Review

Role of rodents in transmission of *Salmonella* and *Campylobacter*

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Abstract: *Salmonella* and *Campylobacter* are generally regarded as the most important food-borne pathogens in the world. Reduction or elimination of these pathogens in the first part of the food chain (on the farm) is important to prevent disease among consumers of animal products. In organic farming, elimination becomes more difficult, as food animals are allowed outdoors and have easy access to potential sources of hazardous pathogens. Whilst rodents are often associated by organic farmers with infrastructural damage and eating or spoiling of stored feed and products, their zoonotic risks are frequently underestimated. They can amplify the number of pathogens in the environment and transfer them to food animals. Thus organic farmers should be aware of the need for rodent control from a food safety perspective. Preferably, rodent control should form an integral part of a total package of hygiene measures to prevent transfer of food-borne pathogens. These should also include e.g. control of wild birds and flies and obligatory disinfection of boots/clothes and equipment for farm workers and visitors.

Keywords: rodents; *Campylobacter*; *Salmonella*; transmission; contamination; rodent control

INTRODUCTION

Prevention of food hazards in the first part of the food chain is essential to prevent illness of consumers. Control or better elimination of zoonotic pathogens such as *Salmonella* and *Campylobacter* is a priority in today's farming, as human campylobacteriosis and salmonellosis are important causes of gastroenteritis in the industrialised world.[1]

In organic animal production systems, elimination is more difficult, as the animals are allowed outdoors and have easy access to potential sources of hazardous bacteria and/or parasites. Rodents that are present on organic farms can form one of these sources. Organic farms offer an ideal environment for them owing to the application of roughage and straw. Moreover, organic farmers are often less willing to use rodenticides, since they perceive rodent presence as an integral part of the agro-ecosystem.[2]

Whilst rodents are often associated with infrastructural damage and eating or spoiling of stored feed and products, their zoonotic risks are frequently underestimated. Wild rodents can be reservoirs and vectors of a number of agents that cause disease in food animals and humans (e.g. *Leptospira* spp., *Salmonella* spp., *Campylobacter* spp., *Trichinella* spp., *Toxoplasma* spp.).[3,4] In this review we focus on the role of rodents in transmission of *Salmonella* and *Campylobacter* to food animals on farms, particularly when organic production systems are adopted.

CAMPYLOBACTER

Campylobacter are mainly spiral-shaped, S-shaped or curved, rod-shaped bacteria. There are 16 species and six subspecies assigned to the genus *Campylobacter*, of which the most frequently reported in human disease are *C. jejuni* (subspecies *jejuni*), *C. coli* and *C. fetus*. Other species such as *C. laridis* and *C. upsaliensis* are also regarded as primary pathogens but are reported far less frequently in cases of human disease. *Campylobacter* are generally regarded as the most common bacterial cause of gastroenteritis worldwide.[5] In both developing and developed countries they cause more cases of diarrhoea than e.g. food-borne *Salmonella*. Although *Campylobacter* do not show any seasonal variation in developing countries, they do in the developed world, where they peak in summer.[6] *Campylobacter* can survive in the environment for several weeks at temperatures around 4 °C but can also be present in surface water at higher temperatures.[7] Although it is unknown why, in almost all developed countries the incidence of human *Campylobacter* infections has increased steadily over recent years, apart from in 2002 when for the first time a 5% decrease was reported in the European Union (EU).[8] In the Netherlands the rise in *Campylobacter* infections until 2002 could be partially explained by an increase in the level of Dutch poultry meat consumption, which rose from 17.3 kg per head in 1990 to 22.4 kg per head in 2002.[9] It is unlikely that consumption of

