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Bacterial and Parasitic Hazards and Consumption of Meat

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Average meat consumption in the European Union now stands at 84 kg of meat per capita per year, with pork accounting for 41 kg, poultry 28 kg and beef 15 kg (FranceAgriMer 2019). The global average is around 43 kg of meat per capita per year, representing a total consumption of more than 330 million tons in 2020 compared with 67 million tons in 1957, a 5-fold increase in 60 years, according to the FAO. This figure is expected to continue to rise, reaching 470 million tons in 2050, with 76% of this increase coming from emerging countries. In China, meat consumption rose from 7 million tons in 1978 to 86 million tons in 2017, including 55 million tons of pork.

While excessive meat consumption is nowadays increasingly associated with an increased risk of developing certain cancers, such as colorectal cancer and also breast cancer (Dialo et al. 2019), meat has long been considered a potential source of biological hazards responsible for foodborne diseases (Fegan and Jenson 2018). Meat-producing animals can carry bacteria, parasites or viruses and zoonotic agents, which can contaminate meat consumers. Meat can also serve as a vector for pathogens of human origin that contaminate meat during meat processing or preparation operations.

Hazards in the Food Processing and Distribution Chain,
coordinated by Nabila HADDAD.

According to European Regulation 853/2004, the term “meat” is defined as any edible part of all production animals (domestic ungulates, poultry, rodents, lagomorphs, wild game and farmed game), including blood and offal. In this chapter, we will narrow this definition by equating meat with the muscle flesh and offal of the main production animals: ruminants, pigs and poultry.

According to data published by the EFSA (European Food Safety Authority 2018) on foodborne outbreaks reported by the member states of the European Union between 2010 and 2017, meat, all species combined, is at the origin of almost 20% of cases in which the agent responsible has been identified, making “meat and meat products” the food category most frequently involved in food-poisoning outbreaks in the European Union.

1.1. Meat contamination

The biological hazards transmitted by meat are of different kinds: bacteria, viruses, parasites, and unconventional transmissible agents (UTAs). Table 1.1 shows the main hazards potentially transmitted by meat consumption, according to the main modalities of meat contamination.

Hazards located inside the digestive tract of food-producing animals, transmitted to meat through fecal contamination	***Campylobacter jejuni/coli*** ***Salmonella enterica*** **EHEC** ***Listeria monocytogenes*** ***Clostridium botulimun*** ***Clostridium perfringens*** *Staphylococcus aureus* *Yersinia enterolitica*
Hazards located inside the digestive tract of food-producing animals, transmitted to meat by bacteremia digestive in origin	*Clostridium perfringens* *Clostridium botulinum* *Salmonella enterica* *Listeria monocytogenes*

Hazards located inside the muscles or organs of food-producing animals	***Cysticercus bovis*** ***Cysticercus cellulosae*** ***Toxoplasma gondii*** ***Sarcocystis hominis*** ***Sarcocystis suihominis*** ***Trichinella spiralis*** **Prion ESB** **Hepatitis E virus (genotypes III and IV)** ***Yersinia enterolitica*** ***Mycobactérium bovis*** ***Bacillus anthracis*** ***Brucella* spp.** *Listeria monocytogenes* *Staphylococcus aureus*
Hazards present in humans: meat contamination during handling	***Staphylococcus aureus*** **Human enteric virus (Norovirus, Hepatitis A virus, Rotavirus, Hepatitis E virus (genotypes I and II))** *E. coli* pathogènes *Salmonella enterica*
Persistent hazard in the production environment: contamination during the manufacturing process	***Listeria monocytogenes*** *Salmonella enterica* *Campylobacter jejuni* *Yersinia enterolitica* *Staphylococcus aureus*

Table 1.1. *The biological hazards transmitted by meat and how they are contaminated. The main meat contamination the modality for each agent is indicated in bold*

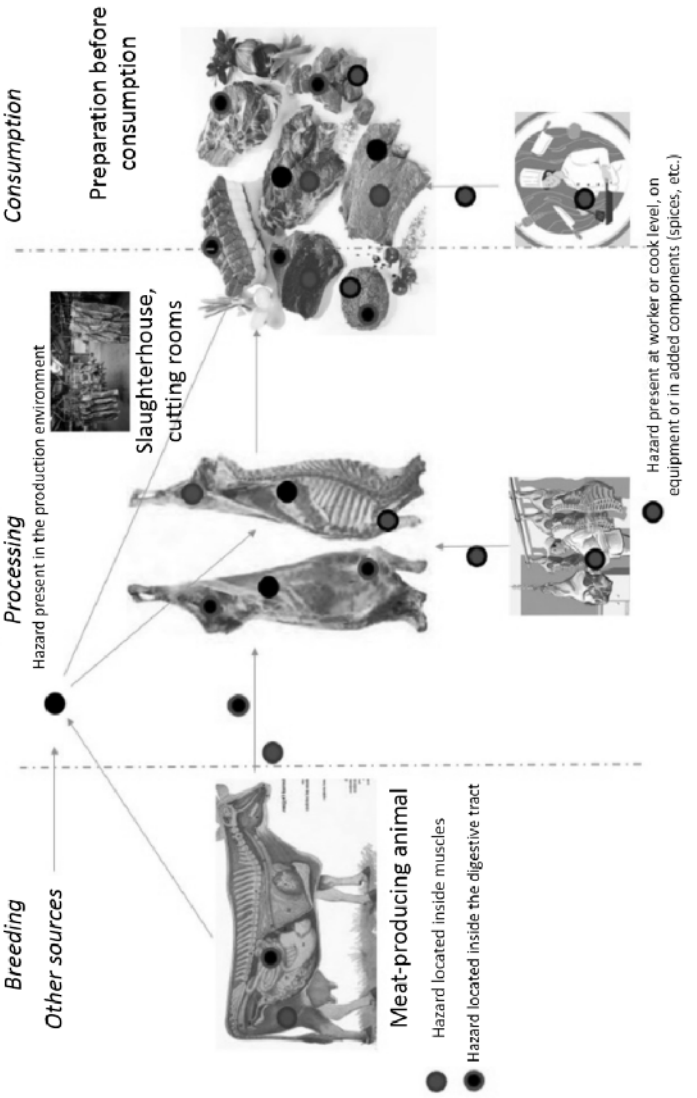

Figure 1.1. *Meat contamination routes throughout the food chain. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

Meat contamination can occur through different routes, as illustrated in Figure 1.1. If we apply the 5M method, the origin of the contamination may be the animal itself (Material), staff handling the meat (Manpower), the equipment used for preparation (Machine), handling errors (Method) or the environment in which operations are carried out (Milieu).

1.1.1. *Contamination by the food-producing animal*

The food-producing animal can carry hazards during its lifetime. This can of course occur in infectious diseases that are accompanied by septicemia, therefore in diseased animals that will normally be excluded from the food chain during *ante*- and *post-mortem* health inspections, when the disease is accompanied by clinical signs or lesions (e.g. systemic tuberculosis by *Mycobacterium bovis,* Anthrax by *Bacillus anthracis,* Brucellosis by *Brucella* spp., etc.). These zoonotic hazards, which are not only transmitted to humans through food but also through inoculation or inhalation, are not detailed in this chapter.

This particular case aside, two main localizations can be responsible for meat contamination. First, the hazard may be present in the muscle masses of the living animal or in other organs. This is exclusively the case for parasites whose cycle involves muscle localization of larval forms that will be responsible for contamination of the meat consumer (*Cysticercus bovis, Cysticercus cellulosae, Trichinella* spp*., Toxoplasma gondii, Sarcocystis hominis* and *suihominis*). It is also the case for the Bovine Spongiform Encephalopathy (BSE) prion, localized in the central nervous tissue.

Bacterial hazards can also be responsible for in vivo organ contamination. This is the case with *Yersinia enterolitica*, potentially present in the head lymph nodes in pigs, or the hepatitis E virus, present in the livers of pigs. In addition, in the event of mastitis of *Staphylococcus* in dairy cows, the presence of the bacteria in milk may lead to superficial contamination of the muscle when the udder is removed at the slaughterhouse; in some cases of clinical Listeriosis in cattle, *Listeria monocytogenes* septicemia may be accompanied by the contamination of different organs.

In these specific cases, the slaughter process, and therefore the four other “Ms”, are not involved in meat contamination: indeed, these hazards are already present in the muscles or organs of the animal arriving at the slaughterhouse and will, therefore, necessarily be found in the meat at the end of the process.

The other localization corresponds to the digestive tract. Indeed, the main reservoir of many zoonotic agents is the digestive tract of production animals. Meat can then be contaminated in two ways. The first relates to the animal slaughter/dressing process[2]. In the course of this process, fecal contamination can occur, mainly at two specific stages: skinning, which consists of removing the skin from animals (ruminants and equidae) and evisceration, which consists of removing the animal's digestive tract and viscera.

During skinning, contact may occur between the skin, soiled by fecal matter containing microorganisms from the digestive tract, and the subcutaneous tissues, resulting in the transfer of these microorganisms from the skin to subjacent tissues.

During evisceration, the transfer of germs from the DT may also occur from both ends of the digestive tract or through the tearing of the latter. This "fecal" contamination represents the main contamination modality for *Campylobacter jejuni, Salmonella enterica* and enterohemorrhagic *E. coli*, which are the top three causes of zoonoses identified in the European Union. These contaminations, therefore, have animals (raw material) as their source, but they are favored by errors committed during the other four Ms (Method, Machine, Milieu and Manpower).

In order to minimize these transfer risks, strict rules of good hygiene practices are applied throughout the slaughter/dressing process and in particular during these two stages of removal and evisceration. This "fecal" contamination may also be at the origin of cross-contamination that occurs later, at the food preparation stage, by the consumer for example. This modality is described in particular for *Campylobacter jejuni* and poultry meat. Contamination occurs at the slaughterhouse, where *Campylobacter jejuni* can pass from the digestive tract to the skin of poultry.

During the consumer's handling or cutting of raw poultry prior to cooking, material (refrigerator shelf, worktop, knife and chopping board) can become contaminated, as can the hands of the cook. This can then lead to contamination of other food, such as lettuce, if it is placed on the same medium or cut with the same knife, or handled by the cook's hands. It is this cross-contamination that will cause campylobacteriosis in the consumer since the lettuce will be consumed raw, whereas *Campylobacter* left on the poultry will be destroyed by cooking (Figure 1.2). The phenomenon of cross-contamination can occur at all stages of the production chain and also at the consumption stage, through the material used.

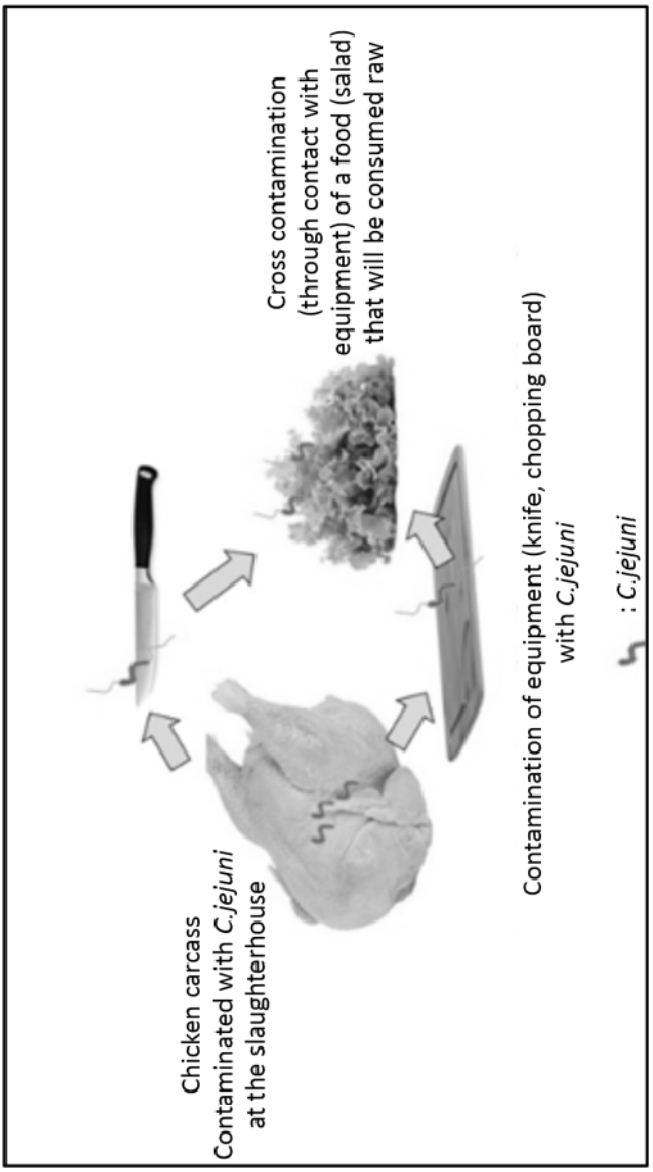

Figure 1.2. *Illustration of the cross-contamination mechanism from a poultry carcass contaminated with Campylobacter jejuni. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

