

Chapter 10

What Are Zoonotic Diseases?

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Abstract Humans consider themselves as “crown of the creation,” since they feel to be guided by a breeze of the “divine spirit” that enables some insights into the scene of life and into basics of earth. However, with respect to their relationships on earth, humans are just members of the animal kingdom and thus endangered by the same agents of disease that threaten the life of animals. Since humans as predators are also members of the food chain on earth, they may also become infected with agents of diseases that are on or inside of animals that belong to the daily human food. Diseases due to such animal-based or animal-transmitted pathogens are called *zoonosis*. Such pathogens may belong to the groups of prions, viruses, fungi, bacteria, and animal parasites, which may interact in a broad spectrum of pathways. The main topics of this book—*Blastocystis* species—belong to these pathogens. Thus many pathways of transmission of the numerous above-cited pathogens will run identically or are at least very similar. Therefore it is worthwhile to throw a glimpse onto the, in general, already available transmission pathways of the agents of zoonotic diseases while giving definitions and showing important examples.

10.1 Introduction

Since about 20,000–100,000 years, when larger settlements were founded, the specimens of the *Homo sapiens* started to live in close neighborhood to farmed animals and/or animals (such as rats, mice, dogs, flies, etc.), which seek shelter or food inside or close to human dwellings. This was the chance for several, primarily strictly host-specific agents of diseases or of parasites of animals to begin a phase of coevolution inside animals and humans and thus to enlarge their host spectrum

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considerably and as consequence to obtain better chances for a long-term survival on earth. Such processes of adaptations were found in viral, bacterial, fungal, and parasitic infections, which after a slow beginning then occurred more and more often simultaneously in the constantly growing human population. Many of these natural cross-infections surely failed, while others probably ended fatal for those hosts—animals or humans—which were infected as a second host after the agent of disease had been propagated for a long time in its first hosts, which therefore had the chance to become adapted. Thus then these first hosts suffered only from low-graded symptoms of disease or even showed no disturbances after an infection. Therefore there are mostly much heavier symptoms of disease in those cases, where agents of diseases hit a host population, which had not yet been in contact with such aggressors. For example, mankind probably suffered longer from infections with *Plasmodium vivax* than with *P. falciparum*, which demands today a much higher death toll per year than all other malaria types together. Similarly opportunistic parasites such as *Giardia lamblia* introduce practically no symptoms of disease in immunocompetent hosts, while they lead often to severe, slimy diarrheas in naïve hosts (=those without any previous contact to the parasite) or in immunocompromised persons (e.g., in AIDS-patients). Another factor supporting the development of zoonotic diseases was given when mankind started farming of animals close to their housings and enlarged it in our days of mass production of animals in overcrowded stables. This close neighborhood of animals and humans (often at short distances to towns with huge populations) increases the chances for ping-pong transmissions of agents of diseases between animals and humans. This is especially the case, when flies that are attracted by feces, get the chance to become vectors by taking up pathogens from feces of some hosts and transport them to the food of other hosts. The potential importance of this often-neglected transportation system was recently shown in a study lasting 3 years, which proved the occurrence of more than 100 important parasites and bacteria on the surface and in the intestine of different fly species which were caught close to stables of horses, pigs, cattle, chicken, or rabbits respectively close to dog ponds or common human recreation sites (Gestmann et al. 2012; Förster et al. 2012). These examinations showed that severe agents of diseases such as EHEC (Enterohemorrhagic *Escherichia coli* bacteria), *Staphylococcus aureus*, *Campylobacter* sp., etc. and/or several common parasites might be easily transmitted. Thus they may introduce severe outbreaks of diseases among crowded populations of humans and animals.

10.2 Which Are the Definitions of Zoonosis?

This term has its origin in the *Greek* words *zoon* = animal and *nosos* = disease that originally meant “disease of animals.” The present use of many *Greek* terms as *termini technici* for diseases is based on the facts that this early human high culture left many written documents on diseases and potential medications and that their physicians also used the medicinal knowledge of previous human high cultures

such as the Assyrians, Babylonians, Sumerers, or Egyptians. Table 10.1 summarizes some important landmarks in the discovery of zoonotic agents of disease from the early beginning to the early twenties of the last century that was full of discoveries, that are still today of high importance (Mehlhorn 2012a, b, c; Grüntzig and Mehlhorn 2010a, b).

In the more recent past the original sense of the word “zoonosis” as “disease of animals” was erroneously changed by scientists who apparently were not familiar with the “old Greek” language. They started to use the word “zoonosis” to describe a peculiar disease, the agents of which were constantly transmitted in a cycle between animals and humans. However, since there are significant variations in the ways, modes, and directions of the transmission pathways, many international groups of researchers described several subgroups of zoonosis. However, there is no final agreement in the use of the terms among the international community of scientists. In general up to five different types of zoonosis were used in literature (see Mehlhorn 2008, 2012a, b, c).

1. *Direct zoonosis*: This term is often used to characterize the transmission of agents of disease, the shape of which remains identical in animals and later in infected humans. Examples of such direct zoonosis occur during the transmission of the viruses of the rabies disease (*Lyssa-virus*), of the bacteria of the brucellosis (*Brucella* species), of the parasitic protozoans introducing the entamoebiasis (*Entamoeba histolytica*) or the giardiasis (*Giardia duodenalis*). Furthermore the top targets of this book—the *Blastocystis* species—would also fit into the category of a direct zoonosis, since the fecally excreted cyst stages are taken up orally and then may colonize the intestinal tract of humans.
2. *Cyclic zoonosis*: In these cases a successful transmission from one host to another one may only occur after a morphologic transformation of the agent of disease has occurred in an animal or in a human. This example is the case in the life cycles of tapeworms such as *Echinococcus granulosus* or *Taenia solium*, both of which are found as adult worms in the intestine of the final host (=predators, carnivores) and as larval stages in the muscles and/or other tissues of intermediate hosts (prey animals, herbivores or omnivores).
3. *Metazoonosis*: This term is used to describe transmission pathways that include arthropods as distributors (=vectors), which in some cases ingest an agent of disease, allow its reproduction in their bodies or not, but transmit these pathogens to another host during a bite or by fecal contamination. Examples for this category would be the transmission of plague bacteria (*Yersinia pestis*) by fleas, the viruses of the Dengue or Yellow fever by *Aedes* mosquitoes, or the mechanical transmission of bacteria and parasites that are attached at the feet, mouthparts of flies, or that are included in the feces of any other insect (e.g., cockroaches).
4. *Saprozoonosis*: This group contains agents of disease that occur in nature on the soil or on degenerating contaminated food of animals which thus become infected. These pathogens may be transmitted, when humans eat raw meat or come into contact with fecally contaminated plants or waste waters. Examples

Table 10.1 Historical landmarks in the discovery of zoonotic parasitic diseases (selection)

Date	Event
5000 BC	Nematode eggs discovered recently in a frozen human body (Ötzi) in Austrian Alps
2000 BC	<i>Taenia</i> and <i>Schistosoma</i> ova in Egyptian mummies
1500 BC	Papyrus Ebers (Egypt) gives reference to roundworms (<i>Ascaris lumbricoides</i>), threadworms (<i>Enterobius vermicularis</i>), and tapeworms (<i>Taenia</i> sp.)
1234–1300 BC	Biblical references to <i>Dracunculus medinensis</i> in Red Sea region
700–600 BC	Records of <i>Dracunculus medinensis</i> worms from Mesopotamia (today Irak)
500 BC	The Greek poet Herodot reported that the Egyptians used fine nets against mosquitoes
430 BC	The Greek physician Hippocrates (460–399 BC) described <i>Ascaris</i> , <i>Oxyuris</i> , adult <i>Taenia</i> , and Malaria
342 BC	The Greek natural scientist Aristotle (384–322 BC) established a first classification system for animals (<i>Historia animalium</i>) and described flat and round worms
300 BC	Chinese description of threadworms, tapeworms, hookworms, and hookworm disease
20 AD	The Roman Aulus Celsus recognized as tapeworms <i>Taenia</i> , <i>Tinea</i> , and <i>Taeniola</i> (<i>Taenia</i> sp.), vermes cucurbitini (tapeworm proglottids), “hailstones” (cysticerci), and roundworms, lumbrici teretes (<i>Ascaris lumbricoides</i>)
62 AD	In his <i>Historia naturalis</i> , the Romans Lucius Columella (40–88 AD) and Plinius Secundus (23–79 AD) reported on parasitic animal diseases
129–199 AD	The Greek-Roman physician Galen recognized three types of worms: roundworms (<i>Ascaris lumbricoides</i>), threadworms (<i>Enterobius vermicularis</i>), and tapeworms (<i>Taenia</i> sp.) and also cysticerci in livers of slaughtered animals
1000 AD	Avicenna (Abu Ali El-Hosein Abdallah Ibn Sina, 980–1037). This Persian theologian and physician reported in his book <i>Liber canonis medicinae</i> on malaria and many worms, especially on <i>Dracunculus</i> , which today in French is still called <i>Fil d' Avicenne</i>
1150 AD	Hildegard of Bingen (1098–1179). This German nun published plant based methods of treating worms in her book <i>De causis et curis morborum</i>
1498 AD	The Italian monk Girolamo Savonarola (1452–1498) described in his <i>Tractatus de vermibus</i> the occurrence and treatment (by mercury) of worm-infected humans
1520 AD	The German physician P.A.T. Bombastus von Hohenheim, called Paracelsus (1496–1541) introduced inorganic salts (e.g., zinc salts) as anthelmintica
1684–1698 AD	The Italian physician and philosopher Francesco Redi (1626–1697) described in his book <i>Osservazioni interno agli animali viventi</i> about 108 different worms and published (in 1798) a detailed study on <i>Fasciola hepatica</i> . Due to his leadership he is considered the Father of Parasitology—the name “rediae” for special trematode developmental stages honors this
1699 AD	The Dutchman Nicolaas Hartsoeker (1656–1725) and the Frenchman J. Andry (1658–1742) proposed that helminth infections derive from oral intake of excreted worm eggs
1717 AD	The Italian Lancisco postulated that malaria is caused by bites from mosquitoes
1739–1778 AD	The Swedish physician Carl von Linné (1707–1778) created within the different editions of his book <i>Systema naturae</i> a comprehensive system of classification of animals and plants
1756 AD	The English physician Alexander Russel (1715–1768) discovered in Aleppo (today in Syria) the skin leishmaniasis