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organic poultry meat will have affected these figures, since the market share during this period was less than 1%. While, in the developed world, incidence peaks in infants and young adults, in developing countries, *Campylobacter* infections in children under the age of 2 years are especially frequent, sometimes resulting in death.[10] A total of 149 287 cases of human campylobacteriosis were reported in the EU and Norway in 2002[8] (39 cases per 100 000 inhabitants). Individuals acquire *Campylobacter* infections mainly through contaminated poultry (chicken and turkey) or via consumption of untreated surface water or unpasteurised milk. Infection can also be acquired by direct contact with infected animals, mainly in particular situations such as that of workers in poultry processing plants.[11]

When ingested, *C. jejuni* moves to the ileum and adheres to the surface of epithelial cells of the mucus membrane. Then a toxin is released which leads to the over-secretion of electrolytes into the gut. This results in diarrhoea, which may be bloody, accompanied by headache, fever, vomiting and abdominal pain. These symptoms last for 2–7 days. *Campylobacter fetus* is even more invasive. Infection can lead to spread of the organism from the gut, leading to systemic infection. This can result in septicaemia, pneumonia, meningitis and, in pregnant females, infection of the foetus, which can sometimes lead to spontaneous abortion. Moreover, *Campylobacter* are also linked to Guillain Barré syndrome, a rare but serious paralytic autoimmune disease. Serological evidence of *C. jejuni* infection occurs in about 30% of patients with Guillain Barré syndrome.[12]

Campylobacter are frequently encountered in poultry flocks.[13] They spread easily among live birds[14] through faeces, shared water sources[15] or in the slaughterhouse. In 2002, prevalence in broiler flocks in the Netherlands was 27% and in Denmark 42%.[8] Mostly, *C. jejuni* was isolated, which does not cause disease in chickens but can result in food-borne illness. In poultry meat, *Campylobacter* prevalence was around 30% in the Netherlands in 2002.[8] In France a contamination rate of 88.7% was shown on poultry meat at retail level.[8] In pig herds, high infection rates of 50–80% can also be encountered,[8] but low contamination rates (2.1–4.7%) were found in pig meat. In order to reduce the number of cases of human campylobacteriosis, some countries (Denmark, Sweden, the Netherlands and Norway) have started monitoring programmes on *Campylobacter*. In these monitoring schemes, faecal samples or cloacal swabs are taken at the farm, at the slaughterhouse or at both locations. In 2005, all faecal samples from organic broiler farms ($n = 9$) in the Netherlands were positive for *C. jejuni*.[16] High antibiotic resistance was observed against amoxicilline, doxycycline (tetracycline), metronidazole and the quinolones nalidixine and ciprofloxacin. However, no difference in antibiotic resistance was observed between *C. jejuni* samples obtained from organic and regular broilers. Of interest is the observation that *C. jejuni* is not often found in faeces from organic pig herds, whereas *C. coli* was found on 24 of 31 farms tested (77%).[16]

SALMONELLA

Salmonella are rod-shaped, motile Gram-negative bacteria of the family Enterobacteriaceae. Non-motile exceptions are *S. gallinarum* and *S. pullorum*. More than 2300 serotypes have been described,[17] most of which are non-host-specific.[1] The seroytpes *S. typhi* and *S. paratyphi* are adapted to humans. Individuals usually obtain *Salmonella* by eating contaminated beef, pork, poultry or eggs or by eating vegetables contaminated with animal faeces. Infection with *Salmonella* often causes gastroenteritis with symptoms similar to those seen in *Campylobacter* infections. Young children, older people and immunosuppressed persons are more susceptible to acquiring severe symptoms after infection.[18] The onset of a *Salmonella* infection starts with attachment and internalisation of *Salmonella* to the cells of the small intestine. The invasion of enterocytes results in the extrusion of infected ephitelial cells into the intestinal lumen with consequent villus blunting and loss of absorptive surfaces. Furthermore, *Salmonella* elicit a polymorphonuclear leucocyte influx into infected mucosa and induce watery diarrhoea.[19] If the bacteria are then passed out of the mucosa cells into the underlying tissues, the more severe type of infection can result as bacteria reach the blood and are distributed widely.