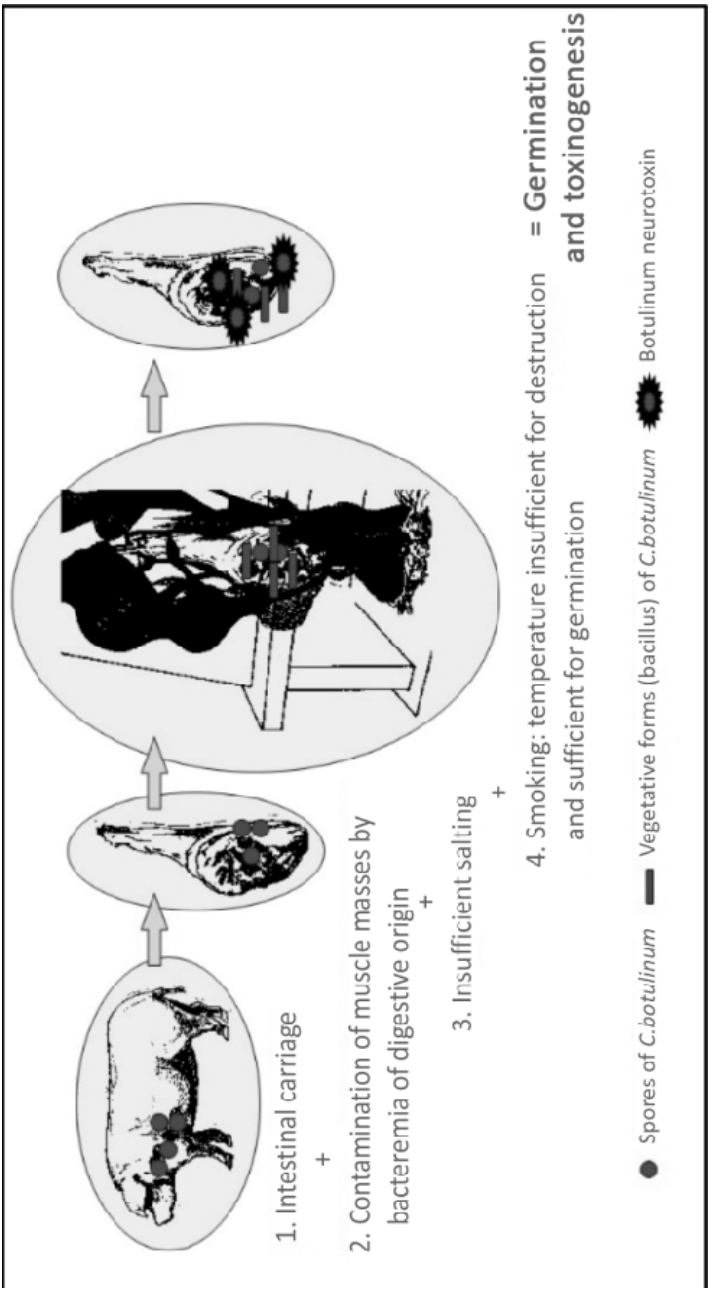

Figure 1.3. *Clostridium botulinum: contamination, germination and toxinogenesis in homemade smoked ham. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

Another possibility for bacteria in the digestive tract is for them to pass into the bloodstream from the digestive tract via the portal vein while the animal is alive. This phenomenon is a "bacteremia of digestive origin". The microorganisms that take this route reach the liver, where they are normally stopped given the role of filter provided by this organ. A very significant "unloading" or a deficient liver can result in the temporary passage of the hazard, unstopped by the liver filter, into the general circulation and therefore throughout the body, including the organs and muscle masses.

If this phenomenon occurs just prior to slaughter, the animal's immune system will not have time to eliminate these agents, which will then be found in the meat or offal. This modality is possible for many bacteria present in the digestive tract and in particular for spores of clostridia, for example, those of *Clostridium botulinum*, which can be subjected to conditions favorable to their growth through this route. These spores can thus reach the center of muscle masses, without the possibility of germination while the animal is alive, since clostridia, which are strict anaerobes, requires a negative oxidation-reduction potential. After the slaughter of the animal, we witness the installation of muscle anaerobiosis, and the redox potential becomes favorable for the germination of these spores, the growth of clostridia and thus toxinogenesis, about 8 h after the death of the animal, where other factors (temperature, water activity, acidity, etc.) are favorable to the survival and growth of mesophilic clostridia. Poisoning by botulinum toxin thus mainly concerns homemade products, for which destructive or inhibitory factors such as temperature, salt concentration, etc., are not monitored (Figure 1.3)

1.1.2. *Contamination by the handler*

In the meat industry and the preparation of dishes, humans can be at the origin of meat contamination. Like other mammals, humans can host foodborne disease agents that can be transmitted to food through various handling operations. Of course, people suffering from enteritis (*Salmonella enterica*, pathogenic *E. coli*, human enteric viruses, etc.) that can shed considerable amounts of bacteria or viruses must be able to be detected and removed from the handling of foodstuffs. But asymptomatic carriage also exists and is much more difficult to detect (e.g. asymptomatic carriage of *Staphylococcus aureus* in the nose, throat, or on the skin, is estimated at 37%) (Hennekinne et al. 2017). Fifteen percent of collective food-poisoning outbreaks reported to the EFSA in collective catering are the result of contamination by an infected handler, hence the importance of following the rules of good hygiene practices in food handling (European Food Safety Authority 2018).

1.1.3. *Environmental contamination*

The working environment, the slaughterhouse, and cutting and processing rooms can be at the origin of the contamination of meat and meat products. In this case, infectious agents are regularly brought onto the premises by-products entering the structure, usually in small quantities, and these microorganisms can survive, or even multiply, in the production environment on various equipment. This can lead to cross-contamination of the products. This capacity to "colonize" the production environment is facilitated by the ability to produce biofilms, three-dimensional structures composed of extracellular polymers.

Biofilms are today considered to be one of the main factors in the persistence of pathogens in the production environment. While this capacity has been described in the majority of bacteria responsible for food poisoning (*Salmonella enterica*, EHEC, *Campylobacter jejuni*, *Yersinia enterolitica*, *Staphylococcus aureus*, etc.), the production of biofilms on surfaces and been particularly studied in *Listeria monocytogenes* (Dubois-Brissonet et al. 2017). Combined with its large capacity to resist hostile conditions, this capacity allows *Listeria monocytogenes* to persist in food processing plants and to be particularly difficult to eradicate from the production environment (Mazaheri et al. 2021). This bacterium can thus adhere to all abiotic surfaces encountered in the food industry (glass, stainless steel, polypropylene, rubber, etc.) after only 20 min of contact. The neutral pH, low temperature and high humidity promote the formation of these structures. In addition, microorganisms that are included in biofilms are particularly resistant to disinfection cleaning processes.

1.2. Growth and survival in meat

The meat consumer needs to have ingested a sufficient number of pathogens in order for a disease to occur. This is referred to as the minimum infectious dose (MID). This dose varies from one agent to another and even from one strain to another, and also depends on host sensitivity, and is not always known. For infectious agents transmitted by meat consumption, the following average doses are established: several hundred bacteria for *Campylobacter jejuni* and Enterohemorrhagic *E. coli* (EHECs), 10^{10}*Listeria monocytogenes* for sensitive populations, 10^5 for *Salmonella enterica*; several dozen viral particles for norovirus,

and one or several cyst(s) for parasites. While the initial contamination may not be sufficient to reach this dose at the outset, DMI will be reached as a result of multiplication inside the food. This only applies to bacteria, as viruses and parasites cannot grow in foodstuffs and will only survive for what may be a short or long period of time within this environment.

The intrinsic physicochemical characteristics of meat (Aw, pH, etc.) enable most bacteria to grow, and as such it is the extrinsic parameters (temperature, atmosphere, etc.) that will be decisive. The multiplication of bacteria will thus occur when meat storage conditions permit: mesophilic bacteria will only grow if the cold chain is broken; a negative oxidation-reduction potential will allow anaerobic growth; psychrotrophic bacterial growth will be favored by the cold chain. Certain bacteria have specific metabolic requirements and will be unable to grow on the food matrix, whatever the temperature encountered (*Campylobacter jejuni*).

The survival of biological hazards in meat will be affected primarily by cooking operations. An increase in the core temperature to above 60°C will most often be lethal for the majority of agents. Spores of the clostridial genera will require higher temperatures in order to be inactivated (sterilization schedules). Certain hazards that may be present in meat will not be affected by an increase in temperature. This is the case for the *Staphylococcus aureus* enterotoxin, which requires several hours of treatment at 100°C in order to be destroyed. This toxin will only be inactivated by appertization heat treatments if it is small in quantity. This is also the case for the BSE prion, which requires temperatures in the order of 800°C to be inactivated. While freezing has no effect on the destruction of bacteria or viruses, this food preservation method is considered effective for the destruction of parasites. This is true for tapeworm larvae and Toxoplasma cysts, however, efficacy is variable for *Trichinella* spp., with some species, such as *Trichinella nativa*, being resistant to negative cold.

The rest of this chapter is devoted to the description of two hazards for which transmission to humans through meat consumption represents the main route: entero-hemorrhagic *E. coli* and *Cysticercus bovis*, the larva of *Taeniasaginata*, the most commonly occurring tapeworm in humans. The other main biological hazards associated with meat consumption will then be described in the form of summary sheets.

1.3. Enterohemorrhagic *E. coli*

1.3.1. *Hazard description and characterization*

1.3.1.1. *Bacteriology*

E. coli belongs to the family of *Enterobacteriaceae*, alongside the well-known genera, *Salmonella* and *Shigella*. The genus *Escherichia* comprises five species, including *Escherichia coli*, which is a normal host of the digestive microflora of humans and warm-blooded animals. It occupies a prominent place since its concentration varies from 10^3 to 10^8 per gram of fecal matter. In the majority of cases, *E. coli* is a non-pathogenic commensal that is used as an indicator of fecal contamination in microbiological analysis of foodstuffs. *E. coli* is a bacillus with gram-negative, facultative aero-anaerobic, oxidase-negative staining, measuring 2–4 µm in length by 0.6 µm in diameter. This species is characterized by a certain heterogeneity because the genome can contain up to 30% additional genes that are different from one isolate to another. These variations are the consequence of the presence of motile genetic elements (plasmids, phages, transposons, etc.) responsible for great plasticity in the genome. Some *E. coli* clones have thus acquired virulence factors responsible for serious health problems.

E. coli strains can be differentiated according to their antigenic properties. Serogrouping is performed according to somatic O-antigens (complex lipopolysaccharides of the outer membrane), flagellar H-antigens (flagellin) and capsular K-antigens (acidic polysaccharides). Thus, at present, more than 700 antigenic types or serotypes are described (Vernozy 2005).

Pathogenic strains of *E. coli* can be grouped into pathovars (or pathotypes) based on the clinical signs observed in patients. Alongside the extra-intestinal pathotypes (ExPEC), responsible for septicemia or urinary tract infections, six pathotypes are capable of invading the human intestine (Farrokh et al. 2012):

1) STECs: Shiga Toxin producing *E. coli* (also called VTEC, for Verotoxin-producing *E. coli*), which includes EHECs;

2) ETECs: enterotoxigenic *E. coli*;

3) EIECs: enteroinvasive *E. coli*;

4) EPECs: enteropathogenic *E. coli;*

5) AEECs: enteroaggregative *E. coli*;

6) DAECs: diffusely adherent *E. coli.*

The growth characteristics of *E. coli* O157:H7, the first identified serotype of EHEC, are shown in Table 1.2. It is a mesophilic bacterium that presents greater survivability under acidic conditions than other *E. coli* strains.

Growth factors	Optimal values	Threshold values
Temperature (°C)	40	6–45.5
pH	6.5	4.4–9
Aw	0.995	0.95
NaCl (%)	0	8.5

Table 1.2. *Growth characteristics of* E. coli *O157:H7 (ANSES 2018)*

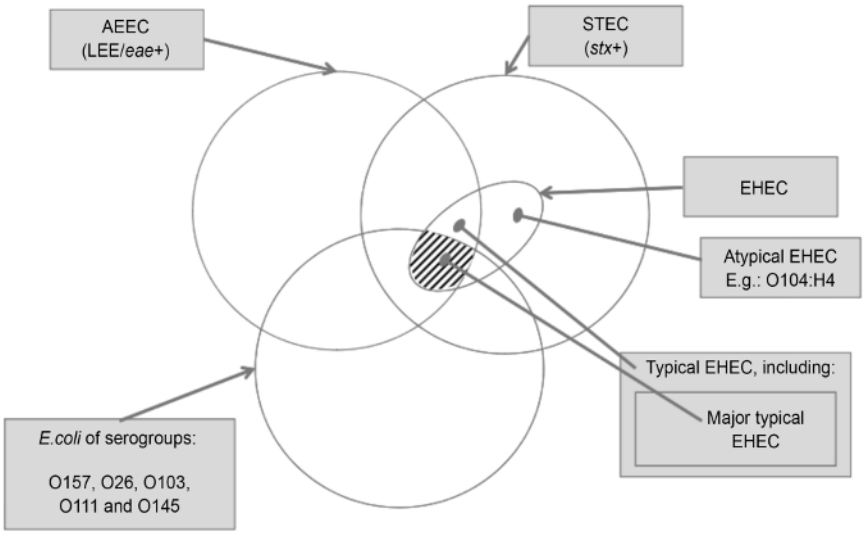

Figure 1.4. *AEEC, STEC and EHEC (according to Brugère et al. 2012). For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

The classification of strains pathogenic to humans remains complex (Figure 1.4). All strains that produce Shiga toxins belong to the STEC pathotype; all strains

possessing LEE are classified as part of the AEEC group. EHECs are a sub-group of STECs; these are the strains pathogenic to humans. All EHEC strains are therefore STECs, but not all STEC strains, even those possessing LEE, systematically cause disease in humans. The strains most frequently involved in epidemics are defined by ANSES as "major typical EHEC" strains. They belong to serotypes O26:H11, O103:H2, O111:H8, O145:H28 and O157:H7 and their non-motile derivatives (H-).

1.3.1.2. *Pathogenesis*

Adhesion to intestinal epithelial cells and colonization of the digestive tract is an essential prerequisite for the expression of the pathogenicity of EHECs. The majority of EHEC strains are capable of causing "attaching-effacing" lesions on the mucosal cells of the distal ileum and colon. This results in intimate adhesion between enterocytes, which is the consequence of the combined action of several proteins encoded by a set of genes grouped together in a pathogenicity island, the LEE (Locus of Enterocyte Effacement). One of these proteins is intimin, encoded by the eae gene. EHEC strains possessing LEE are called typical EHECs (Brugère et al. 2012).

It should be noted that there are atypical EHEC strains that do not possess either the eae gene or the LEE locus. These strains do not induce attaching-effacing lesions but possess other mechanisms for adhesion to the colonic mucosa. For example, the strain at the origin of the 2011 epidemic in Europe is an atypical EHEC of serotype O104:H4, which possesses fimbriae (AAF) and the aggR gene, a regulator of plasmid genes responsible for aggregative adhesion.

EHECs are characterized by the production of toxins, Stx (Shiga toxins, so-called because they have homologies with the *Shigella dysenteriae* type-1 toxin, also called Vtx (verotoxins). These toxins are secreted into the intestine, cross the intestinal epithelium, join the bloodstream and reach specific glycolipid receptors (Gb3: Globotrios ceramide 3) on the surface of endothelial cells. The internalization of these toxins by endocytosis results in the death of target cells by stopping the protein syntheses. This results in lesions to vascular endothelial cells, especially intestinal, renal and brain cells, which are particularly rich in Gb3 receptors in humans. There are two classes of Shiga toxins, Stx1 and Stx2, encoded by the stx1 and stx2 genes. Stx2 toxins are the most potent and show several variants that appear to indicate host specificity (Brugère et al. 2012; Vernozy 2005; Cappelier and Brugère 2013).

