers et al. 1997). On Skomer Island, a part of the UK, prevalence in Skomer bank voles (*Myodes glareolus skomerensis*) for *Cryptosporidium* was 51% (Bull et al. 1998). In Finland, the prevalence of cryptosporidia was determined in high density populations of *Microtus agrestis* and *Myodes glareolus* (Laakkonen et al. 1994). Prevalence in field voles (*Microtus agrestis*) and bank voles (*Myodes glareolus*) was 2%, while in root voles (*Microtus oeconomus*) no infection was

found (Laakkonen et al. 1994). On farms in the United Kingdom (Webster and Macdonald 1995) 63% of the Norway rats (*R. norvegicus*) was infected, while in Japan infection rates in the same species varied between 2-21% (Iseki 1986; Miyaji et al. 1989; Yamura et al. 1990). In black rats in Japan, rates varied between 0–49% (Iseki 1986; Miyaji et al. 1989; Yamura et al. 1990). In Australia, the 'cattle' genotype of *Cryptosporidium* was isolated in 5 mice trapped on wheat-sheep farms. These and earlier studies indicate that sheep and cattle may transmit the 'cattle' genotype to mice. Subsequently mice may transmit *Cryptosporidium* to other domestic animals and to humans (Morgan et al. 1999).

Rodents can share their habitat with farm animals, or travel through grazing land used by them, enabling ample opportunity for transmission of *Cryptosporidia* from them to the farm animals. Moreover, rodents are found commonly in urban areas, thus providing a link between rural and urban disease foci. They could contribute to many of the sporadic human cases of cryptosporidiosis in towns and cities, by leaving their many small droppings wherever they forage, thus contaminating human and animal feed stores and accommodation (Sturdee et al. 1999).

Chagas' disease

Chagas' disease is a tropical parasitic disease which occurs in the Americas, particularly in large parts of South America. It is caused by a flagellate *Trypanosoma cruzi* that is transmitted to humans and other mammals mostly by blood-sucking assassin triatomines of the subfamily *Triatominae* (Family *Reduviidae*). These hemipteran insects deposits parasite-laden feces on the skin, while feeding.

Chagas' disease currently affects 16–18 million people, with some 100 million (25% of population in Latin America) at risk of acquiring the disease (WHO, Geneva, http://www.who.int) killing around 50,000 people annually. Secondary transmission routes include blood transfusion, congenital infection, tissue transplants, and food-borne transmission (WHO, Geneva, http://www.who.int).

The initial phase is acute and can be characterized by patent parasitemia during 40–60 days. Because clinical symptoms are generally mild (except for severe neurological complications in children) and atypical, the infection with *T. cruzi* is often not recognized. After the acute phase, patients enter the indeterminate form of the chronic phase that may last for several years or persist indefinitely (Buscaglia and Di Noia 2003). During this long interval, infected persons themselves also form a parasite reservoir. Up to 20 years after infection, 35% of the patients will develop pathological signs such as cardiomyopathy, peripheral nervous system damage or dysfunction of the digestive tract often leading to megaesophagus and/or megacolon (Buscaglia and Di Noia 2003). During this period, most fatalities occur.

It is thought that rodents can act as a disease reservoir, although the exact domestic/peridomestic and sylvatic transmission cycles of *Trypanosoma cruzi* remain unclear. However, the parasite was found in many rodent species such as: black rats (*R. rattus*), brown rats (*R. norvegicus*), house mice (*M. musculus*), wood rats (*Neotoma micropus canescens*), the hispid pocket mice (*Perognathus hispidus*), Mexican spiny pocket mice (*Liomys irroratus*), Northern grasshopper mice (*Onychomys leucogaster*), white troated wood rats (*Neotoma albigula albigula*), rusty antelope squirrels (*Citellus leucurus cinnamoneus*), pygmy mice (*Baiomys musculus*), Jaliscan cotton rats (*Sigmodon mascotensis*) and fulves, harvest mice (*Reithrodontomys fulvescens*), and the punaré (*Trichomys apereoides*) (Burkholder et al. 1980; Cortez et al. 2006; Herrera and Urdaneta-Morales 1997; Herrera et al. 2005; Mota et al. 2007; Wood 1949; Wood and Wood 1961). Some triatomine species are associated with wild nesting vertebrates (Ramsey et al. 2000; Usinger et al. 1966).

Leishmaniasis

Leishmaniasis is caused by the protozoa *Leishmania*. The leishmaniases can be divided into 3 major clinical syndromes: cutaneous (CL), mucocutaneous (MCL),and visceral leishmaniasis (VL), or kala-azar. The pathogen is transmitted by the bite of an infected female sandfly (*Phlebotomus* sp. in the Old World and *Lutzomyia* sp. in the New World), and humans are mostly incidental hosts. The result of infection in humans can vary from a chronic skin ulcer, to erosive mucosal disease with progressive destruction of the nasopharynx and severe facial disfigurement in case of CL, to a life-threatening systemic infection with hepato-splenomegly in case of VL (Hepburn 2000).

VL is caused by *Leishmania donovani* in India and Eastern Africa and by *L. infantum chagasi* in the Mediterranean basin, western Africa and Latin America (WHO, Geneva, http://www.who.int). In contrast to CL and MCL, in which the protozoan parasite is histologically localized, visceral disease is caused by parasite growth within reticuloendothelial cells throughout the body.

Leishmaniasis is endemic from northern Argentina to southern Texas (not in Uruguay, Chile, or Canada), in southern Europe, Asia (not southeast Asia), the Middle East, and Africa (particularly east and north Africa, with sporadic cases elsewhere), but not in Australia or Oceania (Herwaldt 1999). In total, about 350 million people are at-risk (Herwaldt 1999).There are about 500,000 cases of visceral leishmaniasis each year; over 90% of worldwide cases are in Bangladesh, northeastern

India (particularly Bihar State), Nepal, and Sudan (Old World), and in northeastern Brazil (New World) (Herwaldt 1999). If clinically evident but untreated, visceral leishmaniasis causes life-threatening systemic infection (Herwaldt 1999).