In the EU and Norway in 2002 a total of 145 231 cases of human salmonellosis were reported[8] (38 cases per 100 000 inhabitants). *Salmonella enteritidis* was found to be dominant in human salmonellosis, causing 67% of all notified cases in the EU and Norway.[8] *Salmonella typhimurium* caused 17% of all cases. Other important types were *S. infantis, S. virchow* and *S. hadar*.[8] Besides health problems, economic losses due to human infection with *Salmonella* and *Campylobacter* are also considerable. In a study on the socio-economic impact of infectious intestinal disease in England, average costs per case were £606 for *Salmonella* and £315 for *Campylobacter*.[20]

Results from monitoring programmes at slaughterhouses suggest that 20% of broiler chickens in the USA are contaminated with harmful *Salmonella* strains.[21] A 27% incidence of *Salmonella* was found in faeces from organic pig farms, similar to that found on conventional farms monitored during the same period (2003–2005).[16] Of interest was the observation that farms that had just recently started organic pig production encountered *Salmonella* more frequently than farms that had a longer experience with organic production. A recent study could not detect *Salmonella* in faeces from organic broilers.[16]

Salmonella are very persistent. In a study on the survival of *S. enteritidis* in poultry units and poultry food it was found that the organisms could persist for at least 1 year in a trial house stocked with broilers.[21]

Moreover, it was shown that *S. enteritidis* could survive at least 26 months in artificially contaminated poultry food.[22] To date, no reports have been published addressing possible differences in *Salmonella* contamination rates between organic and conventional feeds.

INFECTION OF RODENTS WITH *SALMONELLA* AND *CAMPYLOBACTER*

Wild birds and mammals are generally regarded as the main reservoir for *Salmonella* and *Campylobacter* in the environment. These warm-blooded animals can carry both bacteria in their intestinal tracts, mostly without showing any clinical symptoms of disease.[23] Infected animals can then cause transmission of pathogens from the farm environment to food animals, as is often mentioned in studies on *Campylobacter* and *Salmonella* epidemiology.[11,24–26] Laboratory studies prove that rodents can in principle be infected with *Salmonella* and *Campylobacter*.

Several studies have been undertaken to estimate the prevalence of *Salmonella* and *Campylobacter* in wild rodents (Table 1). In some of these studies the estimation was based on the analysis of faecal pellets. However, as faecal pellets can become infected by deposition in a contaminated environment, the reliability of these studies is probably lower than that of studies based on the analysis of swabs or intestinal contents.

The degree of contamination and transmission risks may differ substantially between different habitats, so a distinction must be made between rodents living in nature (e.g. woodlands, grasslands), those living in urban environments and those living on farms (Table 1). Contamination and transmission may be different on organic farms as compared with conventional farms in view of the fact that the faeces of held animals is shed in the outdoor run. The condition of the outdoor run (paved with concrete or consisting of a soil base) will determine whether the faeces can be removed or not. If infected faeces remains in the outdoor run for prolonged periods of time, this may result in carryover of pathogenic organisms to rodents. As yet, no studies have been carried out to compare the degrees of infection of rodents caught on conventional and organic farms.

Studies on wild rodents in woodlands have shown that only limited numbers are infected with *Campylobacter*. During a study in Norway, *Campylobacter* were not detected in any of the 44 bank voles and wood mice investigated.[27] Faecal pellets voided by 13 bank voles (*Clethrionomys glareolus*), 17 field voles (*Microtus agrestis*) and 12 wood mice (*Apodemus sylvaticus*) trapped in woodlands and grasslands were investigated for *Campylobacter* presence.[28] The authors were able to detect *Campylobacter* in ten out of 13 bank voles tested; the other bank voles were not infected. Isolates from the bank voles resembled a type of *C. fetus* associated with infectious infertility in cattle. In a study on small rodents (in which water voles (*Microtus richardsoni*), longtail voles (*Microtus longicaudus*), Western jumping mice (*Zapus princeps*) and deer mice (*Peromyscus maniculata*) were trapped) in alpine meadows in the USA, *C. coli* was recovered in less than 1% of the isolates.[29] However, the authors proved that, after artificial inoculation with *C. jejuni*, water voles (*M. richardsoni*) can shed the bacterium for several weeks and have the potential to act as a reservoir in high mountainous areas. Although these studies prove that rodents living in nature can be infected, the risk that they may cause food-borne infections is generally low. For wild rodents living on or nearby agricultural premises, this risk is expected to be higher, whereby, as mentioned above, the risk for rodents on organic animal farms may be higher that on conventional livestock farms.