1.3.2. *Epidemiological data*

The prevalence in the European Union increased from 0.83 cases per 100,000 inhabitants in 2012 to 2.28 cases per 100,000 inhabitants in 2018. EHECs are now ranked third among zoonotic agents identified in EU member countries, behind *Campylobacter*, and *Salmonella* (European Food Safety Authority 2018). However, more than nine times out of 10, EHEC-related infections are sporadic cases, making their identification difficult. The average annual number of cases of foodborne STEC infection is estimated by *Santé Publique France* (Public Health France) at 17,927.

Epidemic outbreaks are regularly described around the world. The largest global epidemic (more than 9,000 patients, 12 deaths) occurred in Japan in 1996, and the largest European outbreak (around 4,000 cases that included 900 cases of HUS and 50 deaths) occurred in 2011. In France, five epidemics of EHEC infections were detected and investigated between 2011 and 2018: an outbreak of O104:H4 linked to germinated seeds consumed raw in 2011 (the same as the aforementioned European epidemic), three outbreaks of O157:H7/H- related to ground beef in 2011 and 2012, and raw-milk cheese in 2013, and an outbreak of O26 in raw-milk cheese in 2018.

1.3.3. *Human contamination*

Humans are mainly contaminated through the ingestion of food of animal or vegetable origin or drinking water contaminated with fecal matter from excreting animals, but person-to-person transmission and contact with excreting animals are also risk factors. In the United States, the relative importance of these different contamination routes is estimated at 66% for the consumption of contaminated food, 20% for person-to-person transmission, 12% for the ingestion of contaminated water, and 2% for contact with animals.

The main foods implicated across the world (Caprioli 2005) are raw or undercooked ruminant meats, mainly ground beef, raw milk and dairy products and raw or undercooked vegetables.

Fecal contamination of food is at the origin of the presence of EHECs in the food involved, most often when hygiene conditions are not controlled at the time of slaughter/dressing of cattle or during milking. Indeed, domestic ruminants, especially cattle, are recognized as the main reservoirs of STECs. They are healthy carriers and are at the origin of environmental contamination. Vegetable contamination is the consequence of the use of irrigation water contaminated by animal or human waste or manure. STECs can survive for several weeks in the external environment.

1.3.4. *Disease in humans*

EHEC infection causes a variety of symptoms, ranging from simple colitis to serious complications such as hemorrhagic and hemolytic uremic syndrome (HUS) mainly in young children, and thrombotic microangiopathy (TMA) in adults (Table 1.3). The main manifestation of this infection is hemorrhagic colitis. Ingestion of EHEC first causes non-bloody diarrhea after an incubation period of 3–4 days on average. The minimum infectious dose is low, in the order of a few hundred bacteria. This enteritis is accompanied by violent abdominal cramps and resolves spontaneously in around 10% of cases. Nine times out of 10, the symptoms progress to bloody diarrhea, which most frequently results in recovery. But in 10% of cases, the clinical picture is complicated by HUS occurring after 7 days. This HUS is responsible for severe renal damage in one-third of cases and can lead to death in 5% of patients (Figure 1.5). HUS occurs more in children and the elderly and is the leading cause of renal failure in children under 3 years of age.

HUS and TMA are the consequence of Shiga toxins attaching to endothelial cells, resulting in lesions of endothelial cells in the microcirculation, followed by cell swelling, platelet adhesion and thrombosis. The clinical picture depends on the vascular bed most affected: that of the kidneys in HUS and that of the brain in TMA. Both syndromes are characterized by severe microangiopathy, a marked reduction in platelets and hemoglobin levels.

In the case of HUS, glomerular thrombotic microangiopathy lesions are thus observed. The clinical signs are then hemolytic anemia, thrombocytopenia and renal failure. It should be noted that humans may be asymptomatic carriers of EHEC (European Food Safety Authority 2018; Vernozy 2005).

Incubation period	Symptoms	Complications
3–4 days	Ordinary diarrhea or Hemorrhagic colitis	Hemolytic uremic syndrome (HUS) lethality = 1% in children under 15 years of age Thrombotic microangiopathy (TMA) lethality = 25% in the elderly Severe neurological complications in 25% of HUS cases Chronic renal failure in 50% of HUS survivors
2–12 days	with abdominal cramps and initially watery then bloody diarrhea	

Table 1.3. *Characteristics of disease caused by EHECs in humans. (According to ANSES 2018)*

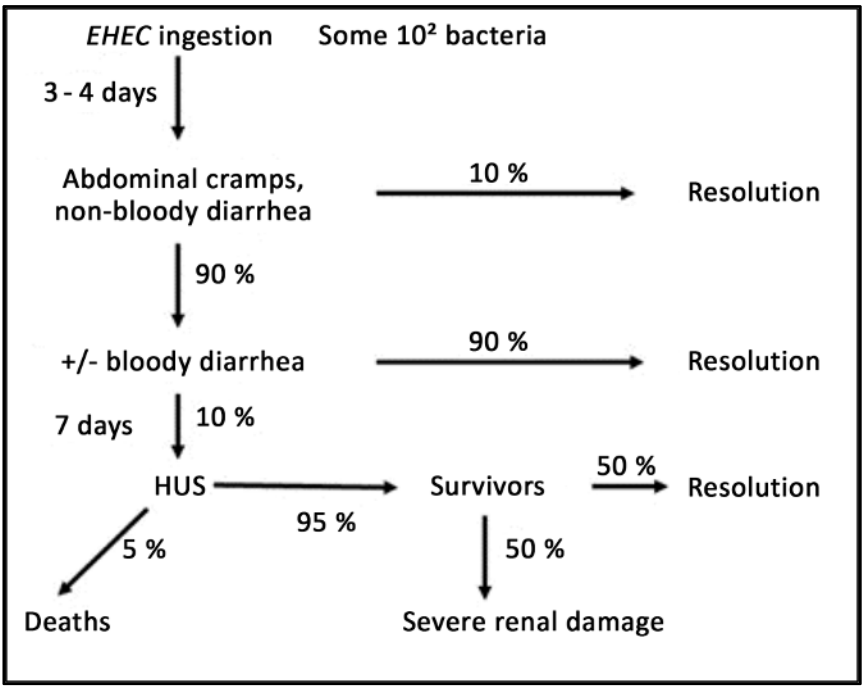

Figure 1.5. *Clinical evolution of a human infection by a strain of entero-hemorrhagic* E. coli

1.3.5. *Disease in animals*

Although some EHECs may cause diarrhea in calves, the absence of receptors specific to Shiga toxins prevents cattle from developing a syndrome similar to that described in humans. Only pork can develop a disease related to Shiga toxins produced by strains not pathogenic to humans (porcine edema disease). EHECs that are pathogenic to humans do not cause disease in production animals, which are therefore asymptomatic carriers (Cappelier and Brugère 2013).

1.3.6. *Means of control*

The presence of EHEC in food is the result of fecal contamination from an animal reservoir. Consequently, control measures must be applied throughout the food chain, using good hygiene practices, including at the consumer level, thus "from the stable to the table" (Cappelier and Brugère 2013).

1.3.6.1. *On the farm*

The measures taken at this stage are very important because they contribute to the reduction of both contaminations of carcasses at the slaughterhouse, contamination of milk during milking and contamination of vegetables or water, and human contamination by direct contact with animals.

The application of good breeding practices helps to limit the excretion of all bovine intestinal pathogens. Vaccination against *E. coli* O157:H7 seems to be able to limit intestinal colonization by this serovar but not against other EHECs. The use of probiotics (*Lactobacillus acidophilus* and *Propionibacterium freundenreichchii*) is patented in the United States and is believed to decrease the survival of the same serovar in the intestine of cattle (Loukiadis 2017). The practice of spreading animal waste should be optimized: crops not intended for human consumption, spreading at particular times of the year, after treatment, etc. It is also important to ensure the cleanliness of cattle integument prior to departing for the slaughterhouse in order to reduce carcass contamination during external dressing. For visits to educational farms, visitors should be reminded of basic hygiene measures such as hand washing.

1.3.6.2. *During transport*

Measures limiting cross-contamination can be put in place: single-deck trucks, avoiding mixing several batches from different sources, washing trucks after each transport, reducing animal stress, etc.

1.3.6.3. *At the slaughterhouse*

The reduction of fecal contamination at the slaughterhouse requires the strict application of good hygiene practices. As soon as cattle arrive at the slaughterhouse, the cleanliness of the fleece must be checked. The animal housing conditions should allow animal cleanliness to be maintained. Measures must be taken concerning animals deemed dirty or very dirty, such as shaving the areas to be cut, slaughtering at the end of the day, or even disposing of excessively dirty animals. This is to avoid contamination of the carcass itself, but also to avoid cross-contamination between dirty and clean animals (REC 854/2004 declares that animals whose "skin or fleece condition is such that there is an unacceptable risk of contamination of the meat during slaughter" are unsuitable for human consumption).

On the slaughter line, good hygiene practices should be observed during all dressing operations, in particular during first skin incisions before skinning, sections, skinning, ligature of the ends of the digestive tract, and evisceration (hygiene of equipment, premises, personnel and of operations). Similarly, the technical level, working conditions, training of personnel, line speed and design of equipment must allow the risk of contamination to be minimized. In the event of accidental contamination of a carcass, corrective measures should be applied: steam disinfection, trimming, and orientation of the carcass toward processed cooked products or downgrading. At the end of the chain, early and rapid refrigeration should prevent bacteria on the carcass from growing. Processes exist for the decontamination of end-of-line carcasses. In the European Union, only the use of steam, or spraying with hot water that is either pure or with added lactic acid is authorized.

1.3.6.4. *During processing*

Good hygiene practices also represent the main means of preventing contamination of food during processing operations. EHECs are not considered to be heat-resistant bacteria. The heat-treatment schedules used for *Salmonella* spp. are also effective for EHECs (D60 = 0.5–3 min and Z = 3.5–7° C) (ANSES 2018), therefore the pasteurization of milk eliminates these bacteria. In addition, these bacteria are sensitive to all disinfectants authorized in the food industry, subject to following the recommendations for use. They have sensitivity close to or lower than other *E. coli* or other pathogenic bacteria such as *Listeria monocytogenes* or *Salmonella enterica*. In contrast, EHECs of serotype O157:H7 appear to

demonstrate greater resistance to acidic conditions. No other resistance capacity is reported.

1.3.6.5. *At the consumer stage*

Hygiene remains the basis of prevention methods at the consumption stage, such as washing hands after using the toilet, or prior to preparing and eating meals. One of the most frequently reported errors is feeding a child or an elderly person a raw or undercooked beef burger. Thoroughly cooking beef burgers is recommended, especially for the most sensitive individuals: a minimum temperature of above 70°C throughout the meat should be reached (the inside of the burger becomes gray-brown in color, with no pinkish traces). Compliance with the cold chain from purchase to consumption of the food is also important. Frozen beef burgers must be thawed either during the cooking process, or in the microwave then immediately cooked, and under no circumstances at room temperature. Fresh beef burgers from the butcher should be cooked on the day of purchase. Raw milk, raw-milk cheeses and raw ground meat should be excluded from the diet of young children and the elderly. Vegetables, fruit and aromatic herbs should be thoroughly washed in water mixed with vinegar and peeled before being consumed raw (Bailly et al. 2012; Vernozy 2005). As with other foodborne disease agents, some common food preparation practices by consumers can lead to cross-contamination: using the same knife or chopping board to cut raw meat and then meat after cooking, or other foods such as lettuce that will be consumed raw.

1.3.6.6. *Detection in food*

In accordance with Regulation EC 2073/2005, amended by REC 1441/2007, the search for *E. coli* is a process hygiene criterion, which makes it possible to highlight fecal contamination of foods. European legislation has not introduced any specific safety criteria for EHECs. However, according to the general principles of the hygiene package, in the event that hazard analysis highlights these bacteria, the search for them must be carried out by professionals. *E. coli* O157:H7 is searched for according to the reference method (NF EN ISO 16654) or using a number of validated alternative methods. In France, the Directorate-General for Food (DGAL, *Direction Générale de l'alimentation*) regularly conducts searches for EHECs as part of its monitoring and surveillance plans, particularly for meats intended for grinding, ground meats and raw-milk cheeses.

1.4. *Taenia saginata*/*Cysticercus bovis*

1.4.1. *Hazard description and characterization*

Taenia saginata is a cosmopolitan tapeworm or cestode (class: cestoidea – family: *Taeniidae*) whose evolutionary cycle is described as a heteroxene cycle with 2 hosts: a definitive host and an intermediate host. The parasite's adult stage (*Taenia saginata*) lives in the small intestine of the definitive host, humans, in the form of a large worm (4–12 m long by 5–7 mm wide), a long, segmented ribbon formed of about 2,000 rings (proglottids) responsible for human teniasis (tapeworm). The larval stage (*Cysticercus bovis*) lives in the striated muscles of the intermediate host, the bovine, in the form of an ovoid vesicular larva (6–8 mm by 3–5 mm) responsible for beef measles (Euzeby 1998; Fosse 2003; ANSES 2012; Dorny 2009).

The parasite's cycle is described in Figure 1.6. Cattle, the intermediate host, become contaminated by ingesting embryophores present in grass, water or soil. These embryophores come from the proglottids or ovigerous segments, emitted into the external environment by the definitive host. These proglottids are lysed in the external environment releasing the embryonated eggs at the origin of the infestation of cattle. Mature proglottids are rectangular (16–20 mm by 6–7 mm) and each contains 30,000–90,000 embryophores, or embryonated eggs (60 µm by 40 µm). Eggs ingested by cattle hatch in the digestive tract and release hexacanth embryos, or oncospheres, that pass through the intestinal mucosa and migrate via general circulation to the striated muscles 8–10 weeks after ingestion of the embryophores. They then transform into cysticerci and become infective to humans after a maturation period of about 10 weeks.

Humans become infected when they ingest muscle tissue, that is, beef, consumed raw or not sufficiently cooked to destroy larvae. After ingestion by humans, the scolex is released and the tapeworm then develops in the intestine, beginning to produce proglottids in about 3 months. Humans can then release up to eight segments a day, or about 500,000 embryonated eggs per day (Euzeby 1998; Villena 2017; Morlot 2011; Murell 2013; ANSES 2012).