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Table 10.1 (continued)

Date	Event
1801 AD	The German physician and natural scientist Carl Asmund Rudolphi (1771–1832) published his book <i>Entozoorum historia naturalis</i> with the taxonomy of all available parasites
1801 AD	The French natural scientist Jean Baptiste Lamarck (1744–1829) presented the first general theory of the evolution within his <i>Philosophie Zoologique</i>
1820 AD	The French scientists Pierre Joseph Pelletier and Joseph Lavandou isolated quinine from plants
1838 AD	The German scientists N. Schleiden and Theodor Schwann (1810–1882) formulated the “cell theory,” which became the dead end for the former de novo-creation-theory
1848 AD	The American Josiah Nott postulated again that mosquitoes were vectors of malaria and yellow fever
1850 AD	The German physician Theodor Bilharz discovered <i>Schistosoma haematobium</i> in Cairo, Egypt
1853 AD	The German C.T.E. von Siebold (1804–1885) showed the life cycle of <i>Echinococcus granulosus</i>
1855 AD	The German physician Gottlieb Küchenmeister demonstrated that <i>Taenia solium</i> infections are acquired by eating infected pork
1859 AD	The Germans Rudolph Virchow (1821–1902) and Rudolph Leuckart (1822–1898) independently discovered the life cycle of <i>Trichinella spiralis</i>
1859 AD	The English philosopher, theologian and natural scientist Charles Darwin (1809–1882) published his book <i>The Origin of Species</i> —another landmark of the evolution theory
1863 AD	The English scientist Thomas Cobbold suggested that snails might be the intermediate host of schistosomes
1867 AD	The German zoologist Rudolf Leuckart described the life cycle of <i>Echinococcus granulosus</i>
1869 AD	The German physician Otto Wucherer (1820–1873) discovered in Brazil microfilariae and schistosomal eggs in human urine
1876 AD	The English scientist Joseph Bancroft observed and described adult <i>Wuchereria bancrofti</i> worms
1878 AD	The Scottish scientist Sir Patrick Manson (1844–1922) working in China showed that <i>Wuchereria bancrofti</i> is transmitted by mosquitoes (<i>Culex</i> sp.)
1880 AD	The French Charles L.A. Laveran (1845–1922) showed malaria stages within erythrocytes (Nobel Prize in 1907)
1881 AD	Leuckart and Thomas independently described the life cycle of <i>Fasciola hepatica</i>
1888 AD	The French physiologist Charles Richet (1855–1935) formulated the basic concept of humoral immunity (Nobel Prize 1919)
1893 AD	The Americans Theobald Smith and F.L. Kilbourne identified the transmission of <i>Babesia bigemina</i> by ticks (<i>Boophilus annulatus</i>)
1895 AD	The English military physician David Bruce (1855–1932) showed that the tsetse fly is the vector of animal trypanosomes
1897 AD	In India the English Army doctor Sir Donald Ross (1857–1932) proved that avian malaria was transmitted by <i>Anopheles</i> mosquitoes (Nobel Prize in 1902). In the same year the Italians Bignami, Bastianelli and Grassi did the same for human malaria

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Table 10.1 (continued)

Date	Event
1898 AD	The German physician and discoverer of the agents of anthrax, cholera and tuberculosis (<i>Mycobacterium tuberculosis</i>) Robert Koch (1843–1910) described <i>Theileria parva</i> , the agent of East Coast fever (Nobel Prize 1905)
1898 AD	The French P.L. Simond succeeded in demonstrating the transmission of plague by rat fleas
1900 AD	In Cuba an American group working with Walter Reed demonstrated the transmission of yellow fever by mosquitoes (<i>Aedes aegypti</i>)
1903 AD	The English scientists W.B. Leishman and C. Donovan independently described <i>Leishmania donovani</i> , the agent of Kala-azar disease (Leishmaniasis)
1904 AD	In Cairo the German helminthologist Arthur Loos (1861–1923) discovered the transmission of the hookworm
1906 AD	The American physician Howard T. Ricketts (1878–1910) recorded the tick <i>Dermacentor andersoni</i> as being a vector of the agents of the Rocky Mountain spotted fever
1906 AD	The German zoologist and discoverer of the agent of syphilis Fritz Schaudinn (1871–1906) described <i>Entamoeba histolytica</i> as a human parasite introducing bloody diarrhea
1907 AD	The American E.E. Tyzzer described stages of the genus <i>Cryptosporidium</i>
1907 AD	The German chemist Paul Ehrlich (1854–1925)—the father of chemotherapy—proposed the drug trypan red against trypanosomiasis (Nobel Prize 1908)
1908 AD	In North Africa the French military doctors Charles Nicolle (1866–1936) and L.H. Manceaux described <i>Toxoplasma gondii</i> in a rodent
1909 AD	The Brazilian Carlos Chagas (1879–1934) discovered the life cycle of <i>Trypanosoma cruzi</i> and described <i>Pneumocystis carinii</i>
1909 AD	Teams working with the French scientist Charles Nicolle (1866–1936) (Tunis) and the American H.T. Ricketts (Mexico) proved that the louse <i>Pediculus humanus corporis</i> is the vector of the typhus-causing rickettsia
1910 AD	The Italian scientist Carini discovered <i>Pneumocystis carinii</i> in rats
Until today	And so on the work of hundred thousands scientists, etc

for this group would be the transmission of the bacterial agents of listeriosis, of the spores of fungi of mycosis, or cysts of amoebic dysentery.

5. *Other authors* grouped zoonotic transmission procedures according to the main direction that is taken by an agent of disease.

- (a) *Zooanthroponosis*: In these cases the infectious agents occur mainly in animals and may be transmitted from there to humans. Examples for this category would be the transmission of the *Trichinella spiralis* larvae in muscles of wild boars to humans when eating undercooked meat. Also the transmission occurs when *Balantidium coli* cysts are swallowed by humans.
- (b) *Anthropozoonosis*: This term describes the pathway of transmission, when a pathogen of an animal has its origin in humans and leads to disease in animals. This may be the case when e.g., wild bears or wolves feed on humans infected with *Trichinella* muscle larvae or when pigs or cattle

swallow human feces containing the sporocysts and/or the oocysts of *Sarcocystis* species. Of course pathways of transmission starting from humans are much more rare than those with a source in animals reaching humans from there.

When looking at the great variations of transmission pathways that have been established by zoonotic agents between humans and animals during coevolution, an absolutely correct classification according to the categories described above is mostly very difficult. Therefore the proposal to use exclusively the term *zoonosis* to characterize any of the numerous ping-pong infections between humans and animals seems most reasonable. In this case, however, it makes sense to differentiate according to the main pathways of transmission into:

- (a) Food-borne zoonotic diseases
- (b) Vector-borne zoonosis
- (c) Cyclic zoonosis
- (d) Contamination-borne zoonosis

10.3 Examples of Zoonotic Pathways in the Transmission of Pathogens Between Humans and the Animals and from Their Surroundings

Except for prions and viruses which depend on living cells and are reproduced by them all other members of the living society on earth—plants, fungi, animals (including humans)—have to fight for their survival, i.e., they have to defend their living space (=biotope) against competitors. Only those, which had been the winners in this daily survival fight, are our contemporaries. All members of the living society on earth belong to a giant food chain—a pyramid with predators at top sites and preys and producers at the lower levels. Therefore it is not astonishing that there had been established correlations among the different members of the food chain and that man and animals are attacked by various organisms seeking shelter and propagation inside their bodies. Such invasions may occur occasionally or might have been established since long and thus run often according to extremely fixed pathways of transmission. Tables 10.2, 10.3, 10.4, 10.5, 10.6 in the following Sects. 10.3.1–10.3.5 list some of the most important human diseases, which have their sources in organisms that exist longer on earth and thus in the human surroundings of our days than mankind. This is not only true for the rather recent *Homo sapiens* with his only ~100,000-years-old history but also for its precursors such as specimens of the genera *Ardepithecus* or *Australopithecus*, etc. Since man still exists although there are these huge amounts of hostile agents of diseases around him, there is good hope that humans will have a future on earth.