The environment around livestock farms varies considerably but is usually rural. Wildlife, including rodents, is attracted to spilled feedstuffs, the availability of water and the presence of shelter. Since organic farmers often feed their animals in the outdoor run, the chance that rodents are attracted to these easily accessible sites is higher as compared with conventional systems. Generally, the species diversity around farm buildings corresponds to what can be encountered in the surrounding natural or semi-natural environment.[30]

Within poultry farms, infected rodents are often reported, although no distinction has been revealed between organic and conventional systems. Some authors[31] found a high prevalence (24%) of *S. enteritidis* in commensal rodents present on contaminated chicken layer farms. On the other hand, in the same study, *S. enteritidis* was not detected in mice on clean farms. This is logical, as another study revealed that the prevalence of *S. enteritidis* in mice from environmentally positive houses was nearly four times that in mice from environmentally negative houses.[32] In another study, mice (*Mus musculus*) captured in hen houses were assessed for the presence of *Salmonella* in their spleens[33] during two consecutive years. It was found that, during the first and second years, 25 and 18% of the spleens respectively were positive for *S. enteritidis*. Furthermore, passage of *S. enteritidis* in mice may also selectively amplify more egg-invasive and virulent strains.[34] The risks of rodents regarding *Salmonella* persistence in poultry houses have been evaluated in only a few studies: in broiler breeder and layer breeder houses in the UK[13,26] and in layer houses in the USA.[31–33,35] Persistence studies have not yet been reported for organic livestock farms. Chances of *Salmonella* persistence on farms are about two times higher when rodents are encountered by farmers.[36] Rodents can be long-term sources of *Salmonella* infection: it was found that 3-week-old chicks can acquire infection via mice artificially infected with *S. enteritidis* 2 and 5 months previously.[26] Artificially and naturally infected rodents (commensal *M. musculus*) were

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Table 1. Studies on *Salmonella* and *Campylobacter* in rodents