Humans are the only source of cattle contamination. The latter is contaminated through the ingestion of embryophores, most commonly found in plants (phytophagy) or in water (hydropinia). To be noted is the specific case of calves that may be contaminated by ingestion of artificial milk reconstituted from powder in water and stirred by the soiled hands of a tapeworm-contaminated operator (proglottids under the nails). Proglottids are unknowingly dispersed in nature by human fecal matter, emitted in wastewater (overflow of treatment plants), and through dissemination by different animals such as earthworms and diptera.

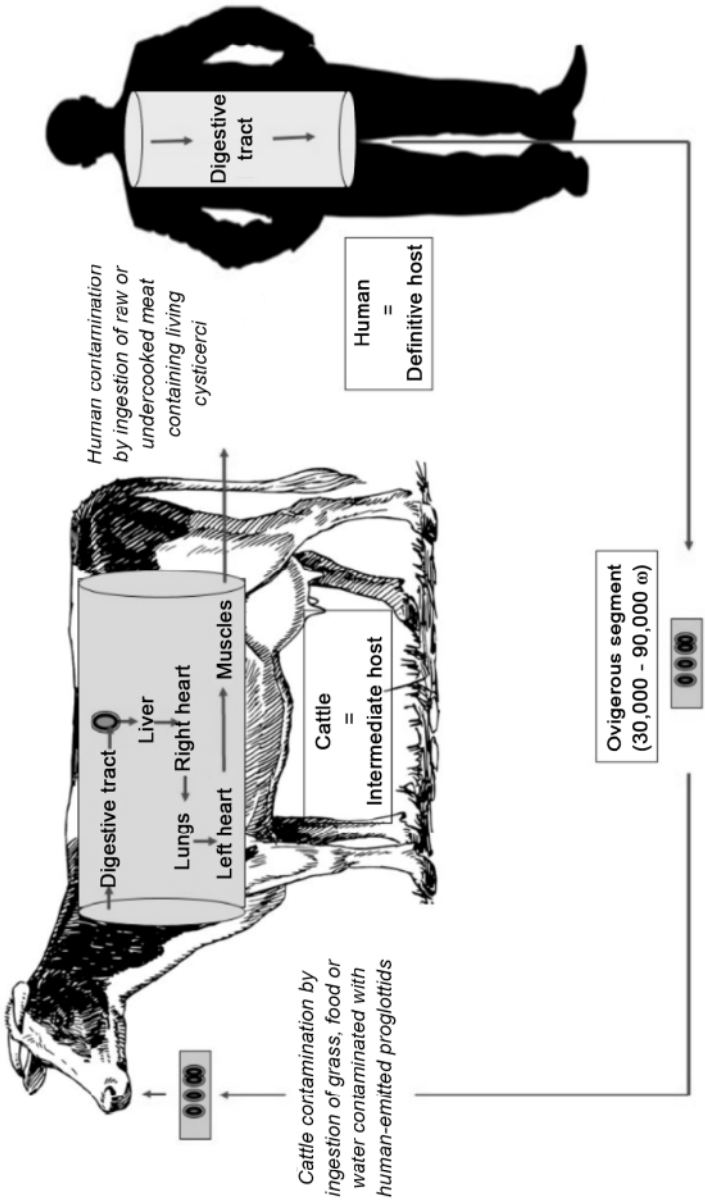

Figure 1.6. *Evolutionary cycle of Cysticercus bovis/Taenia saginata. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

The survival of forms infective to cattle and humans is an essential element in the transmission of this parasite. The longevity of the adult worm in the human intestine can reach 25 years (4–10 years on average). Embryophores possess considerable resistance properties in the external environment: 6 months–1 year in a humid environment; 3 weeks in dry fodder; 70 days in manure; 15 days–1 month in sewage, and up to 3 months in sludge from sewage plants. Survival of embryonated eggs is favored by low-temperature, humid conditions. In bovine muscle tissue, cysticerci remain viable and therefore infective for about 1 year (Morlot 2011).

1.4.2. *Epidemiological data*

Although *Taenia saginata* is considered the most common zoonotic tapeworm in the world, this parasitic disease is not mandatorily reported. Indeed, this parasite is widespread around the world, present in both industrialized and developing countries. The number of human *Taenia saginata* teniasis cases worldwide has been estimated at 45,000,000. According to an estimate by the French Institute for Public Health Surveillance (INVS, *Institut Nationale de Veille Sanitaire*), the number of cases of human teniasis in France in the 2000s stood at about 65,000 cases per year, that is, a prevalence of 0.1% (INVS 2003; Villena 2017).

Data for prevalence in cattle are derived from slaughterhouse data entries, but the detection technique is not very sensitive in the case of discrete infestations that are the most common form in developed countries: prevalence worldwide is currently estimated at 0.007–6.8%, depending on the country. In France, the true prevalence is estimated at 0.88% (Morlot 2011; Alban 2020).

1.4.3. *Human contamination*

The only route of human contamination is the consumption of raw beef (beef tartare) or undercooked beef (rare meat). In theory, one viable larva is sufficient to infest humans (Euzeby 1998; ANSES 2012; Murell 2013; Villena 2017; Alban 2020).

1.4.4. *Disease in humans*

Human Teniasis in *Taenia saginata* is a benign disease that often goes unnoticed due to the absence or lack of specific symptoms. Rarely described signs include

abdominal pain, nausea, weight loss, anorexia or bulimia, anal itching and epigastric pain. Sometimes only the discovery of rings (proglottids) in bedsheets or underwear gives reason to suspect this parasitosis. In humans, treatment is with anthelminthic molecules given orally (Praziquantel 10 mg/kg–Niclosamide 2 g/adult) (Euzeby 1998).

The lack of severity associated with infestation by *Taenia saginata* in humans contrasts with the severe clinical picture observed in the case of infestation by another tapeworm, *Taenia solium*, whose source of transmission is pork. The *Taenia solium* cycle is established between the pig, the intermediate host, and the human, the definitive host. Firstly, the clinical manifestations of *Taenia solium* teniasis are often more severe than for *Taenia saginata* teniasis due to the traumatic action of the scolex hooks on the intestine (armed tapeworm). Secondly, humans can be susceptible to larvae and be contaminated by cysticerci of their own tapeworm, thus becoming an intermediate host. In this case, larval localizations that concern the brain are responsible for neurocysticercosis characterized by severe clinical manifestations such as convulsions and epileptic seizures.

In France and Europe, the disease is no longer reported partly because of the structuring of pig farms and pork being consumed well-cooked. In contrast, it remains endemic in Central and South America, Sub-Saharan Africa and Asia (Euzeby 1998; Fosse 2011; Villena 2017; Porphyre 2019).

1.4.5. *Disease in animals*

The common name for the disease is beef measles. This corresponds to muscular cysticercosis due to the accumulation of larvae in the interfascicular connective tissue of striated muscle masses in the form of small, thin-walled, translucent, vesicular cystic lesions containing a clear, pinkish fluid. The symptoms associated with bovine cysticercosis are particularly discrete, even absent, and non-specific (Alban 2020). Although rare cases of heart disease or myocardial degeneration are described, these symptoms, when they exist, are too mild to be perceived and are never related back to their true origin.

Vesicles are theoretically present in all muscles, but with preferential localization, known as muscles of choice, which correspond to the most irrigated

muscles, as they are the most active in cattle: the myocardium, the tongue muscles, the muscles of mastication (masseters and pterygoids), the esophagus, the muscles of the diaphragm, etc.

The degree of infestation depends on the cattle contamination mode. With so-called pasture cysticercosis, the infestation is low, with a small number of cysticerci appearing only sporadically: it often corresponds to indirect contamination by wastewater, runoff, etc. In so-called barn cysticercosis, a significant and continuous source is present in the cattle's immediate environment, often an individual carrying a tapeworm within the farm. Infestation is massive and affects many animals.

Larvae installed in muscle tissue have limited longevity. Although cysticerci are considered to remain viable for about 1 year, the degeneration can begin as early as 6 months, and significant variations have been observed depending on the degree of infestation and the animal's age. A heavy infestation thus reduces cysticercus longevity. Neonatal infestation extends cysticercus longevity. Localization of the cysticerci also occurs, with degeneration starting more rapidly in the heart muscle. During degeneration, the appearance of the cysticerci changes: the fibrous capsule thickens and turns opaque, the transparent liquid is gradually replaced with a greenish material, then the cyst becomes calcified (Euzeby 1998; Morlot 2011; Jeuffé 2017).

1.4.6. *Means of control*

The means of combatting human teniasis aim to interrupt the parasite cycle. It is thus possible to intervene at different points in the cycle with measures that will aim to: detect cysticerci at the slaughterhouse; destroy the parasite present in beef; and prevent contamination of farmed cattle. Moreover, if medical treatment is indicated for humans (see chapter "Disease in Humans") to destroy the adult stage, the use of anthelmintic drugs in cattle is rarely practiced (Morlot 2011). Indeed, the disease is most often asymptomatic, so it is not possible to know whether the bovine is infested, and furthermore, while the efficacy of the molecules used (praziquantel, albendazole, oxfendazole) seems good, it depends on the timing of treatment application and the treatment cost makes it economically unfeasible.

1.4.6.1. *Preventing contamination of farmed cattle*

Cattle contamination may be limited by preventive measures put in place in farming. These are simple hygiene measures that include the presence of employee toilets on the farm, and facilities for hand washing, especially prior to tasks such as feeding calves. All employees working on the farm must also receive precise information on the disease and the methods of transmission of the parasite. In case of doubt, it seems logical to request screening for personnel working in direct or indirect contact with cattle. Wild camping must be prohibited within a close perimeter of farms. Wastewater treatment plants should be adequately sized so that their capacity is not overwhelmed by climatic phenomena or by an influx of tourists during holiday periods.

When a case of cysticercosis is discovered in a bovine at the slaughterhouse, an epidemiological investigation should be conducted to determine the origin of the infestation. All persons connected with the farm of origin should be interviewed, tested and treated as necessary. When the infestation discovered at the slaughterhouse is heavy, the contamination very often originates from a person working on the farm, and this investigation often helps to identify the origin. In contrast, where the infestation is discrete, contamination has most often occurred in pasture and it is difficult to determine its origin. For example, the few embryonated eggs that contaminated cattle may have been brought in by runoff from a neighboring contaminated pasture. The spreading of sewage sludge on agricultural land, therefore, needs to be carried out in strict compliance with the regulations. For example, following the spreading of unsanitized sludge on areas of grasslands or fodder crops, it is mandatory for a period of 6 weeks to be observed before turning animals back out to grass or harvesting fodder crops. In the case of sanitized sludge, this time period is 3 weeks. Furthermore, the health standards at the end of the treatment chain must be respected (Salmonella: <8 MPN per 10 g of dry matter, Enterovirus: <3 MPN per 10 g of dry matter, viable eggs of helminth pathogens: <3 per 10 g of dry matter (Morlot 2011)).

1.4.6.2. *Detection at the slaughterhouse*

Veterinary inspection of slaughter animals is conducted at the slaughterhouse by agents of the official monitoring services, official veterinarians and official

veterinary assistants, otherwise known as the Veterinary Service. The regulatory inspection method is detailed in one of the texts of the hygiene package, 854/2004/EC of the European Parliament and of the Council of April 29, 2004. This text details the inspection technique for meat-producing animals, specifying the gestures and incisions to be applied, on the organs and carcass of the different species concerned, for the purpose of detecting lesions or anomalies that would render meat harmful to the health of the consumer or unfit for human consumption.

The regulatory steps in this inspection that relate more specifically to the search for bovine cysticercosis are:

– visual inspection of the carcass, section surfaces and organs;

– visual inspection and palpation of the tongue (or only the free part of the tongue, exceptionally in the case of calves);

– visual examination of the pericardium and the heart. The heart is incised once along its length at the left ventricle and the interventricular septum. This exposes the internal surfaces and the cut surfaces, with eversion possible in order to better see the structures. The incisions go from the base to the apex;

– inspection of internal, external and pterygoid masseters by one or two incisions. The cuts are parallel to the bone and perpendicular to the muscle;

– examination of the diaphragm after resection of the peritoneum, with or without incision;

– examination of the esophagus.

If one or more cysts are found in the muscles defined above, additional incisions may be made while respecting the butcher cuts so as to avoid any downgrading of the meat's value.

The objective is to highlight the cysticerci in these different muscle localization (Figures 1.7–1.9). While theoretically, cysts can be viewed or perceptible to touch in tissues such as the tongue in heavily infested animals, as early as the second week after the infestation, cysts become truly visible from 6 weeks and reach full development after 16 weeks.