There are 1–1.5 million new cases of cutaneous leishamaniasis each year (Desjeux 2004) of which more than 90% occur in Afghanistan, Algeria, Iran, Iraq, Saudi Arabia and Syria, in the 'Old World,' and Brazil and Peru in the 'New World' (Hepburn 2000). In the Old World, cutaneous leishmaniasis is caused by *Leishmania* tropica in urban areas and *Leishmania major* in dry desert areas. In the New World, *Leishmania leishmania (*e.g., *Leishmania amazonensis, Leishmania mexicana, Leishmania chagasi)* and *Leishmania viannia (*e.g.*, Leishmania braziliensis, Leishmania guyanensis, Leishmania panamensis)* can cause the disease.

The pathogen is most prevalent in rural areas or forests, with a moist climate in which the sand flies thrive. Rodents can form a reservoir for the protozoa. In Mexico, the protozoa was found in the Yucatan deer mouse (*Peromyscus yucatanicus*) and the black-eared rice rat (*Oryzomys melanotis*) (Chable-Santos et al. 1995). *Leishmania panamensis* was also reported in black rats (*R. rattus*) in Brazil (Vasconcelos et al. 1994). Moreover, spiny rats (*Proechimys* spp.) appear to be reservoir hosts of *L. amazonensis* in Brazil and French Guyana (Ashford 1996). Also, the paca (*Cuniculus paca*) is said to be the reservoir host of a little known pathogen species *L. lainsoni* (Ashford 1996). In Belize, the climbing rat (*Ototylomys phyllotis*) is the principal host of *Leishmania mexicana* (Disney 1968), with 53–56% of the captured rodents infected. In the southern USA, the woodrat (*Neotoma micropus*) forms a reservoir for *L. mexicana* in Texas (Kerr et al. 1995). In Jordan, 23% of the fat sand rats (*Psammomys obesus*) were carrying the pathogen *L. major* (Saliba et al. 1994). In Israel, the fat sand rat (*P. obesus)*, Sundevall's gerbil (*Meriones crassus*) and probably the short-tailed bandicoot rat (*Nesokia indica*) function as reservoirs for *L. major* (Schlein et al. 1984). The same is happening in the Nile grass rat (*Arvicanthis niloticus luctuosus*) in Sudan (Hoogstraal and Heyneman 1969) and in Libyan jirds (*Meriones libycus*) and great gerbils (*Rhombomys opimus*) in Iran (Yaghoobi-Ershadi et al. 1996) In Egypt, the great Egyptian gerbil (*Gerbillus pyramidum*) are carriers of *L. major* (Fryauff et al. 1993). In the old world, black rats (*R. rattus*) are mentioned as incidental carriers of *L. donovani* (Ashford 1996). In conclusion, it can be said that rodents play an important role both as reservoirs and incidental hosts for this protozoa.

Giardiasis

Giardiasis is an infection of the intestines that is caused by *Giardia lamblia*, a flagellated protozoan parasite (Adam 1991) that is present throughout the world. One of the important symptoms of the disease is diarrhea, both in man and in animals. Other symptoms include loss of appetite, lethargy, fever, stomach problems (cramps), projectile vomiting (rare), bloating, and flatulence. Symptoms typically begin 1–2 weeks after infection and may wane and reappear cyclically (Adam 1991). Symptoms are caused by the thick coating of *Giardia* organisms coating the inside of the small intestine and blocking nutrient absorption. Most cases are asymptomatic; only about a third of infected people exhibit symptoms. Untreated, symptoms may last for six weeks or longer.

Humans can get infected through consumption of contaminated food products or infected water, also during swimming. Giardiasis usually occurs sporadically, although outbreaks do occur.

Rodents may form a pathogen reservoir and could cause contamination of the water with *G. lamblia cysts* (Appelbee et al. 2005; Bednarska et al. 2007). Beavers and muskrats have been reported as carriers of this parasite (Frost et al. 1980). Voles also seem to be an important reservoir (Wallis et al. 1984): red-back voles (*C. gapperi* Vigors), meadow voles (*M. pennsilvanicus*), water voles (*M. richardsoni*), and the long-tailed voles (*M. longicaudus*) have been identified as carriers with high parasite prevalence (Pacha et al. 1987; Wallis et al. 1984). Less important, though still carriers, are deer mice (*P. maniculatus*) (Wallis et al. 1984) and yellow-necked mice (*A. flavicollis*) (Karanis et al. 1996).

Taeniasis

Taeniidae are highly characteristic tapeworms in the subfamilies Taeniinae and Echinococcinae. Adults are present in the intestine of carnivorous and omnivorous mammals, including humans, throughout the world. Life cycles for Taenia are indirect and consistently involve two mammalian hosts, a carnivorous or omnivorous definitive host (e.g. canids, felids, viverrids, mustelids, hyaenids, and humans) and a herbivorous intermediate host (principally artiodactyls, rodents, and lagomorphs) (Hoberg 2002).

The mature tapeworms are 40–100 cm long and inhibit the small intestines of the definitive host (Krauss et al. 2003). This infection is called taeniasis. Humans are the definitive hosts for *Taenia solium* (the pork tapeworm) and *T. saginata* (the beef tapeworm). Humans are also the definitive hosts for *T. asiatica*, a newly recognized tapeworm found in Asia (Anonymous 2005). Animals are the definitive hosts for *T. crassiceps*, *T. ovis*, *T. taeniaeformis*, *T. hydatigena*, *T. multiceps*, *T. serialis,* and *T.* brauni (Anonymous 2005). Taenia larvae are found in the muscles, central nervous system (CNS), and other tissues of the intermediate hosts. Larvae are

more likely to cause disease than the adult tapeworms. There are two forms of larval infection, cysticercosis and coenurosis (Anonymous 2005).

Infection with the larvae of *Taenia solium*, *T. saginata*, *T. crassiceps*, *T. ovis*, *T. taeniaeformis,* or *T. hydatigena* is called cysticercosis. The larvae of these organisms are called cysticerci (Anonymous 2005). Humans are intermediate hosts for *T. solium*, *T. crassiceps*, *T. ovis*, *T. taeniaeformis,* and *T. hydatigena*. *T. solium* is often found in humans; the other four species are very rare (Anonymous 2005). *T. solium* is the only *Taenia* species for which humans are both the definitive and an intermediate host. Animals can be intermediate hosts for these five species as well as for *T. saginata* and *T. asiatica* (Anonymous 2005).