Study	Trapping location	Focus	Species trapped	Number	% infected	Remarks
Rosef *et al*.[27]	Woodlands	Campylobacter	*C. glareolus* (bank vole)	24	0	Swabs
			A. sylvaticus (wood mouse)	22	0	
Fernie and Park[28]	Woodlands and grasslands	Campylobacter	*C. glareolus* (bank vole)	13	77	Faecal pellets
			A. sylvaticus (wood mouse)	12	0	
			M. agrestis (field vole)	17	0	
Pacha *et al*.[29]	Alpine meadows	Campylobacter	*M. richardsoni* (water vole) *M. longicaudus* (longtail vole) *Z. princeps* (western jumping mouse) *P. maniculatus* (deer mouse)	551 in total, not specified	<1	Faecal pellets
Hald *et al*.[49]	Cattle, pig and poultry farms	Campylobacter	Species not specified	44	17	
Henzler and Opitz[31]	Layer farms	*Salmonella*	*M. musculus* (house mouse)	713	24 (species unspecified)	
			R. norvegicus (Norway rat)	2		
			P. maniculatus (deer mouse)	1		
Garber *et al*.[32]	Layer farms	*Salmonella*	*M. musculus* (house mouse)	129	4	Swabs
Singh *et al*.[50]	Urban	*Salmonella*	*Rattus* (species not specified)	254	6	
			M. musculus (house mouse)	109	10	
Davies and Wray[26]	Broiler and layer breeder farms	*Salmonella*	Species not specified	83 dead mice, 152 droppings	47 (intestinal analysis) 35 (liver analysis) 9 (droppings)	
Guard-Petter *et al*.[33]	Layer houses	*Salmonella*	*M. musculus* (house mouse)	621 (year 1) 526 (year 2)	25 (year 1) 18 (year 2)	Spleens
Pocock *et al*.[48]	Mixed agricultural farms	*Salmonella*	*M. musculus* (house mouse)	341	0	Faecal pellets
Hilton *et al*.[51]	Urban	*Salmonella*	*R. norvegicus* (Norway rat)	50 carcasses 100 faecal swabs	10 (carcasses) 8 (swabs)	
Davies and Breslin[43]	Free range layer farms	*Salmonella*	*M. musculus* (house mouse)	25	44	Faecal pellets
			R. norvegicus (Norway rat)	4	0	
Shimi *et al*.[52]	Not specified	*Salmonella*	*M. musculus* (house mouse)	170	10	
Jensen *et al*.[1]	Organic pig farms	*Salmonella*	*M. musculus* (house mouse)	2	0	Intestinal contents
			R. norvegicus (Norway rat)	2		
			A. sylvaticus (wood mouse)	9		
			M. agrestis (field vole)	8		
Meerburg *et al*.[46]	Organic pig and poultry farms	*Salmonella* and *Campylobacter*	*M. musculus*	83	1 (*Salmonella*) 10 (*Campylobacter*)	Intestinal contents
			R. norvegicus	8	0 (*Salmonella*) 12.5 (*Campylobacter*)	
			Other (*A. sylvaticus, M. arvalis, M. minutus* (harvest mouse), *M. agrestis, C. glareolus*)	62	0 (*Salmonella*) 0 (*Campylobacter*)	

found to excrete 10^4–10^6 colony-forming units (cfu) g^{-1} in some individual droppings,[23,26] while their droppings can be contaminated for up to 3 months post-infection.[37] Only a few bacteria are necessary to infect a mouse: in the case of *Salmonella*, 15 are sufficient.[31,38] Mice are also easily colonised by *Campylobacter*.[39,49] Rodents can further amplify the number of pathogens present in the environment: isolates from mice contained three times more *Salmonella* than isolates from the environment of contaminated houses.[31] It has therefore been suggested that rodents constantly reintroduce unstable, orally invasive phenotypes back into the environment of poultry.[40,41] The presence of a resident infected mouse population is thus an important risk factor for egg contamination.[42] Rodents can be a source for oral infection of laying hens with *Salmonella*. In this case, high cell density growth will take place in the intestines of layer hens and the bacteria will have easy access to the eggs. If human pathogenic variants of *Salmonella* survive within the eggs, this could lead to human disease after consumption of contaminated eggs. Molecular fingerprinting[40] demonstrated a close relationship between orally invasive phenotypes by laying hens that resulted in egg contamination and isolates obtained from naturally infected mice. A source for oral infection of poultry is rodent droppings, which are actively sought out by the broilers or layers when mixed in their food or bedding,[26] thus increasing chances of further colonisation. Dead mice can also be a problem, especially if their carcasses are found in poultry houses which have been cleaned and disinfected: *Salmonella* in them may be a hazard for the new flock, as they contain higher levels of organisms than droppings, and dead mice may be pecked and consumed by mature chickens.[26,43]

Rodents can acquire their infection from various sources: they can come into contact with faeces of infected livestock on the farm (a known *Salmonella* infection route[44]), acquire it from other wild animals (e.g. wild birds) or get it from their own family members. Rodents tend to live close to each other, thus enabling the infections to remain resident in the population.[50,51,52]