Table 10.2 Prions with a zoonotic potential

Species	Characteristics	Disease/treatment	Symptoms of disease	Hosts	Pathway of transmission	Geographic distribution
Prions of the variation of Creutzfeld-Jacob (Bovine Spongiform Encephalopathy; BSE)	Infectious proteins, misfolded in β -constellation: PrP ^{Sc}	New variation of Creutzfeld-Jacob/no treatment	Grimasses, giddiness, ataxia, brain destruction, death	Humans, cattle	Eating infected beef, blood transfusion, iatrogen (during surgery)	Worldwide; epidemic in Great Britain (1995–1998), a few cases in France, USA, Spain, Italy, Canada, etc.
Prions of Kuru	Infectious proteins, misfolded in β -constellation: PrP ^{Sc}	Kuru/local name: laughing death/no treatment	Grimasses, tremor, brain destruction, death; local name: laughing death	Humans	Cannibalism	Papua-New Guinea
Scrapie-prion	Infectious proteins, misfolded in β -constellation: PrP ^{Sc}	Scrapie in sheep/ (German: Traberkrankheit)/ no treatment	Sticky walking of sheep and cattle, turning movement	Sheep, cattle	Cows and sheep are probably infected by eating fly larvae in grass that had previously fed on infected sheep bodies or on placental material (our group showed experimental transmission via fly larvae to hamsters) ^a	Worldwide

^aPost et al. (2009)

Table 10.3 Virus-borne diseases with zoonotic potential (selection)

Family/genus	Virus	Main symptoms of disease	Hosts	Pathway of transmission	Geographic dislocation
Togaviridae <i>Alphavirus</i>	Sindbis	Fever, exanthema	<i>Humans</i> , birds	Bite of mosquitoes	Europe, Africa, Asia, Australia
	Chikungunya	Fever, pain in joints	<i>Humans</i> , monkeys	Bite of <i>Aedes</i> -mosquitos	Africa, Southern Europe
	American horsevirus	Fever, encephalitis	<i>Humans</i> , horses	Bite of midges, mosquitoes	North-, South America
Flaviridae <i>Flavivirus</i>	Dengue fever	Hemorrhagic fever, classic fever	<i>Humans</i> , monkeys	Bite of mosquitoes (<i>Aedes</i>)	All tropics and subtropics
	Japanese Encephalitis	Encephalitis, flu, mortality ~25 %	<i>Humans</i> , birds	Bite of mosquitoes (<i>Aedes</i>)	North, South Asia, India
	West-Nile fever	Fever, encephalitis	<i>Humans</i> , birds, dogs, horses, etc.	Bite of mosquitoes (<i>Aedes</i> , <i>Culex</i>)	Africa, South America, Israel, South France
	Yellow fever	Fever, icterus, mortality up to 50 %	<i>Humans</i> , monkeys	Bite of mosquitoes (<i>Aedes aegypti</i>)	South America, Africa
	Spring-summer-meningo-encephalitis	Meningitis, encephalitis, fever, mortality ~10 %	<i>Humans</i> , mice, many other animals	Bite of <i>Ixodes</i> ticks	Russia, Central Europe, countries of the Balcony
Bunyaviridae	Hanta	Lung inflammations, hemorrhagic fever	<i>Humans</i> , mice, rodents	Fecal-orally, flies	Europe
	Phlebo Rift-Valley	Fever, meningitis Fever, encephalitis, hepatitis	<i>Humans</i> <i>Humans</i> , cattle, sheep, ruminants	Sandfly bite Bite of midges, many mosquitoes	Mediterranean Sea Central, South Africa, Egypt
	Crimean-Congo	Haemorrhagic fever	Cattle, <i>humans</i>	Bite of <i>Hyalomma</i> -ticks	Africa, Turkey, Black sea region

(continued)

Table 10.3 (continued)

Family/genus	Virus	Main symptoms of disease	Hosts	Pathway of transmission	Geographic dislocation
Reoviridae	Colorado-tick-virus	Encephalitis	<i>Humans</i> , roe deers	Bite of ticks	Rocky-Mountains, USA
Coltivirus	Banna-virus	Encephalitis	<i>Humans</i>	Fecally, fly contact	China, Indonesia
Seadorna	<i>Rotavirus</i> (A-G)	Gastroenteritis, diarrheas, enterotoxigenicity	<i>Humans</i> , many animals	Oral-fecal, fly contacts	Worldwide
Rotaviruses					
Rhabdoviridae	Lyssa (rabies)	100 % death toll	<i>Humans</i> , foxes, dogs, cats, wolves, roe deers	Bite of animals, smear infection, wound contamination, flies?	Worldwide
<i>Lysnavirus</i>					
Orthomyxoviridae	Influenza-virus	High fevers, lung infection	<i>Humans</i> , animals	Aspiration, fly transmission	Worldwide
Calciiviridae	Calici-virus	Diarrheas	<i>Humans</i> , animals	Oral-fecally, flea feces and bites	Worldwide

Table 10.4 Bacteria of zoonotic importance (selection)

Species	Characteristics	Disease/treatment	Symptoms of disease	Hosts	Pathway of transmission	Geographic distribution
<i>Anaplasma phagocytophylum</i> (syn. <i>Ehrlichia</i>)	Coccoid, gram-negative, pleomorphic, nonmotile, obligatorily intracellular bacteria of 0.4–1.9 µm in size	Human granulocytic anaplasmosis (HGA)/ Doxycycline	High fevers, occasional meningitis, exanthemes	<i>Humans</i> , ruminants, roe deers, dogs, cats, mice	Bite of <i>Ixodes</i> ticks	North and Central Europe, North and East of USA
<i>Bartonella</i> (syn. <i>Rochalimaea quintana</i>)	Microaerophilic, Gram-negative, facultatively intracellular rods	French fever; 5 days fever; bacillary pelliosis; Wolhynic fever/ Doxycyclin/Rifampicin	Bacteremia, endocarditis	<i>Humans</i>	Inhaling of feces of body lice	Worldwide
<i>Bartonella bacilliformis</i>	Microaerophilic, Gram-negative, facultatively intracellular rods	Carrión disease, Oroya-fever, Verruga peruana/ Doxycyclin	High fevers, intraerythrocytic bacteremia	<i>Humans</i>	Bite of sandflies	Peru, Ecuador
<i>Bartonella henselae</i>	Microaerophilic, Gram-negative, facultatively intracellular rods	Cat-scratch-disease/ Azithromycin in severe cases; normally not needed	Lymphadenitis	<i>Humans</i> , cats	Scratching by cats or by contaminated mouthparts of fleas and ticks	Worldwide
<i>Borrelia burgdorferi</i> , <i>B. afzelii</i> , <i>B. garinii</i> , etc.	Spirillic with irregular windings 0.2–0.5 × 8–30 µm, possess endoflagella	Borreliosis; Lyme borreliosis/Doxycyclin	Rosacea migrans, Acrodermatitis; arthritis, paralysis	<i>Humans</i> , many warm-blooded animals	Tick bites (<i>Ixodes</i> species)	Europe, USA
<i>Borrelia duttoni</i>	Spirillic with irregular windings 0.2–0.5 × 8–30 µm, possess endoflagella	Tick-borne relapse fever/ Cephalosporines	High fever, myalgia, icterus, hepatomegaly, high lethality	<i>Humans</i> , reservoir mice, camels	Bites of <i>Ornithodoros moubata</i> -ticks	Africa
<i>Borrelia recurrentis</i>	Spirillic with irregular windings 0.2–0.5 × 8–30 µm, possesses endoflagella	Louse-borne relapse fever/ Cephalosporines	High fever, myalgia, hepatomegaly, high lethality	<i>Humans</i>	Inhaling of contaminated body louse feces	Worldwide
<i>Borrelia turicata</i> , <i>B. parkeri</i> , etc.	Spirillic with irregular windings 0.2–0.5 × 8–30 µm, possesses endoflagella	American tick-borne relapse fever/Doxycyclin	High fever, myalgia, icterus, etc. hepatomegaly, lethality	<i>Humans</i> , mice, rodents	Bites of <i>Ornithodoros</i> species (argasid ticks)	Northwestern America

(continued)

Table 10.4 (continued)

Species	Characteristics	Disease/treatment	Symptoms of disease	Hosts	Pathway of transmission	Geographic distribution
<i>Brucella melitensis</i> , <i>B. suis</i> , <i>B. canis</i>	Gram-negative, coccoid rods; reproduction in macrophages	Brucellosis, Malta-fever; Mediterranean fever; Bang disease/Doxycycline + Rifampicin	Severe influenza-like; lethality 2–5 %; abortions	<i>Humans</i> , sheep, goats, cows, dogs, pigs	Infected raw meat, raw milk, fly contact?	Worldwide in endemic regions
<i>Campylobacter</i> species; <i>C. jejuni</i> , <i>C. coli</i>	Spirillum bacteria (with flagella) of 0.5–5 µm in length inside intestinal lumen	Enterocolitis/Fluorchinolones, Erythromycinsteart	Enterocolitis with fever; Guillain-Barré-Syndrom	<i>Humans</i> , birds, cattle, pigs	Fly transmission or oral uptake of bacteria from bird feces, raw meat of milk.	Worldwide
<i>Coxiella burnetii</i>	Gram-negative, obligatorily intracellular bacteria; similar to legionellids	Q-fever (Query-fever)/Doxycycline	Like a summer flu	<i>Humans</i> , sheep	Bites of about 50 tick species	Worldwide
<i>Ehrlichia chaffeensis</i> , <i>E. ewingii</i>	Coccoid, Gram-negative, pleomorphic, nonmotile, obligatorily intracellular bacteria of 0.4–1.9 µm in size	Human monocytic ehrlichiosis/Doxycycline	High fevers, occasional meningitis, exanthemes	Dogs, wild ruminants, <i>humans</i>	Bites of <i>Amblyomma</i> -ticks	Southern USA
<i>Francisella tularensis</i> , <i>F. holarctica</i> and other subspecies	Gram-negative rods with capsule; intracellular reproduction	Tularaemia/Gentamycine, Streptomycine	High fevers, respiratory and/or dermal syndromes; lethality up to 50 %	<i>Humans</i> , rodents, hares, wild animals	Inhalation, contaminated mouthparts of arthropods	Northern hemisphere, East Asia
<i>Helicobacter</i> species; <i>H. pylori</i> , <i>H. suis</i> , <i>H. heilmanni</i> , etc.	Spirillum bacteria (with flagella) of 3–6 µm in length; inside stomach (closely attached to antrum and corpus regions of stomach)	Gastric and hepato-intestinal symptoms/Clarithromycine, Amoxicillin, etc.	Chronic gastritis, often symptomless	<i>Humans</i> , monkeys, pigs, etc.	Fly transmission or oral uptake of fecally contaminated food	Worldwide
<i>Legionella pneumophila</i>	Cocci, reproduction in macrophages and in water amoebae	Legionnaire's disease; Pontiac fever/Macrolids, Rifampicin	Pneumonia; lethality up to 80 %	<i>Humans</i> , free water amoebae	Inhalation of infected amoebae in biofilms of water systems	Worldwide