Infection with the larval forms of *T. multiceps*, *T. serialis* and *T. brauni* is called coenurosis. Human coenurosis is much less common than cysticercosis. Approximately 100 or more cases have been reported worldwide (Anonymous 2005), mainly in Africa (Hoberg 2002) and South America. Only a few cases have been documented in the United States and Europe (Anonymous 2005). Humans can be intermediate hosts for *T. multiceps*, *T. serialis,* and *T. brauni*. Animals can also be intermediate hosts for these three species (Anonymous 2005).

In humans, taeniasis is caused by eating inadequately cooked pork (*T. solium* and *T. asiatica*) or beef (*T. saginata*) (Anonymous 2005). In animals, taeniasis is caused by *T. crassiceps*, *T. ovis*, *T. taeniaeformis*, *T. hydatigena*, *T. multiceps*, *T. serialis,* and *T. brauni* and is acquired by eating tissues from a variety of intermediate hosts including ruminants, rabbits and rodents (Anonymous 2005). Eggs are shed in proglottids with the feces of definitive hosts and are ingested by the intermediate hosts, allowing the oncospheres to hatch in the intestine (Krauss et al. 2003). Humans usually ingest tapeworm eggs on fruits and vegetables or acquire them directly from the soil. They can also acquire infection by consumption of contaminated water (Anonymous 2005).

Worldwide, taeniasis and cysticercosis are common parasitic infections: 2-3 million people are thought to be infected with adult *T. solium*, 45 million with adult *T. saginata*, and 50 million with *T. solium* cysticerci. An estimated 50,000 people die annually from the CNS or cardiac complications (Anonymous 2005).

The level of involvement of rodents in human infection depends on the *Taenia* species that is contracted. Rodents (and also lagomorphs) are intermediate hosts of metacestodes of *T. multiceps* and *T. serialis* and contribute to the life cycle of this parasite. *T. taeniaeformis* is a parasite that is present in rodent intermediate and felid definitive hosts, but there are also reports of human infections from Argentina, Japan and Sri Lanka (Ekanayake et al. 1999; Sterba and Barus 1976). *T. crassiceps* usually develops in rodents, while the adult form of the cestode can be found in foxes. For *T. brauni*, wild rodents (gerbils) are the usual intermediate hosts (Anonymous 2005). There are cases reported of subcutaneous and muscular tissues invasion by proliferative larval forms of *Taenia crassiceps* in AIDS patients with severe immunodeficiency in France (Chermette et al. 1995; Francois et al. 1998).

Rodentolepiasis

The bile duct cestode of mice and rats, *Rodentolepis* (= *Hymenolepis*) *microstoma* was only recently encountered in humans, during a survey of gastro-intestinal parasites in remote communities in north-western Western Australia (Macnish et al. 2003; Spratt 2005) There, four human cases of mixed infection with *R. microstoma* and *R. nana*, the latter being the more common enteric parasite, were reported. *Rodentolepis microstoma* has an indirect life cycle using beetles as obligatory intermediate hosts (Spratt 2005). The parasite is normally maintained in lab colonies using the flour beetle, *Tribolium confusum*, and there has been no evidence of direct transmission between experimental animals in laboratories where the parasite has been passaged for many years. Spratt (2005) has found natural infections in the native beetle, *Pterohelaeus darlingensis*, in grain-growing regions of the Darling Downs, Queensland, where mouse plagues occur.

He concludes that those four individuals may have accidentally ingested an infected intermediate host, one that is normally a dietary component of the normal rodent definitive host (Spratt 2005). Fortunately, it was found that the human strain of *R. nana* is essentially non-infective to rodents (Macnish et al. 2002) which might further disturb the life cycle of this pathogen.

Echinococcosis

Rodents contribute to the lifecycle of *Echinococcus multilocularis*, a tapeworm of 1–4 millimeters. Wild foxes, coyotes, dogs, and cats are definitive hosts and get infected when they eat *E. multilocularis* larvae in infected rodents. In total 6 genera of Cricetidae, 3 genera of Muridae and 7 genera of Arvicolidae have been recorded as susceptible species (Zhou 2001). Among them, the field mouse (*Microtus arvalis*) (Zhou 2001), lacustrine voles (*Microtus limnophilus*) (Craig et al. 2000) and water voles (*Arvicola terrestris* Scherman) (Viel et al. 1999). Musk rats (*Ondatra zibethicus*) can also be infected with these larval cestodes (Borgsteede et al. 2003). Alveolar Echinococcosis (AE) is widely distributed, occurring mostly in northern latitudes. Cases have been reported in central Europe, Russia, Central Asia, China, Japan, and North America. Although the prevalence among foxes and other infected mammals

can be up to 50%, transmission to humans seldom occurs (Gottstein et al. 2001). Humans acquire infection by accidentally swallowing the eggs of the tapeworm, e.g., by consumption of contaminated food items (such as wild-growing blackberries) or by handling infected domestic animals. However, when infected, infection causes parasitic tumors to form in the liver, and, less commonly, the lungs, brain, and other organs (Basset et al. 1998). If left untreated, infection with AE can result in fatalities in 80% of the cases (Wilson et al. 1995). Treatment is possible with benzimidazoles (Reuter et al. 2000), which have a parasitostatic effect.

Changes in the landscape can have effects on host-pathogen interaction. In China, deforestation has taken place driven by the need for agricultural land. This in turn results in creation of optimal peri-domestic habitats for rodents that serve as intermediate host species for the spreading of *E. multilocularis* (such as *M. limnophilus*) and subsequent development of a peri-domestic cycle involving dogs (Craig et al. 2000).

E. vogeli is the agent of the polycystic hydatidosis, and rodents are an intermediate host. The larval stage proliferates externally from the germinal layer and forms septa within the cyst generating microcysts. It is endemic in Central and South America. Until now at least 106 human cases have been reported (Tappe et al. 2008).

Rodents are not involved in the transmission cycle of *Echincoccus granulosus*, which takes place between men, sheep and dogs (Torgerson and Heath 2003).