On swine farms, infected rodents can also be encountered. In a recent study, 5% of the 180 mice caught on swine farms were *Salmonella*-positive.[45] However, in other ecological compartments in that study, *Salmonella* were even more abundant: in cats (12% of samples positive), boots (11%), bird faeces (8%) and flies (6%). It needs to be said that the pigs on nine of the 12 investigated farms were shedding *Salmonella* in their faeces. In an earlier study by our group on organic pig farms we found that about 10% of the house mice (*M. musculus*) were *Campylobacter*-positive and 1% *Salmonella*-positive.[46] Moreover, one out of eight Norway rats was *Campylobacter*-positive. Other rodent species and all insectivores (shrews) were negative for *Campylobacter* and *Salmonella*.[46]

Salmonella are not always encountered in rodents on farms, as has been shown by three studies.[1,47,48] In two of these studies, no *Salmonella* were detected in isolates of any of the house mice caught on the farms.[47,48] In the other study, all small mammals (rats and mice) on the farms were negative.[1] On the other hand, the true *Salmonella* prevalence could be underestimated in these studies, as faecal pellets were analysed.

The use of antimicrobials for prophylaxis, therapy and growth promotion in conventional animal production has led to an increase in antimicrobial resistance of animal pathogens and commensal bacteria, among others *Salmonella* and *Campylobacter*. This can have important implications for human health, as antibiotics used to cure infected humans will work less effectively. Resistance of food animals to some antimicrobials is high. In a recent study on pigs,[53] susceptibilities of *Campylobacter* strains were determined for five antimicrobial drugs. Resistance to tetracycline and erythromycin was high (79 and 55% respectively).[53] Susceptibility testing of *Campylobacter* isolated from poultry showed similar results concerning antimicrobial resistance.[54] Unfortunately, no information is currently available on antimicrobial drug resistance of *Salmonella*/*Campylobacter* isolates from rodents on conventional farms.

The presence of rats on farms has been associated with an increased risk of *Campylobacter* introduction into broiler houses.[15] Another study found that 87% of rat faecal samples tested were positive for *C. jejuni*.[55] Within poultry operations, *Campylobacter* can also be found in mice intestines.[4] A study in New Zealand revealed that seven out of 65 house mice (*M. musculus*) caught in snap traps on a dairy farm were infected with *C. jejuni*.[56,57] These infected rodents may contaminate feed and water, which can then become a source for *Campylobacter* colonisation of food animals.

Potential transmission risks of rodents are probably even higher in organic farming,[5,58,59] as rodents live in closer contact with food animals within these farming systems for the following reasons: (1) food animals have the possibility to go outdoors; (2) food animals are offered roughage and straw in which rodents often hide themselves; (3) organic farmers are less willing to use rodenticides for rodent elimination as it does not fit into their farming philosophy. Moreover, cleaning of outdoor paddocks and practising good hygiene may be difficult in an organic context.[60]

Some studies on pigs[61,62] have shown that risks of meat juice samples being *Salmonella*-positive are higher for free range and organic pig herds than for conventional herds (based on cut-off OD% >10, where OD% is the optical density of the sample relative to the optical density of a positive reference sample).

IMPORTANCE OF RODENT CONTROL

Decontamination of farms is an important step in the reduction of *Salmonella* and *Campylobacter* infection throughout the food chain. Although the disinfection procedure is the main risk factor for pathogen persistence after cleansing and disinfection,[36] the