<i>Leptospira</i> species; <i>L. interrogans</i> ; <i>L. borgpetersenii</i> , etc.	Spirillic, very thin, highly motile bacteria $0.2 \times 6-30 \mu\text{m}$ in size	Weil's disease, leptospirosis/Penicillin G, Doxycyclin	Septicaemia, fever, diarrhea, icterus	<i>Humans</i> , wild animals, rats, mice	Fecal contamination of wounds, inhaling bacteria from feces of rats	Worldwide
<i>Listeria monocytogenes</i> (syn. <i>Corynebacterium infantisepticum</i>)	Facultative anaerobe rod-like Gram-positive, peritriche bacteria, intra- and extracellular	Listeriosis/Ampillicin	Flu-like symptoms, but also meningitis, evtl. abortion	<i>Humans</i> , ruminants, pigs, birds	Fecal contamination, milk of animals	Worldwide
<i>Neorickettsia sennetsu</i> (syn. <i>Ehrlichia</i>)	Cocciid, Gram-negative, pleomorphic, nonmotile, obligatorily intracellular bacteria of $0.4-1.9 \mu\text{m}$ in size	Japanese sennetsu-ehrlichiosis, mononucleosis/Doxycyclin	Fever, hepatosplenomegaly, leucopenia	<i>Humans</i>	Tick bites	Japan, Malaysia
<i>Orientia</i> (syn. <i>Rickettsia tsutsugamushi</i>)	Obligatory intracellular short or cocciid rods of $0.8-2 \mu\text{m}$ in size	<i>Tsutsugamushi</i> -fever/Doxycyclin	High fevers, skin exanthemes	<i>Humans</i> , dogs, rodents	Bites of mites (<i>Trombicula</i> sp.)	Asia, India, Australia
<i>Rickettsia conorii</i>	Obligatory intracellular, short or cocciid rods of $0.8-2 \mu\text{m}$ in size	Mediterranean tick-bite fever/Doxycyclin	High fevers, skin exanthemes	<i>Humans</i> , dogs, rodents	Bites of <i>Rhipicephalus sanguineus</i> -ticks	Mediterranean Sea region, India, Africa
<i>Rickettsia prowazekii</i>	Obligatory intracellular short or cocciid rods of $0.8-2 \mu\text{m}$ in size	Louse-borne spotted typhus, Brill-Zinsser disease up to 60 % lethality/Doxycyclin	High fevers, skin exanthemes	<i>Humans</i>	Bites and feces of body lice	Worldwide
<i>Rickettsia rickettsii</i>	Obligatory intracellular, short or cocciid rods of $0.8-2 \mu\text{m}$ in size	Rocky-Mountains spotted fever/Doxycyclin	High fevers, skin exanthemes	<i>Humans</i> , dogs, rodents	Bites of <i>Dermacentor</i> , <i>Rhipicephalus</i> , <i>Amblyomma</i> ticks	North and South America
<i>Rickettsia typhi</i>	Obligatory intracellular short or cocciid rods of $0.8-2 \mu\text{m}$ in size	Murine spotted fever/Doxycyclin	High fevers, skin exanthemes	<i>Humans</i> , rodents	Bites of fleas, e.g., <i>Xenopsylla cheopis</i>	Worldwide

Table 10.5 Fungi with an anthropophilic zoonotic potential (selection)

Species	Characteristics	Disease/treatment	Symptoms of disease	Hosts	Pathway of transmission ^a	Geographic distribution
<i>Pneumocystis carinii</i> , <i>P. jiroveci</i>	Thin-walled cysts with 5–7 µm in diameter, containing up to eight 1 µm sized trophozoites	Pneumocystosis in immunodeficient persons/ Sulfonamids	Lung exsudate, oedema, pneumonia, bacterial super infections	<i>Humans</i> , rats	Inhalation of cysts or by sputum contact; fly transmission?	Worldwide
<i>Microsporium canis</i> (syn. <i>M. equinum</i> , <i>M. distortum</i>)	Colonies grow quickly, borders irregular; microconidia, macroconidia with enlarged tip	<i>Tinea capitis</i> , <i>T. corporis</i> / Terbinafin, Fluconazol, Griseofulvin	<i>Tinea capitis</i> , <i>T. corporis</i>	<i>Humans</i> , cats, dogs	Body contact with infected pet animals	Worldwide
<i>Trichophyton rubrum</i>	Slowly growing colonies are flat and appear yellow reddish-brown; mostly no macroconids	All human types of tinea (<i>T. capitis</i> , <i>T. unguinum</i>)/ Ciclopirox or Amorolfin-nail-set	All human forms of tinea	<i>Humans</i> , animals as transporters	Body contact, common use of shoes, clothes	Worldwide
<i>Trichophyton interdigitale</i>	Quickly growing colonies with a whitish surface and yellow borders, microconids; coccoid shaped; macroconids 7–50 µm in diameters	<i>Tinea corporis</i> , <i>T. pedis</i> / Terbinafin	All human forms of tinea	<i>Humans</i> , cats, mice	Body contact, common use of shoes, clothes	Worldwide
<i>Trichophyton equinum</i>	Quickly growing whitish colonies, later brown	<i>Tinea corporis</i> /Terbinafin, Fluconazol	All human forms of tinea	<i>Humans</i> , horses	Contact with contaminated hair of horses	Worldwide

<i>Trichophyton benhamiae</i> complex	Like <i>T. interdigitale</i>	<i>Tinea capitis</i> , <i>T. corporis</i> / <i>T. capitis</i> : Griseofulvin, Fluconazol; <i>T. corporis</i> : Terbinafin, Fluconazol	Infestations of skin <i>Humans</i> , rabbits, guinea pigs	Contact with hair of infected animals	Worldwide
<i>Candida albicans</i> , and many synonyms	Whitish colonies, pseudomycel, chlamyospores	Candidosis, e.g., vaginitis/ Nystatin, Fluconazol, Itraconazol	Infestations of skin and inner mucuous layers, soor, flour	Body contact, contact to fecal smears, transmission by licking flies	Worldwide

^aAll permanent stages of fungi might be transmitted via contaminated mouthparts or feet of flies

^bThe systematic position of the genus is still unclear—even after molecular biological investigations—since there are characteristics of fungi as well as those of animal cells of protozoan type. Mostly there is no differentiation done between *P. jiroveci* (first described in humans) and *P. carinii* (first described in rats)

Table 10.6 Parasites with a zoonotic life cycle (selection)

Species	Characteristics	Disease/treatment	Symptoms of disease	Hosts	Pathway of transmission	Geographic distribution
Protozoa <i>Giardia duodenalis</i> - assemblages	Trophozoites with 8 flagella, cysts with 4 nuclei, 10–20 µm in size	Giardiasis/metronidazol	Slimy diarrheas	<i>Humans</i> , many animals	Oral uptake of cysts from feces; fly transmission	Worldwide
<i>Cryptosporidium hominis</i> and animal assemblages	Oocysts are tiny, spherical, measure only 5 µm and contain no sporocysts, but only 4 sporozoites	Cryptosporidiosis/no definite treatment available	Extreme watery diarrheas in immune-suppressive persons, none in immunocompetent ones	<i>Humans</i> , many animals	Oral uptake of oocysts from feces; fly transmission	Worldwide
<i>Toxoplasma gondii</i>	Spherical oocysts (10 µm) in cat feces; trophozoites (7–8 µm) in macrophages and tissue cysts in muscle fibers and brain cells of warm blooded hosts	Toxoplasmosis/sulfonamides	None in immuno competent persons; brain damages in immunocompromised ones; fetal damages possible, if pregnant woman had not been infected before	<i>Humans</i> , many animals	Oral uptake of oocysts from cat feces Eating of raw or undercooked meat of infected animals Fly transmission	Worldwide
<i>Plasmodium</i> species	Schizogony in liver cells Schizogony in red blood cells Gamogony and sporogony in mosquitoes	Malaria/mefloquin, proguanil, atovaquone, artemisinin, etc.	High, often rhythmic fevers, high mortality in cerebral malaria	<i>Humans</i> , some monkeys	Bite of 60 <i>Anopheles</i> mosquito species	Tropical countries around the world
<i>Sarcocystis suihominis</i> , <i>S. bovihominis</i>	<i>Humans</i> excrete oocysts/ sporocysts, in pigs (or cattle) occur schizonts, muscle and brain cysts	Sarcosporidiosis	<i>Humans</i> : Watery diarrheas of high intensity 4–24 h after eating raw meat	<i>Humans</i> , pigs	<i>Humans</i> eat raw or undercooked meat of pigs; pigs feed human feces with oocysts; fly transmission	Worldwide
Microsporidia <i>Encephalitozoon cuniculi</i>	Spores with an extrudable hollow tube, through which the plasm enters host cells	Encephalitozoonosis/ albendazole	Destruction of kidney and other cells	Rabbits, dogs, immune suppressed <i>humans</i>	<i>Humans</i> : oral uptake of spores from rabbit feces or other infected hosts	Worldwide