Schistosomiasis

Schistosomiasis or bilharzia is a parasitic disease that is caused by several species of flatworm. The disease can be encountered in tropical countries in the Caribbean, the eastern part of South America, East Asia, Africa, and in the Middle East. *Schistosoma mansoni* is found in the Caribbean, in parts of South America, Africa, and the Middle East; S. *haematobium* in Africa and the Middle East; and *S. japonicum* in the Far East. *S. mekongi* and *S. intercalatum* are found focally in Southeast Asia and central West Africa, respectively. According to WHO-estimations 207 million people have contracted the disease, of which 120 million symptomatic (WHO, Geneva, http://www.who.int). Above all, schistosomiasis is a chronic disease. The pathological signs of *S. mansoni* and *S. japonicum* schistosomiasis include Katayama fever, hepatic perisinusoidal egg granulomas, Symmers' pipe stem periportal fibrosis, portal hypertension, and occasional embolic egg granulomas in brain or spinal cord. Pathology of schistosomiasis by *S. haematobium* can include hematuria, scarring, calcification, squamous cell carcinoma, and occasional embolic egg granulomas in brain or spinal cord. The number of bladder cancer cases and mortality is generally elevated in affected areas (Mostafa et al. 1999).

The most common way of acquiring schistosomiasis in developing countries is by wading or swimming in lakes, ponds, and other bodies of water which are infested with snails (usually of the *Biomphalaria*, *Bulinus*, or *Oncomelania* genus) that are the natural reservoirs of the *Schistosoma* pathogen. Although the definitive host is usually man, numerous other mammalian species have been also found infected by this parasite. Of these, rodents are most commonly affected (Duplantier and Sene 2006). Factors that could explain the existence of *Schistosoma* worms in a rodent are: a habitat in humid areas, and activity rhythms corresponding to emergence of cercariae and immunological compatibility (Duplantier and Sene 2000). In a study in Senegal, *A. niloticus* and *M. huberti*, were found to be infected with *S. mansoni* (Duplantier and Sène 2000) with prevalences of 5.5% and 4.5% respectively. Prevalences of 59% for *Rattus rattus* and 80% for *R. norvegicus* have been reported in Guadeloupe (Alarcon de Noya et al. 1997). In Brazil, prevalences of about 50% have been recorded in several foci (Rey 1993) and two rodents of with aquatic habitats were mainly mentioned: the water rat (*Nectomys squamipes*) and the web-footed marsh rat (*Holochilus brasiliensis*). Also other species of rodents can be infected (Duplantier and Sene 2006). However, the exact contribution of rodents to disease burden in humans remains difficult to quantify.

Human fasciolosis

Human fasciolosis is caused by the trematodes *Fasciola hepatica* (also known as liver fluke) and *Fasciola gigantica*, two parasites of herbivores which can accidently cause infections in humans. The parasite has a very broad host range, which includes many mammals, including humans. Infection of livestock induces productivity losses in terms of reduced production of meat, milk, and wool. Human infections with *F. hepatica* occur where sheep and cattle are reared, and where humans consume raw watercress, including Europe, the Middle East, and Asia. Infections with *F. gigantica* have been reported, more rarely, in Asia, Africa, and Hawaii (CDC, Atlanta, http://www.cdc.gov). The WHO has recognized fasciolosis as an emerging human disease; it is estimated that currently 2.4 million people are infected with the parasite, while 180 million people in 61 different countries are at-risk of infection (Haseeb et al. 2002). Worldwide losses in animal productivity due to *Fasciola spp*. were estimated at USD 3.2 billion per annum (Spithill et al. 1999).

Freshwater snails are an intermediate host of the parasite. Rodents are probably a reservoir and can function as definitive hosts. In France, presence of *F. hepatica* was

detected in the large rodent, nutria (*M. coypus*), and in the lagomorph domestic rabbit (*O. cuniculus*). Infection rates were respectively 55% and 34% (Ménard et al. 2000). In another study in France, liver fluke prevalence in nutrias varied from 8.7% in regions where no *F. hepatica* was reported, to 40.1% in fasciolosis areas (Ménard et al. 2001). In Argentina (Kleiman et al. 2004), the European hare (*Lepus europaeus*) demonstrates low prevalence (0.08%). Nevertheless, according to these authors, the European hare is still able to play a significant role in the parasite transmission cycle due to the high number of eggs shed into the environment, whenever it shares its habitat with livestock and snails, and if it has a high abundance. In Belorussia (Shimalov 2000) the prevalence was 9.1% in the European hare (*L. europaeus*). In Australia, bush rats (*Rattus fuscipes*) had a prevalence of 4% (Spratt and Presidente 1981). High prevalence of over >40% has been reported in black rats (*Rattus rattus*) on the Mediterranean island of Corsica (Mas-Coma et al. 1988).

The size of *F. hepatica* eggs that are shed differ among host species: murid liver flukes were smaller than Corsican cattle flukes (Valero et al. 2002). Moreover, egg shedding by black rats (*R. rattus*) showed a lower average number of eggs per fluke per day (850–2,150) than the range normally found in sheep (8,800–25,000) or cattle (10,000–12,000), but much higher than the values found in rabbits (19–69) (Valero et al. 2002). Thus, rodents may play a role in the maintenance and the dissemination of *F. hepatica* in various environments.

As mentioned before, fasciolosis causes important economic losses in the cattle and sheep industry, and its present control is primarily based upon application of anti-helminthic drugs and pasture management. However, during the last decades drug resistant *F. hepatica* strains have emerged. Vaccination, which is seen as a non-chemical alternative for control of fasciolosis, is therefore being developed (Reszka et al. 2005).

Brachylaimiasis

Some genera of rodents (*Rattus*, *Eliomys*, *Mus* (Mas-Coma and Montoliu 1986)) are the definitive hosts of several trematodes of the Brachylaima family (Trematoda: *Brachylaimidae*), while the intermediate hosts can be two different kinds of snails. In Spain, operculated, assymetric, embryonated eggs were passed with feces of wild Norway and black rats (Gracenea et al. 2002). Also house mice can be infected with these trematodes (Butcher and Grove 2005; Milazzo et al. 2003; Tattersall et al. 1994). In the semi-arid areas of South Australia trematode eggs of *Brachylaima cribbi* were reported in the feces of two 21-month-old children, with abdominal pain and diarrhea. These children were reported to have consumed snails. Apparently, brachylaimid trematodes can infect humans and thus potentially cause fatal infection (Spratt 2005).

Alariasis

Alariasis is caused by the intestinal fluke *Alaria* spp. These trematodes create few, if any, clinical signs. Canids (Shoop et al. 1989) and mustelids (Swanson and Erickson 1946) contract this parasite when they eat infected aquatic animals, such as frogs (Koprivnikar et al. 2006), tadpoles, or snakes. Rodents can act as paratenic hosts of metacercariae. More rarely, the same applies to humans (Shoop 1994). However, some fatalities are reported in humans as well (Fernandes et al. 1976; Freeman et al. 1976).