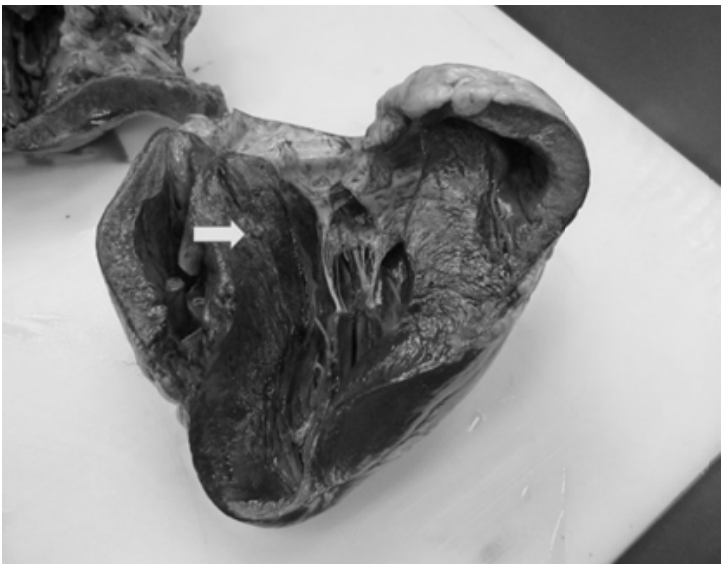

Figure 1.7. *Live cysticercus discovered in the myocardium of a bovine during the regulatory incision (Photo Pr. J.M. Cappelier). For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

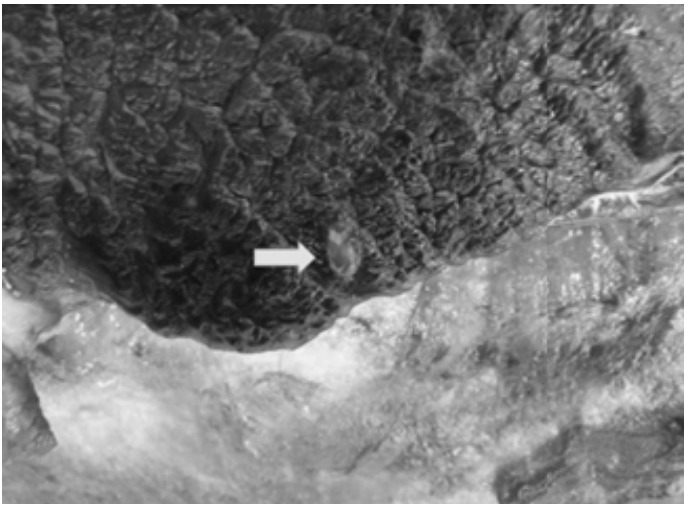

Figure 1.8. *Live cysticercus discovered in a bovine masseter muscle – dimensions: 6 mm by 4 mm (Photo Pr. J.M. Cappelier). For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

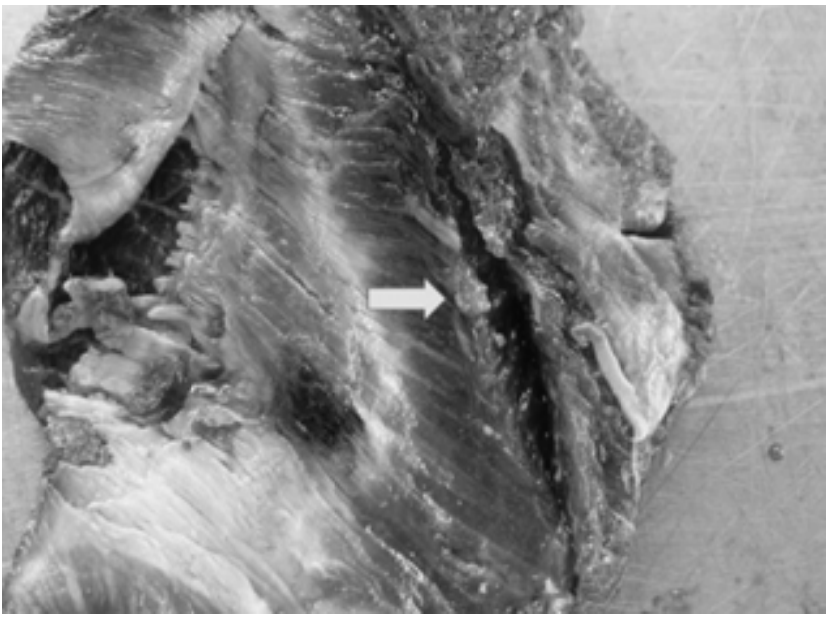

Figure 1.9. *Calcified cysticercus discovered in a bovine masseter muscle (Photo Pr. J.M. Cappelier) . For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

The course to be followed further to the discovery at the slaughterhouse of a carcass presenting lesions of cysticercosis is specified in Regulation 854/2004/EC of the European Parliament and of the Council of 29 April 2004, Annex 1 Section IV Chapter IX point B2:

> Meat infected with cysticercus is to be declared unfit for human consumption. However, when the animal is not generally infected with cysticercus, the parts not infected may be declared fit for human consumption after having undergone a cold treatment.

The degree of infestation of the carcass, therefore, needs to be determined. Contamination is considered generalized when more than one larva per dm^2 is

observed, which corresponds to several cysticerci visible in conventional inspection areas. According to the recommendations of the OIE, this concerns the discovery of cysts in two territories of choice, including the masseter muscles, the tongue, the esophagus, the heart, the diaphragm or the superficial musculature, or in more than two sites on the limbs. In this case, in accordance with the regulations, the carcass is withdrawn from human consumption in its entirety.

When the cysticerci discovered amount to less than one lesion per square decimeter, the organs or carcass parts carrying lesions are seized, and the remainder of the carcass, including the head, heart and esophagus, is sanitized under negative cold conditions (see "Parasite destruction" below).

Screening for bovine cysticercosis at the slaughterhouse demonstrates a sensitivity problem. Indeed, while the inspection method described above is reliable in the case of generalized cysticercosis, its sensitivity in detecting lightly-infested carcasses is in the order of 17%. Yet today, the majority of cattle carcasses at the slaughterhouse present a low level of contamination.

Multiplying the number of heart muscle incisions increases the probability of detection of discrete infestations, but leads to impairment of the cardiac muscle. Combining serological testing and targeted inspection at the slaughterhouse would increase the probability of detecting low-infested carcasses and thus reduce the prevalence of *Taenia saginata* tapeworm in humans (Morlot 2011; Murell 2013; Alban 2020).

In accordance with European regulations, a breeder who sends cattle to the slaughterhouse should inform the monitoring services via a veterinary health certificate (ASDA: *Attestation Sanitaire à Délivrance Anticipée*) of the presence of particular hazards that may affect human health (Food Chain Information, FCI).

Cysticercosis is among the elements that are to be reported. Thus, when cysticercosis is detected in a bovine, the animal's owner is required to report this event on the FCI for all cattle sent to the slaughterhouse from their farm, by

checking the box: "*provient d'un lot ayant fait l'objet d'au moins une information sur la présence de cysticercose*" (comes from a consignment that has been subject to at least one report of the presence of cysticercosis). This applies for up to 9 months following the discovery of cysticercosis. This information allows the veterinary services to carry out a thorough inspection of these animals. Nonetheless, this reporting requirement ends once no cysticercosis lesions have been discovered on two successive consignments of cattle sent to the slaughterhouse.

1.4.6.3. *Parasite destruction*

Effective treatments for the destruction of *Cysticercus bovis* are:

– Negative cold: freezing at least -10°C for 10–14 days or at least -7°C for 21 days. Cysticerci are known to resist for 40 days in meat at 4°C and 4 h at -10 °C. Shorter, more aggressive freezing can be carried out, provided that the following parameters are applied in order to destroy the parasite: a 30 kg cut of meat requires two 24-h cycles at -30.9°C followed by storage at -23.3°C for 72 h.

– Heat: consumers of raw or undercooked meat (blue or rare) expose themselves to the risk of teniasis. Cooking meat thoroughly enables the larvae to be destroyed. It should reach a core temperature of at least 56°C for several seconds.

– Other treatments:

- Salting is effective if the meat is covered with salt (2.5 kg pieces, Water activity <0.86 or for 3 weeks) or in brine (at least 20% NaCl for 5 days).

- Pressure treatment is effective from 0.49 kg/cm^2.

- Treatment by 0.5–0.4 kGy ionizing radiation inactivates 100% of cysticerci but this process is not authorized in the EU.

- Both refrigeration and smoking are entirely ineffective (Villena 2017; Euzeby 1998; Morlot 2011).

Freezing treatment of the carcass is associated with a 30% loss in the carcass value. Total losses in France related to bovine cysticercosis are estimated at more than 430,000 euros per year (Jeuffé 2017).

1.5. Other major hazards related to meat consumption

1.5.1. *Thermotolerant campylobacters (C. jejuni and C. coli)*

(Fosse 2003; ANSES 2011a; Rodrigues et al. 2015; European Food Safety Authority 2017; Messaoudi Federighi 2017; Regulation EC 1495/2017; European Food Safety Authority 2018)

Thermotolerant campylobacters are the leading cause of bacterial enteritis in the world. They are also considered to be the leading cause of zoonosis in the EU, with an incidence, far higher than *Salmonella enterica*, of 64.1 per 100,000 inhabitants in 2018. Most cases of foodborne campylobacteriosis are caused by *Campylobacter jejuni* (85% of cases), followed by *Campylobacter coli* (10% of cases) and other more anecdotal species (*C. lari* and *C. upsaliensis*). The characteristics of *C. jejuni* and *C. coli* are presented in Table 1.4.

Human cases most often occur in a sporadic form and are therefore difficult to identify. The significance of *Campylobacter* in foodborne outbreaks is less as *Campylobacter* ranks third in the cases identified in the EU in 2018, far behind Salmonella and bacterial toxins.

Asymptomatic digestive carriage of *Campylobacter* in production animals is very common, particularly for poultry but also pigs and cattle, causing contamination of meat and offal.

Of the 439 outbreaks of Campylobacter food poisoning identified by the EFSA between 2010 and 2017, 127 (29%) were linked to the consumption of poultry meat or poultry meat products, 18 (4%) to the consumption of meat and meat products from other species (pork, beef and other red meats).

Researchers examining the hazard *Campylobacter jejuni* often evoke a paradox: this bacterium is the leading cause of bacterial enteritis worldwide and thus travels from the digestive tract of production animals to consumers throughout the food chain. Yet it is known to be fragile as it is sensitive to atmospheric oxygen, and is incapable, given specific metabolic requirements, of growing on the food matrix. Also notable, despite its prevalence and substantial research, the pathogenic mechanism remains to be fully elucidated today.

Description of the agent	Morphology: Gram-negative bacillus, highly motile, characteristic appearance, thin, comma- and S-shaped capable of evolving into a spherical shape and micro-aerobic (5–10% O_2) Growth: Tp. opt: 41.5°C – Tp. L.: < 30°C; > 45°C pH opt: 6.5–7.5 pH L. < 4.9 > 9 - O_2 opt: 3–5% O_2 L: 0% or > 18% - CO_2 opt: 10% Aw opt: 0.997 – Aw L < 0.987 – NaCl opt: 0.5% - NaCl L > 2%
Reservoir	Animal reservoir: Digestive tract of birds, domestic poultry, pigs, cattle, sheep, and domestic animals (cats and dogs) Soil and water reservoir contaminated by animal waste[2]
Pathogenicity and clinical signs	MID: Several hundred bacteria Colonization of the digestive tract, adhesion to and invasion of intestinal epithelial cells Secretion of cytotoxins (CDT) Incubation: 2–5 days Febrile diarrhea, sometimes with blood in the stool, long and severe abdominal pain Septicemia (under 1% of cases) with several post-infectious syndromes (arthritis, hepatitis, nephritis) and particularly Guillain-Barré syndrome (0.1% of cases): Affecting the peripheral nervous system with flaccid paralysis (lethality 2%, neurological sequelae 20%)
Foods involved	Consumption of contaminated raw or undercooked foods Meats: Poultry meats, red meat and offal, which can be responsible for cross-contamination in the kitchen Other: Raw milk, untreated water or water recontaminated after treatment
Control	Regulatory microbiological process hygiene criterion (REC 1495/2017): Not satisfactory when 20/50 samples > 1,000 CFU/g. Cooking for several seconds at 65°C at the core Domestic hygiene: Wash hands and utensils after handling raw meat

Table 1.4. *Campylobacter jejuni/coli: characteristics*[1]

1 Key for Tables 1.4–1.11:
– Tp opt: optimal temperature - Tp L: limit temperature -;
– pH opt.: optimal pH - pH L: limit pH;
– O2 opt: optimal O2 concentration – O2 L: limit O2 concentration;
– CO2 opt: optimal CO2 concentration;
– Aw opt: optimal water activity - Aw L: limit water activity;
– NaCl inhib.: inhibitory NaCl concentration.

1.5.2. *Salmonella enterica*

(Fosse 2003; Regulation EC 2073/2005; ANSES 2011d; Boumart et al. 2017; European Food Safety Authority 2017; European Food Safety Authority 2018)

Many *Salmonella enterica* serotypes are the origin of diseases in humans and animals. We will not discuss here the serotypes that are qualified as typhoids (*Salmonella Typhi* and *Paratyphi*) responsible for typhoid fevers, but those serotypes are said to be non-typhoid. These ubiquitous serotypes are responsible for asymptomatic digestive carriage in animals and correspond to the vast majority of Salmonella gastroenteritis in humans. While *Salmonella Enteritidis* and *Salmonella Typhimurium* are the two predominant serotypes in foodborne diseases, all serotypes are considered to be pathogenic.

Salmonella was long considered both the dominant cause of foodborne outbreaks (more than 50%) and the leading cause of zoonosis in the European Union until 2005. This predominance led the European Union to introduce numerous control measures in the 1990s, which had the effect of reducing the number of cases and the proportion of salmonellosis in foodborne outbreaks cases. However, today they remain one of the main causes of foodborne gastroenteritis in industrialized countries: the leading cause of foodborne outbreaks in the EU with 24.4% of outbreaks, the third cause in France behind the *Staphylococcus aureus* and *Bacillus cereus* toxins then viruses. Salmonella occupies second place in the ranking of zoonoses identified in the EU, with nearly 92,000 reported cases and an incidence of 11.1 cases per 100,000 inhabitants.

The majority of salmonellosis cases relate to the consumption of eggs and egg products (46% of outbreaks in 2018), ahead of meat: 5.4% of outbreaks related to the consumption of pork; 4.4% to the consumption of meat mixtures; 2.4% to the consumption of poultry meat; 1% to the consumption of beef and veal; and 1% to the consumption of ovine meat. The characteristics of *S. enterica* are presented in Table 1.5.

1.5.3. *Yersinia enterolitica*

(Fosse 2003; ANSES 2017b; European Food Safety Authority 2017; Feurer and Guillet 2017; European Food Safety Authority 2018)

The genus *Yersinia* comprises three pathogenic species, *Yersinia pestis*, the agent of plague, *Yersinia pseudotuberculosis* and *Yersinia enterolitica*, responsible for yersiniosis. The characteristics of *Yersinia enterolitica* are presented in Table 1.6.