Flukes <i>Schistosoma</i> species	♀ and ♂ live as couples inside of blood vessels, eggs possess characteristic hooks at the surface of the shell	Schistosomiasis/praziquantel	Blood in feces or urine (depending on the species); as late symptoms development of cancer in liver or bladder	Finals hosts: humans and many mammals; intermediate hosts: water snails	Skin penetration of cercariae that had been excreted by hosts via urine or feces into waters	Tropical countries
<i>Clonorchis- and Opisthorchis</i> species	Flukes are hermaphroditic animals, tiny eggs possess an operculum = cover	Clonorchiasis/opisthorchiasis/praziquantel	Liver problems, cancer development	Finals hosts: humans, cats, dogs; intermediate hosts: snails and fish	Eating raw or undercooked fish meat with metacercariae	Asia
Tapeworms <i>Taenia solium</i>	Adult worms show 4 suckers and a Rostellum with hooks; eggs = embryophores contain the oncosphaera larva	Cysticercosis, taeniasis/praziquantel	Adult worm: Disturbance of intestinal process; Larva: severe brain problems in case of brain cysticercosis	Humans, pigs	Humans: eating raw or undercooked pork meat;pigs: oral uptake of worm eggs in human feces	Worldwide
<i>Dipylidium caninum</i>	Excreted terminal proglottids appear like rice-grains	Dipylidiasis/praziquantel	Intestinal disturbances	Dogs, cats, humans	Engorging cysticercoid larvae in portions of cracked fleas inside hair of animals	Worldwide
<i>Diphyllobothrium latum</i>	Up to 20 m long, terminal proglottids are empty	Diphyllobothriasis/praziquantel	Intestinal problems, anemia of the pemiosa type	Humans	Eating raw fish with plerocercoids	Worldwide
Nematodes <i>Trichinella spiralis</i>	Larva parasitizes intracellularly in muscle cells	Trichinosis/benzazoles	Breathing problems due to intoxication and malfunction of muscles	Humans and many meet feeding animals	Eating raw or undercooked meat of infected animals	Worldwide
<i>Anisakis</i> species	Only larvae are found in human intestine	Anisakiasis/benzazoles macrolids	Intestinal problems up to penetration of the intestinal wall; peritonitis	Humans, fish	Eating raw soft water fish	Worldwide
<i>Wuchereria bancrofti</i>	Microfilariae free in blood, macrofilariae have their sites in lymph nodes	Filariasis/diethylcarbamazin/doxycyclin	Tropical elephantiasis = swelling of legs, breasts, etc.	Humans, monkeys	Bite of typical mosquitoes (<i>Aedes, Anopheles, Culex</i>)	Tropical countries

(continued)

Table 10.6 (continued)

Species	Characteristics	Disease/treatment	Symptoms of disease	Hosts	Pathway of transmission	Geographic distribution
<i>Ochocerca volvulus</i>	female worms lay as bundles in skin nodules, larvae are found in lymph below skin	River blindness, onchocerciasis/ivermectin, doxycyclin	Itching, skin peeling, blindness due to degeneration of larvae inside eyes (as late symptom)	<i>Humans</i> , monkeys	Bite of simuliids (<i>Simulium</i> species)	Africa, South and Middle America
Acanthocephala <i>Macracanthorhynchus hirudinaceus</i>	Intestine-less worms with a hooked anterior pole	Acanthocephaliasis/loperamid	Intestinal wall perforations, intestinal malfunctions	Pigs, <i>humans</i>	Eating of beetles or parts of them containing larvae	Europe, Asia
Pentastomids <i>Linguatula serricata</i>	Typical anterior pole with rows of hooks	Pentastomiasis/albendazoles	Severe liver problems	Dogs, <i>humans</i>	Eating undercooked, larvae-containing meat	Europe, Asia

Fig. 10.1 Macrophoto of a pupa and an adult of the gray flesh fly *Sarcophaga carnaria*



10.3.1 Prions with a Zoonotic Potential

Prions—an abbreviation of the description as proteinaceous infectious particles—are no true living organisms but wrongly folded intracellular proteins inducing other proteins to do the same under peculiar not yet well-understood conditions (Prusiner 1998; Rabenau 2009). These wrongly folded proteins, which e.g., in the case of the BSE-epidemiology (Bovine Spongious Encephalopathy) in Great Britain asked a death toll of more than 200 persons and left hundres other victims with severe brain damages besides a loss of more than 500,000 cattle, which had been killed either due to acute infections or due to preventive measurements. Since there was a clear proof for transmission from cattle to humans these diseases were also called transmissible encephalopathies. Similar degenerative symptoms may also occur due to hereditarily transmitted destructions in the genome of some families, but are rare compared to the wave of infections that rolled on after the feeding of animal meet powder (produced from BSE-infected animals) to uninfected ones. The reason was that no one had expected that such powdered animal meat should have been heated up to at least 141 °C in order to destroy these aggressive wrongly folded proteins, while normal cell proteins degenerate mostly already at 60 °C.

In nature there exists a broad potential of transmission of such infectious proteins from one host to the other. This was shown by the experiments of our group (Post et al. 1999). During these experiments *Sarcophaga carnaria* fly larvae were fed with brains of Scrapie-infected hamsters (see Table 10.2). One to seven days later these maggots were squeezed and the minced material was orally inoculated to uninfected hamsters. The same was done with pupae and adult flies (Fig. 10.1) obtained from the originally infected fly larvae. In all cases the hamsters showed 3–5 months after the experimental infection the full range of symptoms of BSE respectively Scrapie-disease including the full loss of motility control. Furthermore they were all prion-positive as was shown by molecular biological tests.

These experiments strongly supported the supposition that apparently these wrongly folded proteins are parts of nature being transmitted since long (probably without producing broad outbreaks but only single fatal cases). Such a transmission scenario would be given e.g., in cases when fly larvae feed on the remnant placenta of an infected animal and were later engorged by other grass feeding ruminants.

Fig. 10.2 Macrophoto of the adult green flesh fly *Lucilia sericata*



Such a transmission will also explain the astonishing findings during the BSE-outbreak in Europe that some cows that had definitely not been fed with animal meat powder were positively tested for BSE.

However, evaluating all these aspects together, it can be stated that prions (Table 10.2) will not be a considerable threat for human health. Under natural conditions (i.e., when ruminants are fed by normal grass or plants) the natural infection rate of those animals with prions will never lead to such a significant increase of the amounts of prions inside the ruminant populations, that this later could lead to a significant infection potential.

10.3.2 *Virus-Borne Zoonotic Diseases*

The term virus has its origin in the *Latin* word virus, which means mucus, poison, moisture. Viruses themselves are no living organisms but are obligatory based as intracellular pathogens on the reproduction machinery of their host cells, which is situated in the nucleus and along the ribosomes. Therefore successful viruses had developed methods to become firmly attached to potential host cells, to enter such cells, and to find their way to the steering system, where their genetic material—RNA or DNA (in species specifically varying arrangements)—might be reproduced and finally released from the invaded cell. Thus for viruses it is most important to reach the surfaces of such cells otherwise they will burst or become digested by cells of the defense systems of attacked hosts. Therefore the viruses have developed some major pathways of transmission (Table 10.3):

- Transmission by vector bites (Fig. 10.3)
- Transmission by feces of vectors (Figs. 10.1, 10.2, 10.3, 10.9a, b)
- Transmission by feces of infected hosts
- Transmission by contact or by inhaling of excretions or raw meat of infected hosts

While it is much easier for humans to avoid the last three transmission pathways using normal hygienic preventive methods, the contact to vectors occurs practically unlimited—at least in low numbers. Therefore the transmission and propagation of the pathogens of the so-called *emerging diseases* (Mehlhorn 2012a) increases

Fig. 10.3 Macrophoto of an *Aedes* mosquito sucking blood on human skin



constantly under the recent worldwide accelerating conditions of globalization and global warming (i.e., local change of the climate and huge migration and traveling activities worldwide; Dobler and Aspöck 2008, 2010; Neumeister et al. 2009).

Table 10.3 summarizes important viral diseases of humans which are discussed in more detail in Sects. 10.4–10.7 (Aspöck 2010; Dobler and Aspöck 2008, 2010; Darai et al. 2009; Löscher and Burchard 2010; Neumeister et al. 2009). The existence of viruses and their high potential to create new combinations, however, endangers life on earth, since they are easily transferred by genetic variations into highly dangerous pathogens. Due to their reproduction inside host cells there are only very restricted possibilities of chemotherapy apart from vaccination trials, which, however, are very often not successful, since many viruses often change their surfaces that have to be recognized by vaccines.

10.3.3 *Bacteria of Zoonotic Importance*

The present life on earth would not be possible without bacteria. They are found everywhere in nature, inside body cavities, and they even may settle inside cells. Since long some of these bacteria have undergone (as our mitochondria) a close symbiosis with all living organisms. However, a broad spectrum of these organisms that are invisible with naked eyes, may also introduce severe diseases, which in times of worldwide epidemics and pandemics of plague or cholera reduced mankind significantly in many regions. For example, in Europe the population was reduced during the so-called 30 years-war (1618–1648) down to 15 % mainly by plague epidemics. Even today the bacteria of tuberculosis or cholera request millions of victims every year.

It is not long ago when both “popes of bacteriology,” the German *Robert Koch* (1843–1910) and the French *Louis Pasteur* (1822–1895) and their famous scholars started to enlighten the hidden world of “good and bad” *bacteria* and to develop preventive measurements, which later were supported by the invention of the sulfonamides by the German Bayer Company scientist *Gerhard Domagk* (1895–1964) and the penicillin by the Scottish scientist *Alexander Fleming* (1881–1955). Both got (1939: Domagk and 1945: Fleming) the Nobel Prize for Medicine. However, the bacteria struggled back by a constant and often very quick

development of resistances, so that today many bacterial diseases are far from being under control (and even may start a new career as “emerging diseases.”

Table 10.4 summarizes a selected spectrum of bacterial diseases, which are spread between humans and animals in their surroundings. The peculiar pathways of transmission are discussed in the Sects. 10.4–10.7 of this article (Alam and Zurek 2004; Bielaszewska et al. 2011; De Jesus et al. 2004; Ekdahl et al. 2005; Emerson et al. 2004; Fischer et al. 2001; Fotedar 2001; Fotedar et al. 1992; Grübel et al. 1999; Hold et al. 2007; Karch 2005; Kobayashi et al. 1999; Mian and Jacal 2002; Nichols 2005; Olsen and Hammack 2000; Rasko et al. 2011; Sasaki et al. 2000; Szalanski et al. 2004). Again the vector-transmitted pathogens of this group are most difficult to control, since the bites of the mostly lonely acting blood suckers (ticks, mites, mosquitoes, tabanids) or contact to skin lickers (flies) can only hardly be avoided.