Echinostomiasis

Human echinostomiasis is caused by the ingestion of metacercariae of digenean trematodes of the family Echinostomatidae in raw or undercooked mollusks, fish, crustaceans, and amphibians (tadpoles or frogs) from fresh or brackish water (Graczyk and Fried 1998; Zhou et al. 2008). The disease is endemic to southeast Asia and the Far East, i.e., mainland China, Taiwan, India, Korea, Malaysia, Philippines, and Indonesia (Huffman and Fried 1990). At least 16 species of these trematodes can cause disease in humans (Graczyk and Fried 1998). Rodents can be definitive hosts: rats are natural hosts for *E. caproni* (Jeyarasingam et al. 1972). Others report the distribution of *E. trivolvis* and *E. caproni* in rodents and domestic poultry (Huffman and Fried 1990). Some suggest that rodents are also the natural hosts of *E. paraense* (Huffman 2000). Experimentally, rodents can be infected with *E. hortense* (Saito 1984) and *E. ilocanum* (Lie and Nasemary 1973). Generally, there is limited proof for direct transmission between rodents and humans. However, it seems likely that rodents contribute to the presence of these trematodes in the environment.

Trichinosis

Roundworms of the genus *Trichinella* can be divided in two main groups (Rosa et al. 2003): (1) species with encapsulated larvae in host muscles (*T. spiralis*, *T. nativa*, *T. britovi*, *T.murrelli,* and *T. nelsoni*); and (2) species with non-encapsulated larvae in host muscles (*T. pseudospiralis*, *T. papuae,* and *T. zimbabwensis*).

The distribution of *T. spiralis* has been strongly influenced by the passive introduction of this pathogen by domestic pigs and synanthropic rats (rats associated with human presence) on different continents, followed by the transmission to wildlife (Pozio 2005). It is now known that this parasite is present in many countries of

North, Central and South America, Europe, Asia, Egypt (Africa), Indonesia, and New Zealand (Pozio 2005).

It is possible for humans to contract trichinosis by consuming inadequately cooked meat from certain wildlife species, especially bears, as well as meat products (such as pork or beef sausage) to which game meat has been added; several local outbreaks were caused in this way (Schmitt et al. 1978). Another hazard of unknown potential is the spread of trichinosis from the wild animal reservoir (particularly rodents) to domestic pigs and thus to man (Schmitt et al. 1978).

Upon human infection, the patient will experience nausea, heartburn, dyspepsia, and diarrhea within 1–2 days, while the severity of symptoms depends on the number of worms ingested. Later, if the worms start to encyst in different body parts, other manifestations might occur (headaches, fevers, muscle pain, etc.). If worms enter the heart or central nervous system, this can be fatal.

Although a large number of rodent species is able to carry *Trichinella* (e.g., *Microtus pennsylvanicus*, *Sigmodon hispidus*, *Peromyscus leucopus,* and *Mus musculus* (Holliman and Meade 1980)), it remains largely unknown what species of the parasite they carry (Pozio 2005), and thus, whether a sylvatic cycle of the disease exists independently and poses a potential threat to human health.

Capillariasis

Rodents belonging to many genera (genera *Actomys*, *Akodon*, *Apodemus*, *Arvicanthis*, *Arvicola*, *Bandicota*, *Castor*, *Citellus*, *Cricetomys*, *Cricetulus*, *Cynomys*, *Dasymys*, *Ellobius*, *Geomys*, *Gerbillus*, *Lemmus*, *Lemniscomys*, *Marmota*, *Microtus*, *Mus*, *Myodes*, *Myopotamus*, *Napeozapus*, *Neotoma*, *Ondatra*, *Otomys*, *Peromyscus*, *Rattus*, *Sciurus*, *Sigmodon*, *Suncus*, *Synaptomys*, *Tatera,* and *Thomomys* (R. Ceruti 2001; Spratt and Singleton 2001)) can be definitive hosts of *C. hepatica*, a tiny nematode which belongs to the superfamily *Tichinelloidea*, subfamily *Capillariinae* (Anderson and Bain 1982). Prevalence in rodents may exceed 80% (Krauss et al. 2003). In their liver, adult worms deposit their eggs. If this host is eaten by a cat or dog, the eggs pass with the animal feces. Eggs in the soil are eaten by humans and hatch in the small intestine. The larvae penetrate the intestinal wall and migrate to the liver, where they mature. *C. hepatica* in adults presents the clinical picture of acute or subacute hepatitis with hypereosinophilia. However, infection often occurs in children aged 1–5 because they may ingest soil (Spratt and Singleton 2001).

About 50 proven cases of human hepatic capillariasis have been recorded (Krauss et al. 2003) from many different countries (Brazil (Sawamura et al. 1999), the Czech Republic, Slovakia, India, Italy, Korea, Mexico, Nigeria, South Africa, Switzerland, Turkey, the United States, and former Yugoslavia). In some cases, species other than *C. hepaticum* from the subfamily *Capillariinae* may have been responsible (Spratt and Singleton 2001).

Angiostrongylosis

Nematodes of the subgenus *Parastrongylus* of the genus *Angiostrongylus* belong to the family *Metastrongyloidae* that use rodents as definitive hosts and gastropods as intermediate hosts. They occur worldwide. At least 20 species of *Angiostrongylus* are described, but only two of those are causing human infections (Casanova et al. 2006). *Angiostrongylus cantonensis* can cause a severe human neurological disorder. This lungworm is enzootic in the lungs of many species of rodents in Southeast Asia, throughout the Pacific region and in eastern Australia (Spratt 2005).

Humans can acquire infection by ingesting tissues of raw or undercooked infected intermediate or paratenic hosts, or by eating salad materials, especially lettuce, contaminated with the slime from intermediate hosts containing larvae which have escaped in this secretion. Especially young children are at risk, given their tendency to indiscriminately place objects in their mouths, and their small size relative to the infecting dose in another example of humans appropriating a food chain with potentially fatal consequences (Spratt 2005).

The other species of lungworm that can cause infection and lead to a disease called abdominal angiostrongylosis is *Angiostrongylus costaricensis* (Morera and Céspedes 1971). Human infections have been detected in Central and South America (Casanova et al. 2006).