Description of the agent	Morphology: Small (2–3 mm), gram-negative bacillus, non-spore-forming, facultatively anaerobic and motile by means of peritrichous flagella Growth: Tp. opt: 37°C – Tp. L.: < 5°C; > 50°C pH opt: 7; pH L < 3.8 > 9.5 - Aw opt: 0.99 – Aw L < 0.94
Reservoir	Ubiquitous bacteria, main reservoir = digestive tract of animals (poultry, sheep and cattle) and humans The animal reservoir can be at the origin of environmental contamination = secondary reservoir
Pathogenicity and clinical signs	MID: 10^1–10^5 CFU, depending on the food, the serotype and consumer sensitivity Incubation: 12–36 h Adhesion, invasion and intracellular multiplication Numerous virulence factors (pili, adhesins, flagella, LPS, type-III secretion system, etc.) Abdominal pain, diarrhea, nausea, vomiting, 39–40°C fever Duration of symptoms: 5 days, but excretion for several weeks Bacteremia in 3–10% of cases Risk of extradigestive infections (joints) Hospitalization (dehydration): 22% Lethality: 0.8%
Foods involved	Mainly eggs and egg products (almost 50%) Meats of all species: 15%
Control	Regulatory microbiological safety criterion (REC 2073/2005): Absence in 25 or 10 g Compliance with good hygiene practices throughout the food chain Cooking for several minutes at 60°C

Table 1.5. *Salmonella enterica: characteristics*

Yersiniosis is a foodborne zoonosis that results in febrile gastroenteritis. The bacteria responsible are psychrotrophic and therefore favored by the cold chain. Yersiniosis is now considered the fourth zoonotic disease identified in the European

Union, with 6,699 cases reported in 2018 and a prevalence of 0.8 cases per 100,000 inhabitants. Pigs are considered asymptomatic carriers of *Yersinia enterolitica*.

Fecal excretion or use of parts of the head (tongue, tonsils and head lymph nodes) is at the origin of the contamination of pork and pork products.

Description of the agent	Morphology: Small, gram-negative non-spore-forming, non-encapsulated, facultative aero-anaerobic bacillus Six biotypes Growth conditions: Psychrotrophic Opt temp.: 29°C – L. Temp.: < -2°C; > 42°C - Opt. pH 7.3; L. pH < 4 > 10 Aw opt: 0.995 – Aw L < 0.945 - NaCl L > 5–7%
Reservoir	Pork is the main reservoir, an asymptomatic carrier of pathogenic strains (carriage on the tongue, tonsils, lymph nodes, intermittent fecal excretion) Prevalence of pork tonsils in France at the slaughterhouse: 14% Prevalence in pig fecal matter in France at the slaughterhouse: 9–18%
Pathogenicity and clinical signs	MID: Between 10^4 and 10^6 bacteria Adhesion to epithelial cells (adhesin, invasins), enterotoxin, translocation by M cells, propagation in lymph nodes Especially sporadic forms Incubation: 7 days Enterocolitis: Diarrhea, abdominal pain, fever. Terminal ileitis that can mimic an attack of appendicitis Duration of symptoms: 2–3 days to several weeks Excretion several months without treatment Severe forms with deep abscess or septicemia in predisposed patients (iron overload, cirrhosis, diabetes, immunosuppression)
Foods involved	Pork and pork products: Contamination of the carcass by fecal excretion – incorporation of pieces of head into chopped products Other sources: Plants, raw vegetables, water
Control	Good practices of evisceration, handling of the tongue and tonsils. Cooking: Several seconds at 65°C.

Table 1.6. *Yersinia enterolitica: characteristics*

1.5.4. *Listeria monocytogenes*

(Fosse 2003; Regulation EC 2073/2005; ANSES 2011b; Piveteau 2017; European Food Safety Authority 2017; European Food Safety Authority 2018)

Description of the agent	Morphology: Small (0.5–2 mm by 0.5 mm), gram-positive bacillus, motile, isolated or in chains, facultative aero-anaerobic. Five genosero groups. IVb, IIa then IIb are the most involved in human cases. Growth: Tp opt: 30–37°C; Tp L: < -2 - > 45 (psychrotrophic) pH opt: 7 pH L: < 4 – > 9.6 - Aw opt: 0.99 - Aw L: < 0.92 High persistence capacity in food workshops (Biofilms)
Reservoir	Ubiquitous, telluric bacterium, widespread and highly resistant in the environment (soil, water and feces) – decomposing plants (ensilage not sufficiently acidic) Digestive carriage in 6–30% of cattle, sheep, swine, goats and chickens: The main source of human contamination.
Pathogenicity and clinical signs	MID: Not well known, estimated at 10^{10} in immunocompromised individuals Incubation: 48 h to more than a month Internalization, intracellular multiplication, blood dissemination, tropism for placenta and brain Concept of population at risk (young, old, pregnant and immunodeficient) Non-invasive, rare forms: Febrile gastroenteritis Invasive forms: Severe forms affecting the YOPI In pregnant women: Flu-like syndrome, abortion, death in utero, premature delivery, neonatal infection. In other immunocompromised subjects: Septicemia, meningitis, meningoencephalitis, encephalitis and cerebral abscesses. Mortality: 20–30% for populations at risk, common neurological sequelae.
Foods involved	Ready-to-eat food Meats: Rillettes, jellied pork tongue and meat preparations Other: Raw-milk cheeses and seafood
Control	Regulatory microbiological safety criterion (R EC 2073/2005): Absence in 25 g or < 100 CFU/g at the UBD Heat treatment: 0.2–2 min at 70°C. Strict compliance with good hygiene practices at all stages of production Absolute compliance with the cold chain and manufacturer's recommendations (UBD) Observance of household hygiene

Table 1.7. *Listeria monocytogenes: characteristics*

Listeriosis is a serious foodborne disease (>50% hospitalization, lethality 20%) but has a low incidence (0.47 cases per 100,000 people). It is a reportable disease, presenting a regulatory microbiological safety criterion that is dependent on the characteristics of the food, taking into account the potential for Listeria growth. This criterion ranges from "absence in 25 g" to <100 CFU/g.

Listeria monocytogenes have a very diverse ecological niche. It is a ubiquitous, telluric bacterium, widespread throughout the environment, presenting considerable resistance, capable of developing at refrigeration temperatures and contaminating food processing workshops (biofilms). Contamination of foodstuffs can therefore occur at any moment in the food chain. The initial contamination of foodstuffs is generally low, the products at the origin of cases are products in which multiplication can occur (long UBD, storage error). The characteristics of *L. monocytogenes* are presented in Table 1.7.

Of the 32 outbreaks of listeriosis identified by the EFSA between 2010 and 2017, seven (22%) were linked to the consumption of meat and meat products.

Recommendations to consumers:

– For foods that need to be kept cold, the refrigerator should be set to +4°C or less. Whenever surfaces have been soiled by food, clean them promptly. Do not place unpackaged foods directly on shelves.

– Observe household hygiene: clean utensils and work surfaces before and after use, and wash hands after handling raw products.

– Wash vegetables and herbs thoroughly before eating or cooking.

– Store leftovers for less than 3 days, and in the case of hot food, reheat them to an internal temperature above +70°C.

– Respect use-by dates (UBD) for packaged foods, and consume freshly-cut deli products as rapidly as possible.

– It is recommended for pregnant women and individuals most at risk to avoid foods most frequently contaminated with *L. monocytogenes* such as raw-milk cheeses, particularly soft cheese, ready-grated cheese, cheese rind, smoked fish, raw shellfish, tarama, germinated seeds consumed raw, and cooked cured meats.

1.5.5. *Staphylococcus aureus*

(Fosse 2003; Regulation EC 2073/2005; ANSES 2011e; Piveteau 2017; European Food Safety Authority 2017; European Food Safety Authority 2018)

Description of the agent	Morphology: Gram-positive cocci, immotile, facultative aero-anaerobic Conditions of toxinogenesis: Tp. opt. 34–40°C– Tp. L.: < 10°C; > 45°C - pH opt. 7–8; pH L. < 5 > 9.6 Aw opt: 0.99 – Aw L < 0.86 – %NaCl opt: 0 - 4% - NaCl L.: 10% Enterotoxin stable to temperature (D_{121} = 3–8 min), to ionization and to freezing
Reservoir	Ubiquitous host of the skin, mucous membranes and nasopharyngeal sphere of warm-blooded animals, including humans, purulent lesions and mastitis in dairy cows Humans and animals can thus be at the origin of environmental contamination Carriage in humans: Intestinal (20–30%), nasal (20–55%) and hands of catering personnel (10%)
Pathogenicity and clinical signs	Emetic enterotoxin pre-formed in food, thermostable MID: Between 20 and 200 ng of toxin (10^6 CFU/g required) Incubation: 30 min–8 h Nausea, severe vomiting, abdominal pain, hypotension and dizziness Duration of symptoms: 24 h Hospitalization (16%) and exceptional lethality (0.02%)
Foods involved	Raw milk and dairy products, all handled products (human contamination) Meats: 13% of cases
Control	Detection of mastitis in dairy cows Slaughter hygiene Compliance with hygiene rules during processing Compliance with the cold chain Vegetative form destroyed by cooking for several minutes at 60°C Thermostable enterotoxin

Table 1.8. *Staphylococcus aureus: characteristics*

Staphylococci are well-known bacteria in human pathology. The species *Staphylococcus aureus* (Staphylococcus aureus) produces many toxins, but certain coagulase-positive strains produce an emetic enterotoxin (SE) responsible for a

well-known food-poisoning in collective catering. This toxin, pre-formed in food, remains one of the main causes of foodborne outbreaks at present (first in France, second in Europe behind *Salmonella*) and one of the most impressive given the very rapid onset of violent and severe vomiting (known in the past as "banquet disease"), which can cause severe hypotension. In 2018, 115 outbreaks of foodborne outbreaks caused by *S. aureus* enterotoxin were identified in the EU, involving 1,124 patients, 167 of whom were hospitalized, with no deaths.

The characteristics of *Staphylococcus aureus* are presented in Table 1.8.

1.5.6. *Clostridium botulinum*

(Fosse 2003; Popoff 2017a; European Food Safety Authority 2017; European Food Safety Authority 2018; ANSES 2019)

Food botulism is a well-known disease, which was originally associated with the consumption of meat (*botulus* meaning sausage). This disease represents one of the most severe FBIs, characterized by a neuroparalytic syndrome that is often fatal without treatment.

It is a form of poisoning. The botulinum toxin pre-formed in food during bacterial growth following germination is the most potent toxin known (1 million times more toxic than strychnine). Today, food botulism is rare, representing around one-fiftieth of the 50,000 cases of food-poisoning outbreaks reported in the EU each year. In 2018, 15 outbreaks of *C. botulinum* foodborne outbreaks were identified in the EU, that is, 0.3% of all households, affecting 48 patients, of whom 35 were hospitalized and two died. The *C. botulinum* species groups together strain that has in common the fact that they produce a paralyzing neurotoxin. Strains are grouped into six groups based on related biochemical properties, but strains belonging to different groups have such different characteristics that they could be considered species in their own right. Spore resistance is at the origin of the preserves sterilization schedule, which makes it possible to obtain 12 decimal reductions of spores of *C. botulinum* type A, as a result of treatment for 3 min at 121°C.

While foodborne botulism is most often contracted through the consumption of food in which the toxin has been synthesized, toxi-infections are also described: consumption of spores that germinate in the DT with growth and toxinogenesis in the intestine (this is the case with infant botulism from the consumption of unpasteurized honey).

The characteristics of *C. botulinum* are presented in Table 1.9.

Description of the agent	Morphology: Straight, gram-positive bacillus, motile, spore-forming and strict anaerobic Six groups (I–VI), eight toxinotypes (A–H) Production of neurotoxins in the growth phase Mesophilic, except type E, which is psychrotrophic Heat-resistant spores Tp opt: 37°C Tp L: < 10°C - pH L < 4.5 Aw L < 0.93 - %NaCl inhib. > 10%
Reservoir	Telluric germ: Spores present in the soil, marine sediment and freshwater, digestive tracts of vertebrate and invertebrate animals Highly-resistant spores in the environment
Pathogenicity and clinical signs	Production of preformed neurotoxins in food: Poisoning Most potent toxin known: Lethal dose, per os: 1mg/kg Incubation: 12–48 h on average (5 h–8 days) Flaccid neuroparalytic syndrome, without sensitivity impairment: Constipation, eye damage (blurred vision, diplopia and mydriasis) Oropharyngeal disorders (dryness and difficulty in swallowing), paralysis of the limbs and respiratory muscles, death from respiratory failure Lethality: 0–5% in France Note: Possible poisoning by ingestion of spores that germinate in the digestive tract (infant botulism)
Foods involved	Homemade and handmade products and preserves, for which the production process is not controlled: Germination, growth and toxinogenesis (insufficient temperature, pH > 4.5, insufficient Salt or Sugar content, anaerobiosis Meats (type B - proteolytic): Handmade cured meats (raw ham), homemade preserves, Other: Vegetable preserves (type A) and seafood (type E)
Control	Appertization of non-acidic preserves: Spore destruction: 10 h at 100°C - 3 min at 121°C (*C. botulinum* Group I) Toxin destruction: 10 min at 100°C Pickled products: Nitrite salt (most effective additive) 150 mg/kg max

Table 1.9. *Clostridium botulinum: characteristics*

1.5.7. *Clostridium perfringens*

(Fosse 2003; ANSES 2017a; European Food Safety Authority 2017; Popoff 2017b; European Food Safety Authority 2018)

Description of the agent	Morphology: Large, gram-positive bacillus, non-motile, spore-forming, aerotolerant anaerobic Five toxinotypes (A, B, C, D and E) Thermotrophic, sporulation and toxinogenesis in the digestive tract Tp opt: 40–45°C (Generation time: 7 min) Tp L: < 10°C; > 52°C - pH opt: = 6–7 pH L: < 5; > 8.3 Aw opt 0.96 – Aw L < 0.95 - % NaCl inhib. > 6.5
Reservoir	Commensal bacteria from the human and animal digestive tract (concentration < 10^3/g). Also present in water and soil (ubiquitous) where spores survive for many years
Pathogenicity and clinical signs	MID: 10^8–10^{10} vegetative forms Germination and multiplication in culinary preparations, then sporulation and production of the enterotoxin (CPE) in the intestine (6–8% of strains) Incubation: 8–24 h Acute abdominal pain, diarrhea, nausea and fever Most often benign disease (*C. perf* type A and CPE toxin) Severe, often fatal, necrotizing enteritis (*C. perf* type C and beta toxin), rare
Foods involved	Particularly in collective catering Meat products (low initial contamination:10–10^2/g) Mixed preparations with meat left standing for a long time at room temperature in large volumes (germination and multiplication) Meats in sauce (beef, pork and chicken), beef tongue and stew
Control	Contamination: Hygiene of slaughter and handling Growth: Rapid cooling after cooking Destruction: Vegetative forms destroyed by cooking for several minutes at 60°C Destruction of spores difficult in food: 100°C more than 30 min

Table 1.10. *Clostridium perfringens: characteristics*

Clostridium pefringens has long been one of the most frequently detected agents in outbreaks of food poisoning in collective catering through the consumption of pre-cooked meat dishes. Today the incidence is lower: 6.5% of cases reported in France in 2018 were identified by Public Health France (106 out of 1,630 cases). In 2018, 71 outbreaks of *C. perfringens* were identified in the EU, that is, 1.4% of all households, nevertheless affecting 1,783 patients, of whom 18 were hospitalized and two died.