10.3.4 Fungi with an Anthropophilic Zoonotic Potential

Fungi, the name of which has its origin in the *Latin* word: fungus = mushroom are heterotrophic organisms, which are covered by a chitin containing outer layer consisting of a network of fibrils. Most species of the fungi belong to the free nature, settle and feed at moist places. Several species of the fungi (*French*: champignons) are eatable.

However, some of the fungi species (see Table 10.5) invade body cavities of humans or animals respectively spread on their skin—mostly at places, where a high humidity is guaranteed as it is in the case at the hair-covered scalp, below finger or toe nails, in the groins, or in the shoulder-hollows. These places will be invaded especially in patients with immune suppression and can be treated only with difficulties (Figs. 10.4 and 10.5). This is due to the hidden places of the infection, the poor penetration of chemical substances through the surface of the fungi and due to a general lack of really potent fungicides with low side effects. The main transmission pathways are contacts to contaminated surfaces, direct contacts to contaminated human skin or to hair of infected animals. Other possibilities of transmission of fungi from human or animal feces or from other excretions are given via contaminations of food or materials (Abbot 2002; Banjo et al. 2005; Darai et al. 2009; Neumeister et al. 2009).

10.3.5 Parasites with a Zoonotic Life Cycle

Parasites are per definition in a strict sense animals that live on costs of other animals. Of course there also exist plants that stay as parasites on other plants, but they are here not considered, since they do not harm the health of humans. The name “parasites” has its origin in the *Greek* word “parasitos,” which described

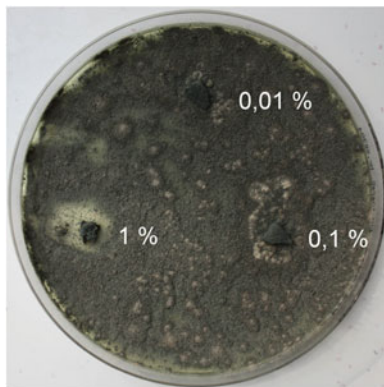


Fig. 10.4 Macrophoto of a culture of *Aspergillus fumigatus* which may enter into the lung of mammals after injection of three different concentrations of the neem-seed derived extract MiteStop (Fa. Alpha-Biocare). Note the clear, doses dependent spots of clearance

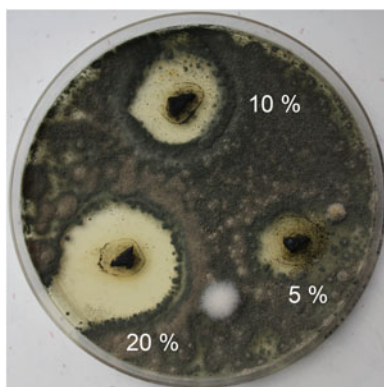


Fig. 10.5 Macrophoto of a culture of *Aspergillus fumigatus* which may enter into the lung of mammals after injection of three different concentrations of the neem-seed derived extract MiteStop (Fa. Alpha-Biocare). Note the clear, doses dependent spots of clearance

“employees” at noble courts that had to taste the food for poisons and thus were nourished on foreign costs. The parasites attack animals and humans. However, although many parasitic species rely on many hosts (e.g., *Toxoplasma gondii*), some had become specialists in the last 10,000–20,000 years and have adapted themselves as specialists (e.g., *Ascaris lumbricoides*) to the “late runner” on earth, i.e., the *Homo sapiens*. Parasites occur in many phyla of the Zoological Kingdom. Thus they are found among the unicellular organisms (Protozoa), among animals with a reduced number of cells (Myxosporida), among the various groups of worms (Platyhelminthes, Nematelminthes, Acanthocephala, Pentastomida, leeches), and among the billion-headed crowds of ticks, mites, and insects. The members

of the latter three groups may also act as *vectors* of different pathogens (prions, viruses, bacteria, fungi, and parasites; see Tables 10.2, 10.3, 10.4, 10.5, 10.6). This transmission might be done just mechanically by means of contaminated mouthparts, by body contacts, and/or by placing pathogen-contaminated feces onto food, skin, or wounds. However, it may also occur as result of a defined developmental cycle with a reproduction of the pathogen inside the vector. Very often such reproduced pathogens even enter the eggs inside the female ovary or uterus so that the next vector generation is already carrier of the once ingested pathogen (i.e., this occurs inside ticks when the viruses of the spring-summer-meningoencephalitis or the kinetes (=motile zygotes) of the protozoan *Babesia* species enter the not yet fertilized eggs. Having entered the egg the parasites are firmly included therein and the thick eggshell, which is formed after fertilization and thickened during the laying process by adding a layer of wax, protects the growing tick larva and the therein included pathogens from desiccation. Examples of important zoonotic life cycles are compiled in Table 10.6) (Aspöck 2010; Darai et al. 2009; Getachew et al. 2007; Greenberg 1973; Kollaritsch and Paulke-Korinek 2010; Mehlhorn 2008; Mehlhorn et al. 2011; Mehlhorn 2012a, b, c; Oyerinde 1976; Szostakowska et al. 2004).

10.4 Food-Borne Zoonosis

When taking into consideration a very wide definition of the term *food-borne zoonosis* any pathogen that occurs inside or on meat of animals (inclusive fish) respectively is found within milk would fit into this group. However, in this case the differentiation from *contamination-borne zoonosis* would be rather difficult. Therefore it seems more reasonable to draw the limitations much stronger. This, however, leads to the fact that only a few groups of pathogens could be placed into this category. The prions—especially those of the bovine spongious encephalopathy (BSE)-group—would surely find their place here, since they introduce disease as well in animals as in humans after eating meat (even cooked one) of infected animals (Table 10.2). The viral pathogens with a zoonotic potential are mainly transmitted by bites of arthropods (mites, ticks, insects) or by fecal contamination, and only rarely—if at all—by infected food of humans and/or animals (Table 10.3). The explanation is that viruses need for their reproduction the very well-adapted DNA/RNA machinery of a living cell, which has problems to survive the passage of the stomach and intestinal system of humans (Altekrues et al. 1997; Darai et al. 2009; Neumeister et al. 2009; Steinmueller et al. 2006; WHO 2002a, b).

Likewise most bacteria prefer pathways of transmission other than the direct inclusion inside organs of warm- or cold-blooded animals respectively, fish. Thus there are not many bacteria like those of the *Brucella*-, *Listeria*- or *Salmonella*-groups that are frequently found not only on contaminated meat but also in milk (see Table 10.4).

Fungi with a zoonotic potential (Table 10.5) have no direct food-borne transmission pathways apart from fecal contaminations, that are common pathways in the species of the *Blastocystis*-group (if these species are really accepted as a fungus—see the other chapters).

On the other hand food-borne transmissions are very common in parasites (Table 10.6). There are protozoans such as several *Sarcocystis* species (Fig. 10.6) and *Toxoplasma gondii* which develop intracellular stages that later are transmitted, if this infected tissue is swallowed by a predator (inclusive humans). Then it starts a further developmental phase there. Man might act as *final host* (where the sexual process is running in his intestine, e.g., in *Sarcocystis* species) or as *intermediate host* with an exclusively asexual reproduction of the parasite in his tissues (e. g. in *Toxoplasma gondii*).

Also among the trematodes (phylum Platyhelminthes) there are many examples of food-borne parasitosis in humans. In general humans are final hosts, since the adult worms live in their intestine (e.g., *Fasciolopsis buski*), in their lung (*Paragonimus* species), or in their liver (*Clonorchis sinensis*, *Opisthorchis* species, *Fasciola hepatica*, etc.; Table 10.6). The infection of humans with stages (metacercariae) of trematodes occurs by eating infected raw or undercooked intermediate hosts (e.g., fish, crustaceans) or plants with metacercariae that had been attached there by intermediate hosts (e.g., snails; Mehlhorn 2008).

For tapeworms (Cestodes; phylum Platyhelminthes) humans might be final or intermediate hosts. In the case of the large human tapeworms *Taenia saginata*, *T. solium*, *T. asiaticum* and *Diphyllobothrium latum*, humans act as final hosts and shelter in their intestine the several meter long, hermaphroditic adult worms after being infected by ingestion of larval stages (cysticercus in the case of *Taenia* species respectively plerocercoids of *D. latum*) inside undercooked meat or fish. In the case of *T. solium* humans can be both final and intermediate hosts, since the cysticercus larvae may also develop inside their muscles and/or brain after they had orally ingested worm eggs from human feces (Mehlhorn 2008, 2012c).

On the other hand, however, in the cases of the tiny dog, respectively, fox tapeworms *Echinococcus granulosus* and *E. multilocularis* humans remain exclusively intermediate hosts, within which large cysts develop. Transmission of tapeworms of dog or fox to humans does only occur via the fecal contamination pathway when ingesting worm eggs from feces of dogs or foxes. However with respect to the general life cycle of these tapeworms humans are “dead ends,” since in general they are not eaten by the final hosts (dogs, foxes).

Among the nematodes there are rather few examples for food-borne worm infections in humans with respect to the huge number of nematode species. However, one prominent example is given by the *Trichinella* species, of which up to now seven had been described. These worms live as adult, rather tiny worms (♀, ♂) of only 1–3 mm in length inside the intestine of predators (inclusive man) (Fig. 10.7). The females produce larvae; however, these stages do not leave this infected individual (i.e., acting here as final host), but they penetrate into the muscle cells, where they stay until another predator feeds such infected meat. Thus these predators are final and intermediate hosts at the same time and free stages do not occur outside a body (Mehlhorn 2008).