Toxascariasis (Toxascaris leonina)

Cats, dogs, foxes, and other wild carnivores (Loos-Frank and Zeyhle 1982; Martínez-Moreno et al. 2007; McTier et al. 2000; Richards et al. 1995; Yamaguchi et al. 1996) are definitive hosts of the roundworm *Toxasacaris leonina*. After such a carnivore ingests the infective eggs, these hatch and the larvae mature in the small intestine. Then, adult worms produce eggs that are passed via feces to the environment. Rodents can be intermediate hosts of these roundworms. The rodent ingests the eggs, the eggs hatch, and the larvae migrate through the tissues of the rodent. If a carnivore eats the mouse, the larvae are released in its digestive system thus closing the parasite lifecycle.

In rare cases, humans acquire infection by direct ingestion of the eggs, especially kids by handling of infected kittens or puppies. It is a cause of visceral larva migrans in children, though less frequently implicated than is *Toxocara canis* (see below).

Baylisascariasis

The raccoon roundworm, *Baylisascaris procyonis*, is the most common and widespread cause of clinical larva migrans in animals (Gavin et al. 2005). In addition, it is increasingly recognized as a cause of devastating or fatal neural larva migrans in infants and young children and ocular larva migrans in adults (Gavin et al. 2005). Humans acquire infection by accidental ingestion of eggs from raccoon latrines or articles (soil, vegetables) contaminated with their feces (Gavin et al. 2005). Most severely, *B. procyonis* can be a cause of rare fatal or neurologically devastating neural larva migrans in infants and young children (Fox et al. 1985). *B. procyonis* causes fatal neurological infection in grey squirrels and several rodent families (*Peromyscus*, *Mus,* and *Sigmidon*). It is an increasingly common zoonosis in children in North America (personal comment DM Spratt). Risk assessments will depend on which species are infecting humans and whether or not there is neurological involvement.

Rodents are intermediate hosts of the parasite as they are known to forage at preferred sites of raccoon defecation. In these hosts, larvae penetrate the gut wall and migrate to various tissues, while forming cysts. If the infected rodent is then eaten by a raccoon, larvae develop into egg-laying worms in the small intestines of the latter (Tiner 1953). The eggs are then passed via the feces into the environment (Gavin et al. 2005).

Aelurostrongylosis

The lungworm *Aelurostrongylus abstrusus* is a common parasite of carnivores in some countries in Southern Europe, North America, Africa, and Australia (McGlade et al. 2003). The intermediate hosts are some species of snails and slugs, but definitive hosts (e.g., domestic cats) are probably infected by paratenic hosts such as rodents, small birds, amphibians and reptiles. Rodents may act as transport hosts after eating infected snails. This worm does not transmit to humans.

Amoebic dysentery

Amoebic dysentery is caused by the amoeba *Entamoeba histolytica* from the *Archamoebae* class. It infects predominantly humans and other primates. It is estimated that about 500 million people are infected with the parasite worldwide (Lucas and Upcroft 2001). Amoebic dysentery is transmitted through contaminated food and water. Symptoms of infection can include fulminating dysentery, diarrhea, weight loss, fatigue, abdominal pain, and amebomas. The amoeba can actually bore into the intestinal wall, causing lesions and intestinal symptoms, and it may reach the blood stream. From there, it can reach different vital organs of the human body, usually the liver, but sometimes the lungs, brain, spleen, etc. A common outcome of this invasion of tissues is a liver abscess, which can be fatal if untreated. Fulminant amoebic dysentery is reported to have 55–88% mortality (Singh et al. 2001). Although rodents can be infected (Abd el-Wahed et al. 1999; Battersby et al. 2002; Wiger 1977) (just like other mammals such as cats and dogs), their exact contribution to transmission remains unclear. It might be possible that infected rodent carcasses are consumed by food animals, thus causing a pathway for transmission to humans via undercooked meat.

Neosporosis

Neosporosis is caused by *Neospora caninum*. Abortions and neonatal mortality are a major problem in livestock operations, and neosporosis is a major cause of abortion in cattle (Dubey et al. 2007). Among cattle, both horizontal and vertical transmission can occur (Dubey et al. 2007). Presence and the number of farm dogs (definitive hosts) has shown to be a risk factor for seropositivity in cattle (Dubey et al. 2007). Although antibodies to *N. caninum* have been reported in humans, the parasite has not been detected in human tissues. Thus, the zoonotic potential remains uncertain (Dubey et al. 2007).

In a study from Taiwan, it was demonstrated that rats which are present in cattle farms can be naturally infected with *N. caninum* and that 16% of the rats were infected with the parasite (Huang et al. 2004). These authors claim rodents help in maintaining the life cycle of the protozoan on farms. In rodents that were collected from an urban environment in the United Kingdom, low prevalence with N. caninum was detected, 3% in house mice (*Mus domesticus*) and 4.4% in Norway rat (*Rattus norvegicus*) populations (Hughes et al. 2006). Also hares have been reported to carry *N. caninum*, with varying prevalences from 1.8% in Granada hares (*Lepus granatensis*) in Spain (Almeria et al. 2007) to 8.0% in Hungary in European brown hares (*Lepus europaeus*) (Ezio and Anna 2003). In another study (Jenkins et al. 2007), 10% of feral house mice (*M. musculus*) and 30% of the feral Norway rats (*R. norvegicus*) were positive in the *N.* caninum-specific PCR assays, while they did not show clinical signs. This suggests their involvement in the parasite life cycle, because they may serve as the parasite source to domestic and wild canids (e.g., dogs and foxes). These canids then form the definitive hosts, as was proven during a study in dogs (McAllister et al. 1998).