C. perfringens secretes numerous toxins, including enterotoxin, designated CPE, which unlike other toxins is synthesized during sporulation. Control is ensured by rapid cooling of meats in sauce (from 63°C to below 10°C in under 2 h), compliance with the cold chain (0–3°C) and rapid heating of meats (from 10°C to above 63°C in under 1 h).

The characteristics of *C. perfringens* are presented in Table 1.10.

1.5.8. *Toxoplasma gondii*

(Euzeby 1998; Fosse 2003; ANSES 2011f; Villena 2017; European Food Safety Authority 2017; European Food Safety Authority 2018)

Toxoplasmosis is a well-known disease affecting pregnant women, and contracting the disease for the first time during pregnancy can have dramatic consequences for the child. Cats, the definitive host of this parasite, are often unfairly demonized, because while responsible for dispersing oocysts, they are rarely at the origin of the direct contamination of their owners, with the oocyst excretion period no more than a few days throughout a cat's whole lifetime.

Toxoplasmosis is more often contracted through food, through the consumption of cysts in the meat of slaughter animals (30–60% of European outbreaks are associated with the consumption of raw or undercooked meat) or by ingesting oocyst-contaminated vegetables (Figure 1.10). The characteristics of *Toxoplasma gondii* are presented in Table 1.11.

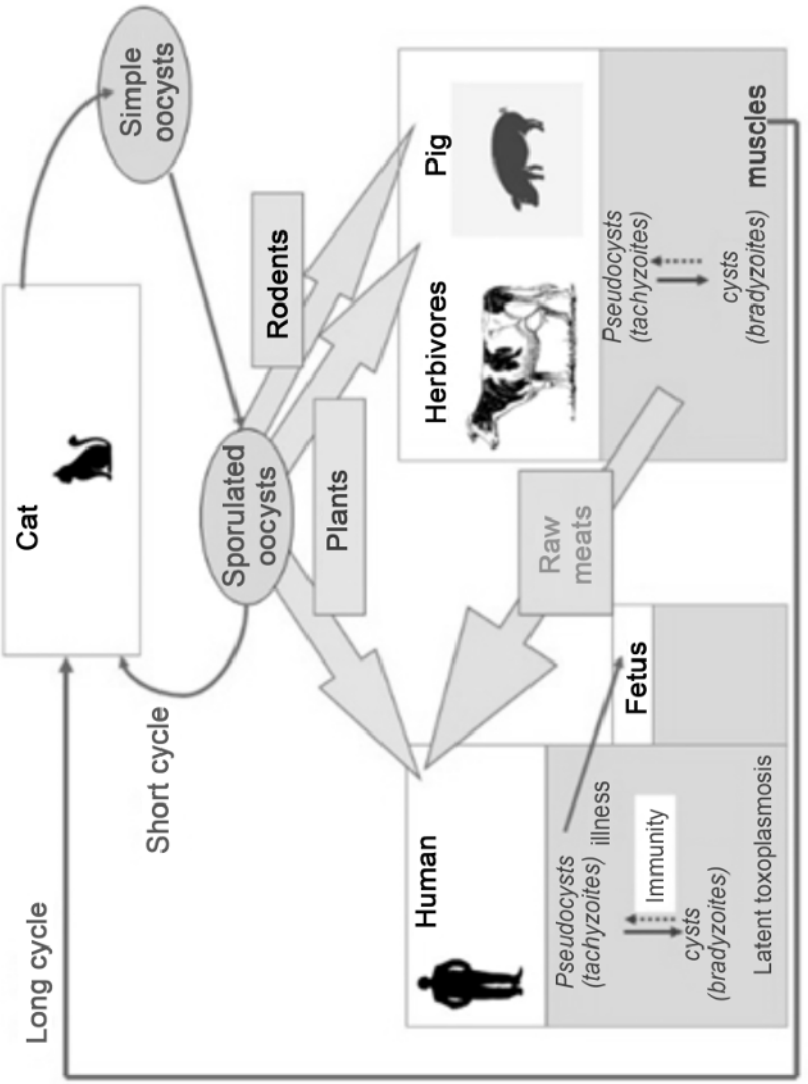

Figure 1.10. *Toxoplasma gondii cycle. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

The disease calls on the notion of a susceptible population because a functional immune system enables the cysts to be maintained in a quiescent state. Its distribution is global: seroprevalence in the world is 7–80% depending on the country, estimated at 35% in France. The prevalence of cysts in slaughter animals varies according to species, age and breeding method: the most significant level is found in sheep (25% on average, 70% in adults, 15% in lambs), followed by goats, free-range pigs, cattle and poultry.

Description of the agent	Order: *Coccidiae*, phylum: *Apicomplexa* Unicellular protozoa, Obligate intracellular parasite Sexual reproduction in the intestine of the definitive host (cat) Asexual reproduction in different tissues of intermediate hosts (mammals and birds)
Reservoir	Animal reservoir (intermediate and definitive hosts) Soil and water reservoir: Dispersion and resistance of oocysts in the environment (2 years)
Pathogenicity and clinical signs	MID: Several dozen oocysts or cysts Incubation time: 2–3 weeks Frequently asymptomatic infection (80%) Immunocompetent population: A benign disease with cervical adenopathy, fever, myalgia and asthenia Immunocompromised population: (Primary infection or reactivation of the disease): Severe forms with multiple localizations (encephalitic, visceral and ocular) Congenital toxoplasmosis: Fetal transmission, all the more severe, the earlier the infection is contracted: Abortion, jaundice and neuro-ocular forms
Foods involved	Meat containing cysts (bradizites): Sheep, goats, free-range pigs, cattle and poultry Plants contaminated by oocysts
Control	Biosecurity measures on farms (prevent cats from accessing animal feed storage) Macroscopic detection impossible at the slaughterhouse Destruction of cysts in meat: 67°C, -12°C at the core for 3 days, 1kGy ionization Destruction of oocysts on vegetables: 1 min at 60°C, survival at -20°C and ionization 0.5 kGy Curing and smoking ineffective

Table 1.11. *Toxoplasma gondii: characteristics*

1.5.9. *Trichinella* spp.

(Euzeby 1998; Fosse 2003; Murell 2013; Villena 2017; European Food Safety Authority 2017; European Food Safety Authority 2018; ANSES 2020)

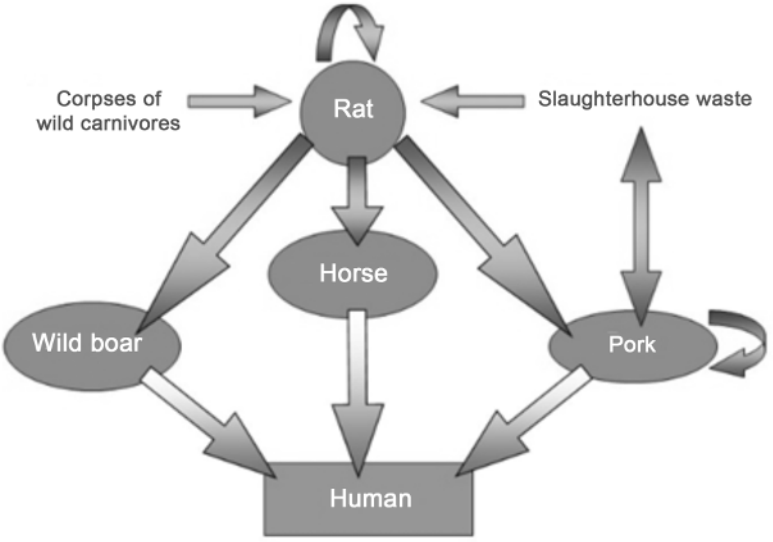

Figure 1.11. *Cycle of Trichinella spiralis. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

Trichinosis is a common helminthosis in humans and many carnivorous or omnivorous mammals, caused by roundworms, or nematodes (Figure 1.11). The species *T. spiralis* is considered the most pathogenic and with *T. britovi*, the species most frequently responsible for disease in humans. The larva and the adult parasitize the same host: the adult form lives in the intestine and the larvae in the striated muscle tissue in the form of characteristic microscopic cysts. Transmission to humans is exclusively foodborne: the ingestion of larvae present in muscle tissue is at the origin of the contamination. The larvae develop into adults in the intestine, where sexual reproduction results in the formation of larvae that travel throughout the body via the lymphatic and blood systems before reaching the striated muscle

fibers where they can remain viable for several years. The associated severe clinical signs are related firstly to the circulation of larvae throughout the body and then to their installation in muscle tissue. While the disease is widespread worldwide, it remains rare in France (two cases per year on average) and Europe (114 cases in the EU in 2018, including 76 hospitalizations, with an incidence of 0.01 per 100,000 inhabitants). The incidence is high in the United States and 90% of the population is reported to be infested in the Arctic region as a result of the consumption of bear meat. In Europe, the incidence among pork pigs is low: on closed farms: 0 positive out of nearly 77 million animals tested: on open farms: 248 positive out of 136 million pork pigs tested. Human cases are primarily associated with the consumption of free-range pork or wild-boar meat.

The characteristics of *Trichinella* spp. are presented in Table 1.12.

Description of the agent	Class: Nematodes Branch: Nemathelminths Nine species described, all pathogenic to humans Six encapsulated species in the muscles: *T. spiralis, T.nativa, T. britov, T. murelli, T.nelsoni* and *T. patagoniensis* Three non-encapsulated: *T. pseudospiralis, T. papuae* and *T. zimbabwensis*
Reservoir	Most significant reservoirs: Wild boars and pigs, bear and walrus Circulation in wild fauna in France: Parasite largely present in foxes in Europe
Pathogenicity and clinical signs	MID: Several dozen larvae Incubation: 5–21 days Diarrheal syndrome (larvae in the intestine) Fever, allergic events, facial edema, myalgia, myocardial and neurological signs (migration of larvae in the body), for 3–4 weeks Chronic muscle pain (implantation of larvae in the muscle cells) for several years

Foods involved	In France: Native cases primarily related to the consumption of undercooked uncontrolled wild-boar meat; imported cases from bear meat (horse meat involved in several outbreaks in the 1980s and 90s) Worldwide: Consumption of pork meat (unmonitored family-scale farms)
Control	Biosecurity on farms: Protection of wildlife animals Detection according to REC 2015/1375: Screening of at-risk animals: Detection method by microscopic examination after artificial digestion. Do not consume pork or wild-boar meat that has not undergone official monitoring. If in doubt, cook thoroughly. Freezing: -21°C for 82 h, -32°C for 22 h: Inactivation of larvae of *T. spiralis*, but larvae of other species (*T. nativa*) are able to resist freezing Safest method: Cooking at 71°C for a few seconds to 58°C for 3 min (meat cooked thoroughly)

Table1.12. *Trichinella* spp.*: characteristics*

1.5.10. *Hepatitis E virus*

(ANSES 2010; Gantzer 2017; Salines 2019; Crotta 2021)

The majority of foodborne viruses are viruses specifically adapted to humans and excreted in large quantities in human feces. Contamination of food then occurs through fecal contamination, due to poor hygiene (handling or use of contaminated water), because the fecal matter of an infected individual contains a very large quantity of viral particles (in the order of 10^{10} viral particles per gram of fecal matter). The food here is simply an inert vector. For example, this fecal-oral transmission occurs in noroviruses, which are very often involved in cases of foodborne outbreaks in industrialized countries, as well as the hepatitis A virus.

Hepatitis E virus (HEV), and in particular genotypes III and IV, is recognized as a zoonotic agent in its own right. It is then transmitted to humans mainly by food, with pork being the main reservoir. This virus can cause acute, fulminant or chronic hepatitis depending on the patient's immune status. No treatment is available at present. Transmission to humans occurs through direct contact with pigs, or by ingestion of raw pork products. This hepatitis is steadily increasing, with

2200 native cases detected by the national reference center (CNR: *Centre National de Référence*) in France in 2017 (number of infections estimated at 68,000 cases per year). Since 2007, more than 90% of the strains identified in humans are of genotype III.

The characteristics of the Hepatitis E virus are presented in Table 1.13.

Description of the agent	Genus *Hepevirus*, family of *Hepeviridae* Non-enveloped, spherical, single-stranded RNA virus of positive polarity, 30 nm in diameter. Eight major genotypes: Genotypes I and II specific to humans – Genotypes III and IV common to humans and to pigs, wild boars and deer. Resistant in the external environment (28 days at room temperature) Susceptible to common disinfectants (bleach, glutaraldehyde)
Reservoir	Humans for genotypes I and II. Suids (pork and boar) for genotypes III and IV. Asymptomatic infection, viremia and dissemination in the body Seroprevalence of pork pigs at slaughterhouse = 50% Genome detected in 4% of pig livers
Pathogenicity and clinical signs	MID: Not clearly known, estimated at 10^5 viral particles per gram Asymptomatic infection in the majority of cases Incubation time: 40 days Acute hepatitis: Jaundice, asthenia, fever and digestive disorders for 1–4 weeks Population at risk of developing severe forms: People with underlying liver disease, immunocompromised individuals, and pregnant women (chronic hepatitis and fulminant hepatitis), leading to hospitalization or death Lethality: 1–4%
Foods involved	Consumption of pork liver or preparation made from raw or undercooked pork liver (figatelli: Raw pork liver sausage) Wild-boar meat and liver, deer liver
Control	Thorough cooking of foods made from pork, wild-boar or deer, meat and liver: Inactivation: 71°C at the core for 5 min or cooking in boiling water for 5 min and 71°C for 20 min in a complex matrix (liver pâté) Hygiene: Wash hands and utensils after handling raw pork liver

Table 1.13. *Hepatitis E virus: characteristics*

1.5.11. *BSE prion*

(Legal 2005; ANSES 2011c; Igel Egalon 2017)

The BSE prion (Proninaceous infectious particles), or mad cow disease, was first detected in the United Kingdom in 1986 and subsequently in most EU countries. Other TSEs were also known in other species (e.g. scrapie of sheep, chronic wasting disease of deer, Kuru and Creutzfeldt Jakob Disease in humans). This proteinaceous UTA was responsible for a health crisis that shook the slaughter-animal meat industry. Mad cow disease was initially considered a strictly animal disease. The discovery in 1996 of a new form of Creutzfeldt Jakob Disease, termed new variant (nvCJD), demonstrated the ability of this agent to infect humans by food through ingestion of cattle tissues infected with the prion. The tissues affected, termed MRS, consist mainly of the central nervous system (brain, spinal cord and dorsal-root ganglion).