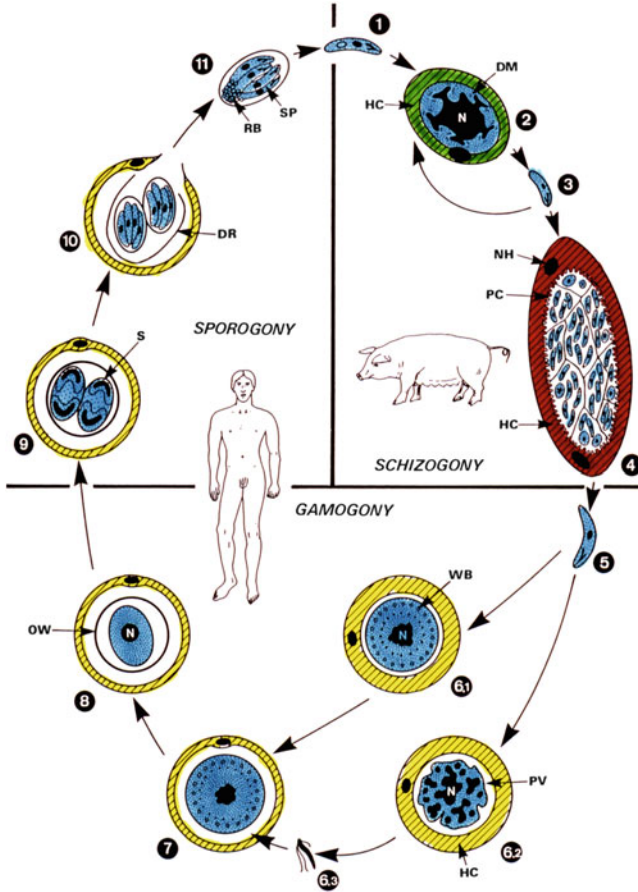


Fig. 10.6 Life cycle of *Sarcocystis suihominis* with two obligatory hosts. 1 Motile sporozoites hatch from the ingested sporocysts inside the intestine of the intermediate host, i.e., swine. 2 Two generations of schizonts are formed (5–6 and 12–17 days after infection) inside endothelial cells of blood vessels, giving rise to 50–100 merozoites by endodyogeny. 3 Free motile merozoites; first-generation merozoites enter other endothelial cells and form schizonts, whereas merozoites of the second generation induce formation of tissue cysts. 4 Cyst formation inside typical cells (muscle fibers, brain cells); within these cysts the parasites are reproduced by repeated endodyogeny leading to thousands of cyst merozoites which are situated inside chamber-like hollows. 5 When the final host man has eaten cyst-containing raw or insufficiently cooked meat, the cyst merozoites are set free and enter cells of the lamina propria. 6 Formation of female (macrogametes, 6.3) via gametes (6.1, 6.2) within 14 h of infection. 7 Fusion of gametes. 8 Formation of the oocyst wall around the zygote. 9–11 Formation of two sporocysts (containing four sporozoites each) inside the host cell. The smooth oocyst wall often becomes disrupted. Thus, fully sporulated oocysts are found in the feces (11). *DM* developing merozoites; *DR* disrupted oocyst wall; *HC* host cell; *N* nucleus; *NH* nucleus of the host cell; *OW* oocyst wall; *PC* primary cyst wall; *PV* parasitophorous vacuole; *RB* residual body; *S* sporocyst; *SP* sporozoite; *WB* wall-forming bodies



Fig. 10.7 Light micrograph of a muscle fiber of a rat containing three larvae of *Trichinella spiralis*

Fig. 10.8 Light micrograph of the muscles of a fish containing many *Anisakis* larvae



In the case of the species of the genera *Anisakis*, *Porrocaecum*, *Contracaecum* (Fig. 10.8), and related worms humans are intermediate hosts. Man, however, is not relevant for the transmission. He is a “dead end,” since the adult worms live in the intestinal tractus of large marine mammals after they had ingested the larvae inside fish. Humans do the same: they eat raw fish containing these worm larvae, which live for a short time (1–3 weeks) in the human intestinal system before they die. Similar infections are also possible, when humans eat raw meat of reptiles; then they may ingest also larval pentastomids which finally may grow up to considerable size in humans (Mehlhorn 2008, 2012c).

10.5 Vector-Borne Zoonosis

Vectors in the sense of parasitological terms are animals that are able to transport pathogens from one host to another, while these vectors search their food on such hosts (Abbot 2002; Aspöck 2010; Hald et al. 2008; Holt et al. 2007; Löscher and Burchard 2010; Neumeister et al. 2009). The simplest way of such a transfer is the

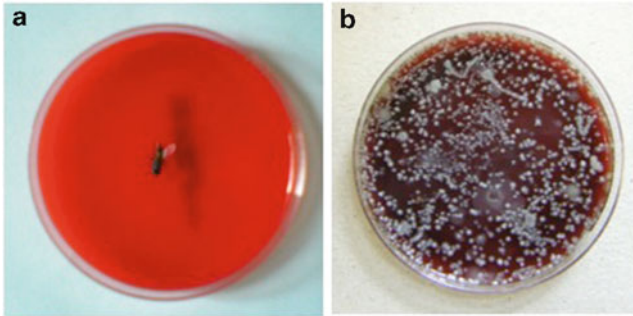


Fig. 10.9 Photograph of a petri dish filled with bacterial culture medium. Onto the surface of this petri dish a *Musca* fly was placed for 30 s (a). The photo at the right (b) shows the growth of bacteria 2 days after the exposition. This proves that the feet of this fly had been highly contaminated with bacteria

Fig. 10.10 Two adult *Ixodes* ticks laying eggs, which already may contain the viruses of the spring-summer meningoencephalitis



transport of pathogens that glue at mouthparts, at feet, or at the body surface of such vectors (Figs. 10.1, 10.2, 10.3, 10.9, 10.10, 10.11, 10.12), which are mostly mites, ticks, mosquitoes, flies, midges, simuliids, tabanids, leeches, etc. This type of transmission is described as mechanical, since the pathogens are not multiplied on the vectors. For example, if 800 bacteria of a special species glue at the fine hair of a fly, the transmission maximum would be 800, but in general the finally transmitted amount is much less. Nevertheless this method might be still highly effective, if it is considered that many bacteria (such as e.g., toxic or hemorrhagic strains of *Escherichia coli* (EHEC, ETEC) need only an initial dose of 20–30 specimens to start severe infections. Recent studies examined various fly species of the genera *Musca*, *Calliphora*, *Sarcophaga*, *Lucilia*, etc. for their “normal load” of bacteria and parasites (protozoans and worm eggs). These flies had been caught close to stables of pigs, horses, cattle, rabbits, chickens, dog ponds respectively close to human recreation sites inside and outside of towns. Astonishing masses and varieties of pathogens were found on the examined flies. These studies of Förster et al. (2012) and Gestmann et al. (2012) confirmed earlier ones (Förster 2009; Förster et al. 2007, 2009) showing that more than 100 important bacteria species and more than 15 infectious stages of parasites might be transmitted via flies as vectors.

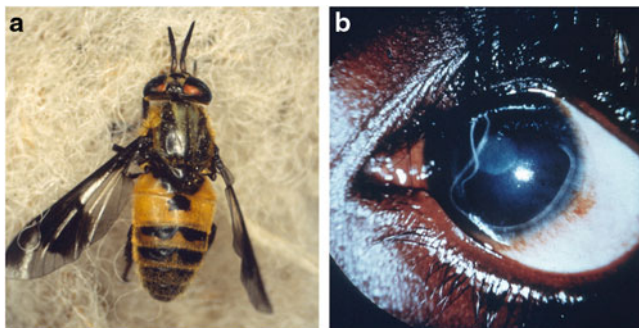


Fig. 10.11 (a, b) Photograph of a tabanid of the genus *Chrysops* and a wandering worm (*Loa loa*) in an eye (b, right)



Fig. 10.12 Scanning electron micrograph of a black fly (*Simulium morsitans*), the vector of *Onchocerca volvulus* worms

Additional experimental transmission experiments showed that practically all types of bacteria were also transferred to bacterial culture plates, if the flies had contact for only 20–30 s to bacteria on a culture plate. In another series of experiments single flies were placed onto a fluid containing a definite number of *Toxocara canis* eggs. Then these flies were washed and squeezed (to get the intestinal fluid). When counting the obtained eggs from these flies the astonishing result was noted that merely two-thirds of the original number was found again.

These experiments clearly show that the importance of licking flies as mechanical vectors is apparently underestimated.

The same should be true for biting and/or blood sucking mites, ticks, mosquitoes, flies, fleas, lice, tabanids, etc., since most of them are able to switch quickly from one host to another, so that many tiny blood droplets remain liquid thus keeping the pathogens in an infectious status.

The situation becomes even worse in those cases, where a regular reproduction of the pathogens has been developed inside the vectors during evolution. Such reproductive processes occur regularly in vectors bearing special species of viruses, bacteria, and parasites (see Tables 10.3, 10.4, 10.6). Since these vectors even are able to

transmit the pathogens into their eggs as e.g., the spring–summer–meningoencephalitis virus in ticks (Fig. 10.10), the distribution intensity is enormously enlarged not only by the flying activity of the vectors but also by the transmission into their progeny.

Therefore flying vectors that give pathogens a chance to reproduce inside their bodies represent a hidden risk, since they may help to spread severe epidemics. The consequences for mankind would have been terrific, if mosquitoes, tabanids, or ticks would be able to transmit the AIDS viruses as easy as they do it with other viruses.

Nevertheless the various vector-borne zoonosis demand even today a high death toll in many regions of the world. Considering the recent enormous increase of the Dengue-virus propagation by *Aedes* mosquitoes, the progress of the West-Nile-Virus into many countries as well as the still not yet solved malaria situation or filarial worm problems new and concentrated control measurements are highly needed. Especially in times of global warming and intense globalization epidemics may quickly become pandemics, which will not stop at the borders of countries, where the population believe even today to live on “safe grounds.”