A rodent disease model

In order to better understand the factors that are important for the interaction between rodents and transmission of pathogens, Figure 2 offers a simplified rodent disease model. As in other models (Meerburg 2006; Mills

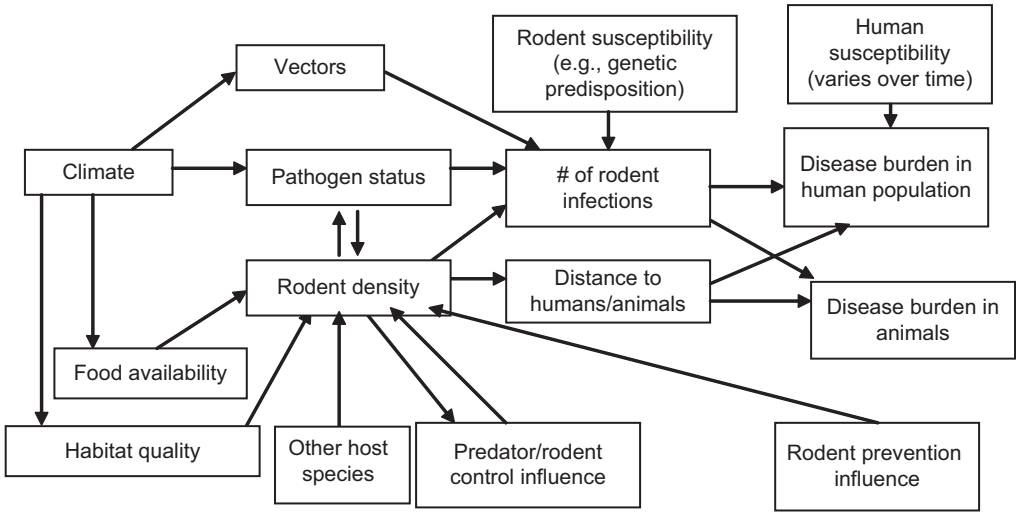

Figure 2. A simplified rodent disease model.

and Childs 1998), this simplified model demonstrates that climate (e.g. temperature, amount of sun or rain) is an important abiotic factor for the biotic elements that influence habitat quality and food availability for rodents.

From a number of studies from different countries we know that survival rate and female reproductive activity of rodents are directly related to these biotic elements (Ambramsky et al. 1985; Godoy Bergallo and Magnusson 1999; Leirs et al. 1994).

Concerning food availability, for the majority of sylvatic and field rodents, insects, vegetation and seeds provide the bulk of their diets. Abundance of these food sources vary greatly in both time and space. Climate can also cause heterogeneity concerning substrate and vegetation structure. These aspects have a direct effect on the habitat quality. For example, from desert areas it is known that desert rodents experience striking and important changes with respect to microclimate, substrate, resource availability, and predatory risk, when moving from under the canopy of perennial shrubs to bare ground only a few meters from the shrub's edge (Kotler and Brown 1988).

Climate (and climatic change) also has an influence on the status of the pathogen (Meerburg and Kijlstra 2009), as some pathogens need warm or wet circumstances to reproduce or die under cold circumstances, although some of these can survive for several years. A good example for the latter is the protozoan parasite *Toxoplasma gondii,* a parasite with a complex life cycle in which rodents play a vital role (Kijlstra et al. 2004; Lind and Buxton 2000; Meerburg et al. 2006). Remarkable is also that pathogens themselves can respond to fluctuations in the abundance of its main reservoir host. In Kazakhstan it was found that *Yersinia pestis* (that can cause bubonic plague in humans) circulates in natural populations of gerbils and responds to fluctuations by invading, fading out and reinvading in response to abundance of its main reservoir host, the great gerbil (Davis et al. 2004).

Vector abundance also is influenced by climate. The ecology, development, behavior, and survival of arthropod vectors and hosts and the transmission dynamics of the diseases they transmit are strongly influenced by climatic factors (Gubler et al. 2001; Randolph and Storey 1999). For example, ticks, that are responsible for various tick-borne diseases such as Lyme's disease, have faster development rates with higher temperatures, whilst their mortality increases with moisture stress (Randolph 2005). As a result, tick-rodent relationships vary with climate (Randolph and Storey 1999; Rosa et al. 2007). The same mechanism is visible for other host-pathogen relationships as well, e.g., in the case of plague (Parmenter et al. 1999). Global climate change can alter regional meteorological patterns and thus has an effect on pathogen, host, and vector.

If the requirements concerning habitat quality and food availability are fulfilled, then rodent density will increase. In variable environments this is often the case in response to periods of unusually high rainfall, probably caused by increase in the quantity of food available to them or its quality. Therefore, rain forecasts are sometimes used as predictive element (Davis et al. 2004). An increase in rodent density has proven to be important for dynamics of pathogens (Olsson et al. 2005), sometimes even more important than vector abundance (Telfer et al. 2007). Moreover, in a recent opinion paper the possibility of vector-independent transmission in wildlife populations is further elaborated for *Trypanosoma microti* (Smith 2008). This host-specific blood parasite of field voles completes its developmental cycle in the hind gut of a suitable flea vector. However, also in populations

where these fleas were absent, a significant degree of trypanosome infection (about one-third) remained within the field vole populations. This might be caused by direct transmission between these rodents (Smith et al. 2006; Smith 2008).

Although the previous paragraph suggests that direct contact between rodents is important for exchange of pathogens, in some cases (i.e., hantaviruses) it is not even necessary as rodents can also acquire infection from the contaminated environment (Hardestam et al. 2007; Kallio et al. 2006).

From a study that investigated the interrelation of bank voles and wood mice in the transmission of cowpox, it was demonstrated that population densities of host species can influence each other (Begon et al. 2003).

An increase in rodent density is beneficial for predators as rodents serve as an important source of food, causing a significant influence on predator population cycles. For example, the proportion of tawny owls (*Strix aluco*) that bred each year in England varied from 0 to 80%, according to the number of mice and voles present (Southern 1970).

Predation can affect prey populations directly through increased mortality, but predation risk is also known to affect some rodent species indirectly. From an experiment where mammalian and avian predators were excluded, we know that the field voles generally lost less or gained more weight in autumn and winter than voles from corresponding control grids (Carlsen et al. 1999). Also, predators can strongly influence the microhabitat use and foraging behavior of prey. In a study in Australia where the presence of red foxes and wild dogs was reduced, the native bush rat (*Rattus fuscipes*) adapted their behavior: they more frequently moved on logs where they would have been exposed to hunting foxes and dogs than normally (Strauss et al. 2008).

Thus, predation has an important influence on the rodent population. In this respect, some authors have explored the idea that predatory vertebrates indirectly protect human health (Ostfeld and Holt 2004), although evidence of this relation is limited.

Beside predation, also human influence can be important. Humans can reduce population sizes of rodent reservoirs and thus reducing the risk of human disease. If preventive measures are taken, rodent problems can often be prevented (Meerburg et al. 2004; Singleton et al. 2004). This is especially important in developing countries, because it is known that rodents can consume or spoil massive quantities of food that otherwise could have been used for human or animal nutrition (Singleton 2003; Stenseth et al. 2003).