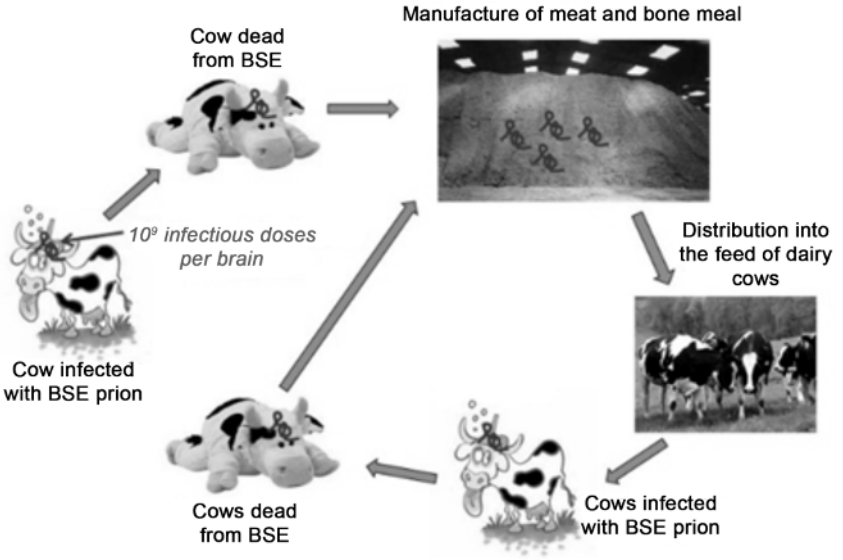

Figure 1.12. *Amplification of the BSE agent through the use of meat and bone meal in ruminant feed. For a color version of this figure, see www.iste.co.uk/haddad/hazards.zip*

This crisis has been an opportunity to apply the precautionary principle, strengthen traceability, prohibit sick animals from the slaughterhouse, manage injured cattle and set up screening tests for cattle considered at risk. It was the

recycling of the transmissible agent in meat and bone meal (animal meal) that was at the origin of the amplification of the bovine disease, which until then had a very low-noise presence (Figure 1.12). The number of cases of cattle testing positive stands at nearly 1,000 in France and about 185,000 in the United Kingdom, although at least 750,000 infected cattle are believed to have entered the food chain. The number of nvCJD cases detected in humans is now 231 worldwide, including 178 in the United Kingdom and 27 in France. Today, while preventive measures have resulted in the control of this epizootic disease, the testing of at-risk animals and removal of MRS from the slaughterhouse nevertheless remain the rule.

The characteristics of the prion responsible for BSE are presented in Table 1.14.

Description of the agent	UTA, infectious protein: PRP (rich in β-sheet), abnormal conformer of the PRP protein (rich in α-helix) normally present. Considerable resistance and stability: Protease resistance – resistant to 600°C for 15 min Destruction: Incineration at a temperature > 800°C, or 133°C at the core under the pressure of 3 atm, in particles <50 mm in size Destruction by chemical treatment: 1M soda bath or Na hypochlorite, 25,000 ppm of chlorine for 1 h
Reservoir	Localization in the central nervous system of cattle affected in very large numbers (10^9 infectious particles per gram of the brain)
Pathogenicity and clinical signs	Incubation time for about 10 years Symptoms secondary to accumulation in the brain of PRPsc aggregates (amyloid plaques) resulting in neuronal loss and the occurrence of vacuoles (sponge-like appearance of the brain) Starts on average at the age of 30 Sensory disturbances, gaze abnormality, mutism and psychiatric signs (depression) Survival is 14 months on average No treatment
Foods involved	Foods of bovine origin contaminated with tissues containing prions (brain and spinal cord)
Control	Prohibition of meat and bone meal Systematic removal of specified risk materials In France, for cattle: The skull and spinal cord of cattle over 12 months of age, the spine and the dorsal-root ganglia in cattle over 3 months of age, the tonsils and the intestine in cattle of all ages Slaughterhouse screening in at-risk animals

Table 1.14. *ESB prion: characteristics*

1.6. References

Alban, L., Häsler, B., van Schaikd, G., Ruegg, S. (2020). Risk-based surveillance for meat-borne parasites. *Experimental Parasitology*, 208, 107808.

Anses (2010). Fiche de description de danger biologique transmissible par les aliments Virus de l'hépatite E. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/MIC2010sa0145Fi.pdf.

Anses (2011a). Fiche de description de danger biologique transmissible par les aliments *Campylobacter jejuni Campylobacter coli*. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0079Fi.pdf.

Anses (2011b). Fiche de description de danger biologique transmissible par les aliments *Listeria monocytogenes*. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0081Fi.pdf.

Anses (2011c). Fiche Maladies animales. ESB Encéphalopathie Spongiforme Bovine. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/SANT-Fi-ESB.pdf.

Anses (2011d). Fiche de description de danger biologique transmissible par les aliments *Salmonella spp*. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0080Fi.pdf.

Anses (2011e). Fiche de description de danger biologique transmissible par les aliments *Staphylococcus aureus* et enterotoxines satphylococciques. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0076Fi.pdf.

Anses (2011f). Fiche de description de danger biologique transmissible par les aliments *Toxoplasma gondii.* Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK 2016SA0271Fi.pdf.

Anses (2012). Fiche de description de danger biologique transmissible par les aliments *Taenia saginata/Cysticercus bovis.* Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/MIC2011sa 0229Fi.pdf.

Anses (2017a). Fiche de description de danger biologique transmissible par les aliments *Clostridium perfringens*. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK 2016SA0073Fi.pdf.

Anses (2017b). Fiche de description de danger biologique transmissible par les aliments *Yersinia enterolitica Yersinia pseudotuberculosis*. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0266Fi.pdf.

Anses (2018). Fiche de description de danger biologique transmissible par les aliments / *E. coli* entérohémorragiques (EHEC). Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK 2017SA0224Fi.pdf.

Anses (2019). Fiche de description de danger biologique transmissible par les aliments / *Clostridium botulinum*, *Clotridium neurotoxinogènes.* Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0074Fi.pdf.

Anses (2020). Fiche de description de danger biologique transmissible par les aliments *Trichinella spp*. Agence Nationale de Sécurité Sanitaire, Alimentation, Environnement, Travail [Online]. Available at: www.anses.fr/fr/system/files/BIORISK2016SA0265Fi.pdf.

Bailly, J.D., Brugère, H., Chardon, H. (2012). *Micro-organismes et parasites des viandes : les connaître pour les maîtriser, de l'éleveur au consommateur*. Cahiers Sécurité des aliments, Centre d'information des viandes, Paris.

Boumar, Z., Namdari, F., Virlogeux-Payant, I., Velge, P. (2017). *Salmonella enterica*. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L. Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc., Paris.

Brugère, H., Auvray, F., Mariani-Kurkdjan, P., King, L.A., Loukiadis, E. (2012). *E. coli* producteurs de Shigatoxines (STEC) : définition, virulence et propriétés des souches entérohémorragiques (EHEC). *Bulletin épidémiologique hebdomadaire*, n°50.

Cappelier, J.M. and Brugère, H. (2013). Les infections à Escherichia coli entérohémorragiques (EHEC). Recueil des conférences des Journées nationales des GTV.

Caprioli, A., Morabito, S., Brugère, H., Oswald, E. (2005). Enterohaemorrhagic *Escherichia coli* : Emerging issues on virulence and modes of transmission. *Vet. Res.*, 36, 289–311.

Crotta, M., Lavazza, A., Mateus, A., Guitian, J. (2021). Viraemic pigs entering the food chain are the most likely source of hepatitis E virus (HEV) in pork meat: Modelling the fate of HEV during slaughtering of pigs. *Food Control*, 121, 107662.

Di Ciccio, P., Conter, M., Zanardi, E., Ghidini, S., Vergara, A., Paludi, D., Festino, A.R., Ianieri, A. (2012). *Listeria monocytogenes*: Biofilms in food processing. *Ital. J. Food Sci.*, 24, 203–213.

Diallo, A., Deschasaux, M., Latino-Martel, P., Hercberg, S., Galan, P., Fassier, P., Allès, B., Gueraud, F., Pierre, F.H., Touvie, M. (2018). Red and processed meat intake and cancer risk: Results from the prospective NutriNet-Sante cohort study. *Int. J. Cancer*, 142, 230–237.

Dorny, P., Praet, N., Deckers, N., Gabriel, S. (2009). Emerging food-borne parasites. *Veterinary Parasitology*, 163, 196–206.

Dubois-Brissonet, F., Naïtali, M., Briandet, R. (2017). Adaptation au stress, tolérance et persistance des bactéries pathogènes dans les environnements alimentaires. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

European Food Safety Authority and European Centre for Disease Prevention and Control (2018). The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2017. *EFSA Journal*, 16, 5500.

European Food Safety Authority and European Centre for Disease Prevention and Control (2019). The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2018. *EFSA Journal*, 17, 5926.

Euzéby, J. (1998). *Les Parasites des viandes : épidémiologie, physiopathologie, incidences zoonosiques*. Lavoisier Tec&Doc, Paris.

Farrokh, C., Jordan, K., Auvray, F., Glass, K., Oppegaard, H., Raynaud, S., Thevenot, D., Condron, R., De Reu, K., Govaris, A. et al. (2012). Review of Shiga-toxin-producing *Escherichia coli* (STEC) and their significance in dairy production. *International Journal of Food Microbiology*, 162, 190–212.

Fegan, N. and Jenson, Y. (2018). The role of meat in foodborne disease: Is there a coming revolution in risk assessment and management? *Meat Science*, 144, 22–29.

Fosse, J. (2003). Les dangers pour l'Homme liés à la consommation des viandes. Evaluation de l'utilisation de moyens de maîtrise en abattoir. PhD Thesis, Université de Nantes, Nantes.

FranceAgriMer (2019). Consommation de viande dans l'UE et en France, Sommet de l'élévage, Direction marchés, études et prospective, Unité élevage.

Gantzer, C. (2017). Virus pathogènes alimentaires. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Hennekinne, J.A., Guiller, F., Herbin, S., Auvray, F. (2017). *Staphylococcus aureus* et autres staphylocoques producteurs d'entérotoxines. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Igel Egalon, A., Béringue, V., Maignien, T. (2017). Prion : un agent infectieux de nature protéique. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Institut National de Veille Sanitaire (2003). Morbidité et mortalité dues aux maladies infectieuses d'origine alimentaire en France. Report INVS AFSSA, Institut National de Veille Sanitaire.

Jeuffé, M. (2017). Étude de l'impact économique de la cysticercose bovine en France métropolitaine en 2016. Thesis, Faculté de médecine de Créteil, Créteil.

Legal, C. (2005). L'ESB : synthèse des connaissances sur le prion et adéquation des mesures de lutte, notamment dans les abattoirs français. Thesis, Faculté de médecine de Nantes, Nantes.

Loukiadis, E. (2017). *Escherichia coli* producteurs de Shiga toxines (STEC). In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Mazaheri, T., Cervantes-Huamán, B.R.H., Bermúdez-Capdevila, M., Ripolles-Avila, C., Rodríguez-Jerez, J.J. (2021). *Listeria monocytogenes*: Biofilms in the food industry: Is the current hygiene program sufficient to combat the persistence of the pathogen? *Microorganisms*, 9, 181.

Messaoudi, S. and Federighi, M. (2017). Campylobacter jejuni et autres campylobacter thermotolérants responsables d'intoxications alimentaires. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Morlot, C. (2011). Etude épidémiologique et statistique de la cysticercose musculaire bovine en France en 2010 – propositions de mesures de contrôle. PhD Thesis, Université Claude Bernard de Lyon I, Villeurbanne.

Murell, K.D. (2013). Zoonotic foodborne parasites and their surveillance. *Rev. Sci. Tech. Off. Int. Epiz.*, 32, 559–569

Piveteau, P. (2017). *Listeria monocytogenes*. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Popoff, M.R. (2017a). *Clostridium botulinum* et autres Clostridium producteurs de neurotoxines botuliques. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Popoff, M.R. (2017b). Clostridium perfringens. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.

Porphyre, V. (2019). Modélisation multi-agents appliquée au secteur de l'élevage porcin à Madagascar pour la conception et l'évaluation de scénarii de lutte contre la cysticercose. Thesis, Université de la Réunion, Saint-Denis.

Règlement (CE) N°853/2004 du parlement européen et du conseil du 29 avril 2004 fixant des règles spécifiques d'hygiène applicables aux denrées alimentaires d'origine animale.

Réglement (CE) N°854/2004 du parlement européen et du conseil du 29 avril 2004 fixant les règles spécifiques d'organisation des contrôles officiels concernant les produits d'origine animale destinés à la consommation humaine.

Règlement (CE) N°2073/2005 de la commission du 15 novembre 2005 concernant les critères microbiologiques applicables aux denrées alimentaires.

Réglement (CE) N°1495/2017 du 23 août 2017 complétant le Règlement (CE) N° 2073/2005 sur Campylobacter chez les carcasses de volailles.

Rodrigues, R., Bronnec, V., Tresse, O., Cappelier, J.M., Haddad, N. (2015). A review of Campylobacter jejuni pathogenesis: Main virulence factors and their use as biomarkers. In *Campylobacter Infections: Epidemiology, Clinical Management and Prevention*, Bertucci, B.A. (ed.). Nova Science Publishers, New York.

Salines, M. (2019). Modélisation de la propagation du virus de l'hépatite E dans la filière porcine et évaluation de stratégies de réduction du risque d'exposition humaine. Thesis, Université de Rennes I, Rennes.

Vernozy-Rozand, C. (2005). *Escherichia coli* producteurs de shigatoxines (STEC). In *Bactériologie alimentaire, compendium d'hygiène des aliments*, Federighi, M. (ed.). Economica, Paris.

Villena, I. (2017). *Toxoplasma gondii*. In *Risques Microbiologiques Alimentaires*, Naïtali, M., Guiller, L., Dubois-Brissonet, F. (eds). Lavoisier Tec&Doc, Paris.