10.6 Cyclic Zoonosis

This term comprises pathways of transmission of agents of diseases that run repeatedly in a fixed direction and are based on at least two different hosts, where always defined developmental processes take place (Mehlhorn 2008). With respect to these conditions the transmission pathways of the malarial, babesial, and sarcosporidian parasites would fit in this category, because in these life cycles asexual processes like merogony (schizogony) start in a first host (human tissues) and are followed by gamogony and sporogony in a second host, which belongs—depending on the species—to the group of vectors (mosquitoes, ticks) or to mammalian species (as it is the case in the *Sarcocystis* species; Table 10.6, Fig. 10.6). Although the life cycle of the trematodes fits into this category, since, for example, the adult schistosomal worms live in the blood system of humans and other mammals, while the asexual reproduction (via sporocysts and the finally infectious cercariae) takes places inside the tissues of water snails (Table 10.6). The life cycles of tapeworms belong to this group, too. Only if humans eat the *cysticercus-larva* with raw meat or the *plerocercoid-larva* in raw fish, the adult worms can develop from these ingested larval stages inside the human intestine (Table 10.6).

Among the nematodes examples of cyclic zoonosis are much more rare than in trematodes and cestodes (Aspöck 2010; Mehlhorn 2008). However, especially the filarial worms have developed very sophisticated developmental cycles, which need a very tight coordination in the different biotopes, if the transmission of the parasite from one host to the other should be successful. In the case of the filarial worms *Wuchereria bancrofti*, *Brugia malayi*, and *Loa loa* the adults live either in the lymph nodes or in the subcutaneous tissues (*Loa loa*), while their sheathed larvae

(=microfilariae enveloped by the egg shell) stay in the blood. However, since these three worm species use different vectors with different behaviors and activity periods, the larvae of these worms are concentrated at different day times in the peripheral blood vessels. *Wuchereria bancrofti* and *B. malayi* are transmitted by nightly active mosquitoes, thus their larvae are found in the peripheral blood mainly at 10 p.m. On the other hand *Loa loa* is transmitted by day time active tabanids (*Chrysops* species; Fig. 10.11). Thus their larvae are found in day-time from 1 p.m. to 3 p.m. in the peripheral blood vessels. The adult worms of *Onchocerca volvulus* (accumulations of their dead larvae in human eyes induce the so-called river blindness) stay in humans inside the subcutaneous tissues, where the females are often found in groups inside nodules. The infectious (unsheathed) larvae stay all day long in the lymph fluid of the skin system. There they are taken up by the simuliids (black flies; Fig. 10.12) which do not have as fine mouthparts as the mosquitoes, but scissor like ones, which lead to the formation of small “lacunes” of blood and lymph in the skin. Such an all-day-presence of the larvae in the skin makes sense for *Onchocerca* larvae, since the simuliids only suck blood during day time.

Besides all differences in their behavior inside humans the first larvae (=microfilariae) of all four species described above proceed a nearly identical development inside their vectors. Having reached the insect’s gut the larva 1 enters the body cavity of the new host, grows up there (while molting twice), and the larva 3 enters finally the head of the vector. If this infected insect starts a new blood sucking action, the relatively thick larva 3 perforates the connecting membranes of the different mouthparts, creeps outside down to the biting site at the human skin and enters there the body of the new host.

Although this cyclic development seems very complicated, it is so perfect, that even today more than hundred fifty million humans in the tropics suffer from diseases due to these worms, since available means of chemotherapy are still rather poor.

10.7 Contamination-Borne Zoonosis

The diseases categorized inside this group are characterized by the fact that both humans and animals carry these pathogens, but their cross transmission occurs more or less occasional and the direction of the transmission is not fixed. This means that humans may get the pathogens from animals and that also the opposite way is possible (Tables 10.2, 10.3, 10.4, 10.5, 10.6). Examples for such contamination-borne zoonosis can be found in all groups of pathogens, which in addition also may use other pathways of transmission (WHO 2002a, b; Mehlhorn 2008).

Rabies viruses can be transmitted directly by saliva during bites of infected animals, by blood transfusion, or by contact to virus-contaminated saliva. Many bacteria change the host if there is contact either to human or animal feces or to contaminated food. The same is true for several species belonging to the group of fungi and thus also for the different *Blastocystis* species. Among the non cyclically transmitted parasites the species of the genera *Giardia* and *Cryptosporidium* as well

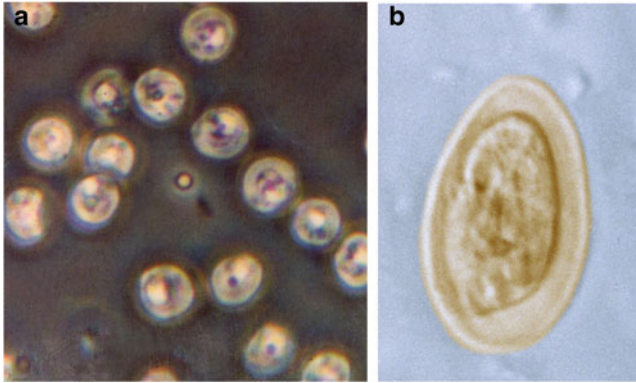


Fig. 10.13 Light micrographs of cysts of *Cryptosporidium* sp. oocysts (a) and a four-nucleated cyst of *Giardia duodenalis*. Such cysts had been excreted by infected animals in lakes in Milwaukee respectively Sydney. Humans became infected after drinking contaminated water and mass epidemics occurred

as the different microsporidian species (Table 10.6) belong to the group of contamination-borne zoonosis. In all these cases fecal stages (cysts, oocysts, respectively, spores) might contaminate food by direct contact with feces or by help of the transportation activity of licking flies (Table 10.6). But even in cyclically transmitted parasites such as *Trichinella spiralis*, *Plasmodium* species, *Trypanosoma* species, *Babesia* species, and *Leishmania* species mechanical transmission of the different pathogens may occur by contaminated mouthparts of large sized blood suckers (tabanids, leeches, blood licking bats) or even more frequently by transfusion of blood obtained from semi-immune donors with a low and thus only hardly detectable parasitemia.

Of course all these contamination-borne transmissions cannot become completely avoided—especially not in rural regions, however, strict hygienic conditions—as far as reasonable—will limit large outbreaks, as they had occurred in the USA or Australia when hundred thousands of humans had used drinking water that was contaminated with oocysts of *Cryptosporidium* species respectively with cysts of *G. lamblia (duodenalis)* (Fig. 10.13a, b).

Therefore preparation of safe drinking water will be worldwide one of the most prominent tasks of the future.

The outbreak of a kidney destroying disease due to hemorrhagic strains of *Escherichia coli* (EHEC, EPEC, etc.), which in 2011 took its start somewhere in Germany, spread due to travelers into many countries within weeks, and killed more than 300 persons, showed that the populations in crowded cities are highly vulnerable. Even today (i.e., 1½ years after the start of an intense search for the source of this epidemic), it is not completely finished and it remains unsolved, whether the finally detected contaminated soja sprouts had been the only source.

Fig. 10.14 Skin hollow (a) and the therefrom extracted second stage larva of *Dermatobia hominis* (b).



10.8 Myiasis

Myiasis, the name of which has its origin in the *Greek* term *myia* = fly, midge, is a very peculiar form of zoonosis (Mehlhorn 2008). Around the world there exists a group of flies, which do not proceed their larval development in debris of any kind or even not in dead bodies as it is done by many fly species, but which place their eggs or freshly hatched larvae onto the skin of animals and humans. These larvae bore— anterior end with the two mouth-hooks forward—into the skin. Exclusively the terminal end of the penetrated larva with its two terminal plates of the tracheal = oxygen uptaking system has contact to the surface. In order to avoid falling out unwillingly from their feeding hollow, these larva developed often very stiff circularly arranged hooks along their different segments. This peculiar appearance led to the description of these larvae as “screw worms.” Some of these fly larvae are specialists and attack exclusively animals (e.g., *Haematobia bovis*). Others enter both humans and animals (e.g., *Oestrus* species), while again others have developed a peculiar preference for humans (*Dermatobia hominis*; Fig. 10.14).

Especially *D. hominis* has developed an unbelievable way of superinfection. The adult females catch while flying, a female mosquito of the blood sucking group and attach a bundle of own eggs on the body of this insect. As soon as this mosquito has a touch down on a host, the *Dermatobia* larvae hatch immediately during the sucking phase of their transporter mosquito and enter the skin— anterior end forward. After two molts the larva 3 finally leaves the inflamed hole in the skin, drops down to earth, and pupates there within a short time. Depending on the outside temperature the development of the adult stage inside the pupa takes only a few days, so that propagation can go on. In this case a broad spectrum of animals and the *Homo sapiens* are involved in a very effective, sophisticated manner. Thus these flies use mosquitoes and motile mammals (humans) to enlarge the spectrum of their biotopes.

10.9 Conclusions

The health of humans and their animals are threatened by a big bunch of pathogens, which have established during coevolution with their hosts a broad spectrum of pathways of transmissions. They all together belong to the group of zoonosis, where ping-pong infections occur between humans and animals. These infections might occur *directly* via contact to pathogen-containing feces, contaminated drinking water, or infected food. The *indirect* methods of transmission are based on the activity of so-called vectors, which transmit pathogens during blood sucking, while licking on skin and/or lips of humans or by contamination on human food.

With respect to these well-established pathways, the use and amelioration of measurements to avoid such transmissions is of high importance especially in increasing populations of animals and humans. Thus the following measurements have high priority in the near future:

- Preparation of safe, noncontaminated drinking water for humans and animals
- Quick clearance of human and animal feces
- To fight against possible vectors of pathogens in human houses and in stables of animals
- To use, wherever possible, vaccines and to develop new ones
- To establish a regular international control system to detect (at the very early moment) possible transmissions of pathogens and thus avoid outbreaks of epidemics and pandemics
- To keep masses of farmed animals in closed systems under most effective hygienic conditions
- To inform regularly farmers, leaders of institutions, veterinarians, physicians, and also the public on recent protection methods
- To intensify at the search for new compounds and new methods to control those pathogens, where treatment is not yet possible or where pathogens have developed resistances

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