However, it should be remembered that the involvement of humans can also cause major problems: the ongoing globalization with mass trade around the globe has led to anthropogenic introduction of exotic pathogens, or pathogen pollution (Daszak et al. 2000). Moreover, the way in which we live can have consequences on pathogens: the low genetic diversity of *T. gondii* strains is probably the result of amplification of a small subset of ancient strains on early farms that served as superior habitat for this pathogen. It is assumed that this parasite was then distributed with the spread of farming technology around the globe during the past 10,000 years (Cavalli-Sforza et al. 1994; Lehmann et al. 2003).

The interaction between different species of rodents and humans is increasing. During wars, crises and military activity (camping conditions) but also during daily farm activities, there are more contact moments between rodents and humans, potentially leading to health problems. Moreover, there is a tendency of encroachment of human populations into wildlife habitat (Molyneux 2003). In many places in the world (though mainly in developing countries), former wildlife territory is gradually transformed into agricultural land, but the original inhabitants (e.g., rodents) are often still there (Mino et al. 2007; Mohr et al. 2007; Villafane and Busch 2007). Thus, disease burden in both humans and livestock can be enlarged. Moreover, commensal rodents are less influenced by climatic factors and they live closer to humans and farm animals, thus imposing a serious health risk.

The on-going mechanization in these countries might lead to a reduction of human-rodent contacts, because human labor will be less necessary for agricultural purposes. On the other hand, the use of artificial fertilizers and improved seed will lead to better harvests, thus perhaps creating the perfect environment for rodent pests.

Presence of predators or application of rodent control methods can limit the number of rodents on-farm, while preventive measures such as rodent proofing are needed to increase the distance between rodents and food animals, thus decreasing the chance for disease transmission. This is especially important in farms with a relative open character such as organic farms (Meerburg et al. 2004).

If rodent density increases, an increase in the risk of human exposure to pathogens might be expected. This is not always a problem, as susceptibility of the human population to pathogens varies in time and place.

However, such relationships are difficult to prove (Glass et al. 2000; Ostfeld and Holt 2004) due to the high complexity of host-pathogen and host-vector interactions. Moreover, if not only rodent density, but also host diversity is taken into account, then the complexity increases even further. For example, from a recent study of vector-borne disease in wild rodents in Ireland (Telfer et al. 2005), it was found that bank voles (*Myodes glareolus*) reduced the infection prevalence of *Bartonella* in wood mice (*Apodemus sylvaticus*). The explanation for this is the so-called dilution effect hypothesis. This hypothesis predicts that for vector-transmitted parasites,

the presence of less competent host species may reduce infection prevalence in the principal host (Telfer et al. 2005). Thus, the relative contribution of each rodent species to the cumulative reservoir competence can differ among species, a fact that was reported earlier (Cooney et al. 2005; Sinski et al. 2006). Moreover, high diversity within the vertebrate host community can lead to dilution effects (Dobson et al. 2006; Keesing et al. 2006; Ostfeld and Keesing 2000; Ostfeld and Keesing 2000) in which significant negative correlations exist between species richness of terrestrial small mammals and per capita numbers of reported Lyme disease cases.

On the other hand, we know that vectors such as the tick *Ixodes scapularis* (which can cause Lyme's disease by transferring *B. burgdorferi*) have adapted their infection dynamics to their vertebrate hosts. Infected nymphs become active in spring and transmit the bacteria to rodents in the summer. Non-infected larvae that are active to the end of the summer then acquire the pathogen from infected rodents. These larvae maintain infection through their molt, giving rise to the population of infected host-seeking nymphs during spring of the following year (Yuval and Spielman 1990). Although the system of ecological and epidemiological interactions between different hosts, different pathogens and different vectors is complex, we assume that changes in rodent density have an influence on each of these equilibria.

Recommendations

The main conclusion of this article is that rodents play a significant role in transmission of a large number of diseases to humans. Also food animals are at-risk. Although the risk levels vary between the different pathogens, a better surveillance of rodent populations is important in order to predict future disease prevalences and to be able to identify novel, not yet found rodent-borne diseases.

There is a need for models that characterize demographic and population density changes of rodents with climate, its effect on their dispersal rates and patterns and the effects on the abundance of vectors and pathogens. These models will be useful in predicting expansions of rodent-borne diseases thus allowing planning which will improve public health enhancement. An example is the percolation model that was recently developed by using archived records of plague (*Y. pestis*) in populations of great gerbils (*Rhombomys opimus*) in Kazakhstan (Davis et al. 2008).

The possible effects of climate change on vectors, rodents and pathogens should be further investigated. Human activity and global climate change might lead to different interaction patterns and differentiating habitats, which could lead to emergence of infectious diseases in areas that were not affected before.

More research is required to find out in what way it is possible to interrupt transmission cycles in which rodents are involved. There is also a clear need for integrated prevention strategies. These strategies depend on a better understanding of the biology and ecology of both the hosts and the vectors, and in the construction of epidemiological models that include key abiotic and biotic factors. (Ulrich et al. 2009).

With application of proper rodent control methods, it will be possible to reduce the hazards of rodent-borne diseases in areas where humans, food animals and rodents are living close to each other. In areas where wild rodents thrive such as in forests, in deserts and mountainous areas etc., it is important that visitors are properly educated so that they are aware of the risks of rodents and can take precautionary measures to protect themselves such as wearing boots, long-sleeved pants, and trousers.

A key message to emerge from this review is the under reporting of rodent zoonoses and, in many cases, insufficient attention paid to the diagnosis of these important set of diseases. For example, information on leptospirosis and typhus from most Asian countries is extremely limited, yet it is in these countries that many of millions of agricultural workers who spend many hours working in flooded rice fields are in the high risk category for these diseases. Indeed, a recent study in Laos, investigating serologically 427 adults with unexplained fever during 2001 to 2003, produced startling results. From this large sample, 10% had acute infection of leptospirosis, 10% had acute murine typhus and 15% acute scrub typhus (PH Newton, unpublished results). These findings further highlight the urgent need for field studies of rodent populations in urban, peri-urban and rural areas to determine the likely role of particular rodent species as reservoirs for these diseases, and to improve our understanding of rodent-human interactions in key ecosystems such as the rice based agro-ecosystems in Asia.

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