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efficacy of a proper disinfection procedure is often reduced by the presence of *Salmonella*-infected mice remaining on or returning to the farm after cleansing and disinfection.[37] Mice can acquire infections from inaccessible parts of the livestock houses or outdoor paddocks and then deposit contaminated droppings in places where food animals reside. Because of this food safety risk, even the smallest infestation with rats or mice on farms needs to be addressed.[63,64] It has been shown that rodent control measures can effectively decrease *S. enteritidis* in the hen house.[21,65,66] In one study, 100 conventional flocks were monitored for one production cycle to investigate risk factors for *Campylobacter* infection of broiler flocks.[13] The authors did not encounter evidence of environmental survival of *Campylobacter* in broiler houses after adequate cleansing and disinfection. Further, they did not find that rodents were a source of infection, although they state that most sites operated effective vermin control programmes.[13] Farmers generally apply rodent control to prevent economic losses, as rodents can cause considerable feed losses or structural damage on farms (e.g. gnawing on insulation). However, they only do so when rodent densities exceed a certain subjective threshold.[63] This threshold apparently varies between different countries and between farming systems (conventional *vs* organic). In Denmark it was found that mice were regularly observed on 69% of the farms but that their presence was rarely considered a problem by the farmers.[30] Only 9% of the farmers considered the presence of mice problematic. Rats were more often seen as a problem (by 26% of the farmers), though they were only observed regularly on 39% of the farms. Of the farmers, 53% performed rat control on a regular basis, while for mice the figure was 25%.[30] On the other hand, a survey of 526 farmers in the USA showed that 28% of the farmers considered their farms to have a moderate or severe problem with mice, but only 9% with rats.[21] However, nearly all (99%) the farmers used some method of rodent control. Chemicals or baits were by far the most common method of rodent control. Traps or sticky tape were used on almost half (46%) of the farm sites but were the primary method of rodent control on only 7% of the farm sites. A professional exterminator was used on 14% of the farm sites that practised at least one method of rodent control.[21]

Another survey[63] has demonstrated that there is a difference in rodent control methods between conventional and organic farmers. Conventional farmers mainly use rodenticides, whereas organic farmers also use traps and cats. Sometimes organic farmers also make use of natural predators by stimulating the presence of barn owls, buzzards and kestrels on their farm by placing perches or nest boxes.[63] Recently, it was found that the use of live traps can be an alternative to the use of poison to reduce rodent pressure on organic farms.[2] Live traps are advantageous for the welfare of non-target animals and cause less pollution. These aspects are in line with the organic farming philosophy. On the other hand, the improvement in animal welfare for the trapped rodent compared with poisoning is questionable and farmers need more time to check the traps on a regular basis. Also, live traps are less cost-effective in the short term.[2] A recent mathematical study[67] has demonstrated that the use of cats as rodent exterminators by organic farmers is not recommendable from a food safety perspective. The presence of a large number of cats was found to be positively correlated with *Toxoplasma gondii* seroprevalence in organic pigs.[67] This protozoan parasite causes toxoplasmosis, which in turn can result in serious health disorders in humans, including mental retardation, encephalitis and blindness. Organic farmers should thus limit the access and number of cats on their farms.[67]

CONCLUSION

Both *Salmonella* and *Campylobacter* can cause serious health problems in humans. Therefore elimination of these pathogens in the first part of the food chain should have priority, especially in an organic context. Wild rodents are generally not much of a problem as they do not come into close contact with food animals, but rodents in agro-ecological surroundings can be infected with *Salmonella* and *Campylobacter* and transfer these pathogens to food animals or amplify the number of bacteria in the farm environment. A resident infected rodent population could lead to continuously returning infections in the farm environment.

Rodent control should therefore not only be applied by organic farmers to prevent economic losses but also from a veterinary perspective. Although many farmers already use various methods of rodent control (some more effective than others), most only apply them after a certain subjective threshold is passed and mainly to prevent economic losses or structural damage. Therefore there is a clear need to stress the importance of rodent control for food safety purposes in organic farming.

Because of their ecology, commensal rodents (house mice and rats) pose a particular threat as they live close to livestock and have good reproduction capabilities. Preferably, rodent control should form an integral part of a total package of hygiene measures. These should also include e.g. control of wild birds and flies and obligatory disinfection of boots/clothes and equipment for farm workers and visitors. Continuous monitoring of rodent populations and prevention (e.g. proofing of farm buildings, removal of piles of old material, removal of habitat elements for rodents near stables, limiting access to feed and water) will limit the development of high rodent densities on organic farms. In this way, contact opportunities between rodents and food animals are reduced as much as possible.

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