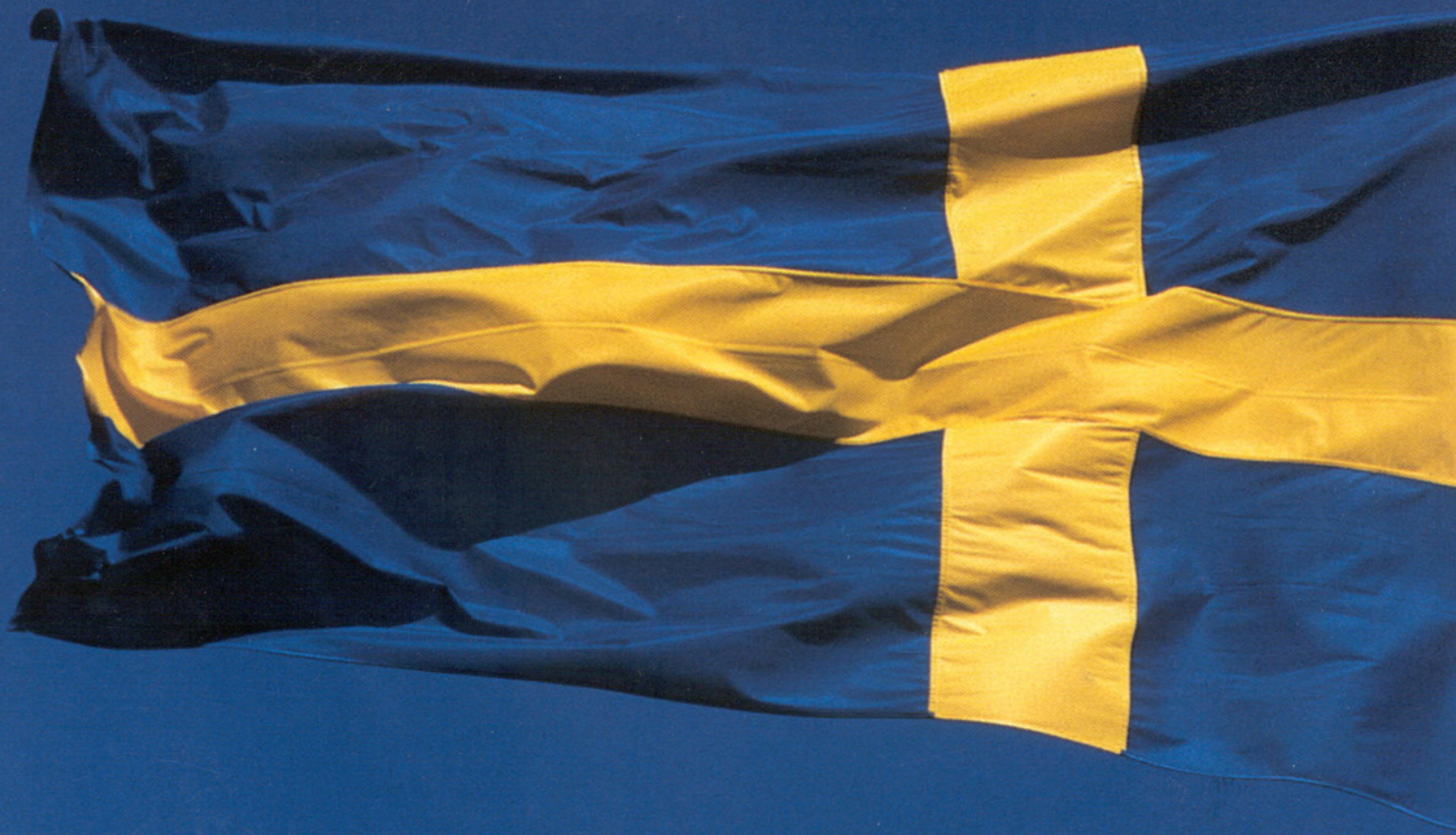

Zoonoses in Sweden

UP TO AND INCLUDING 1999

INDEX

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Introduction

Zoonoses as defined by the WHO are diseases and infections that are naturally transmitted between vertebrate animals and man. Many zoonotic infections in animals are subclinical or mild, and animals may also become long-term carriers, i.e. reservoirs of the infectious agent. Transmission from animals to humans can occur either by direct contact with an infected animal or by indirect contact, for example ingestion of faecally contaminated food or water or transmission by vector animals (eg. arthropods).

Sweden has a long tradition of controlling animal diseases including zoonoses. Control of zoonotic diseases has been performed in the whole production chain, from feed to food. An example of this is the Swedish *Salmonella* control, which has succeeded in keeping the primary production practically free from *Salmonella*.

The zoonosis situation in Sweden is favourable. Of zoonotic infections in animals mentioned in this report, Sweden is free from *Mycobacterium bovis*, *Brucella spp*., *Echinococcus multilocularis* and *Rabies* virus. Sweden has an exceptionally low occurrence of *Salmonella* and the occurrence of *Campylobacter* in broilers is low.

However, the situation in Sweden as well as in other countries is dynamic and several factors may affect the present risk of exposure for humans such as;

- Industrialisation of animal production including increasing herd sizes
- Increasing animal movements, especially trade between countries
- Changes in management of animals, for example more animals kept out-doors
- Industrialisation of food production, including development of new food products and new methods for preservation
- Increased re-cycling of animal, human and industrial waste
- New emerging or re-emerging infections
- Changes in climate, e.g. green house effect
- Changes in susceptibility in the human population, for example an increase in the number of immunocompromised persons

To further improve the zoonotic situation, close co-operation between veterinary, medical and other experts is necessary. In Sweden, a Zoonosis Council (zoonosråd) has been established to promote co-operation and to ensure that all relevant aspects are considered when handling zoonotic issues. The members of the Zoonosis Council are representatives of the National Board of Health and Welfare, the Swedish Board of Agriculture, the Swedish Institute for Infectious Disease Control, the National Food Administration, the National Veterinary Institute, and the Swedish Association of Local Authorities. A zoonosis centre, placed at the National Veterinary Institute, has also been established as an executive body for the Zoonosis Council.

The aim of the present report is to summarise the Swedish zoonosis situation in humans, animals, food and feed reflecting both veterinary and medical aspects. The report has been produced in close co-operation between the Swedish Institute for Infectious Disease Control, the National Food Administration, the Swedish Board of Agriculture and the National Veterinary Institute. In addition, other experts and respresentatives from the industry have contributed to the report.

The report deals with zoonotic diseases considered to be of major interest in Sweden. It covers the period from 1997 (or earlier) up to and including 1999. A brief description of each disease/infectious agent is also included as background information to figures presented.

I would like to thank all who have contributed to the present report. I would also like to thank the Minister of Agriculture, Food and Fisheries, Margareta Winberg, for providing the funding that made this report possible. Finally, I would also like to thank the Zoonosis Council for supporting and promoting the idea of producing this report.
On behalf of all contributors, I hope that this report will give a useful overall view of the Swedish zoonosis situation.

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Notification

Humans

There are two reporting systems for communicable diseases in Sweden (Table 1):

i) Diseases that are notifiable under the Communicable Disease Act. These diseases are reported by the physicians and by the laboratory.

ii) Diseases that are reported on a voluntary basis by the laboratories.

Figures included in the present report are mainly based on notifications by physicians.

Animals

In Sweden, certain diseases are compulsory notifiable already on the basis of a clinical suspicion. In such cases, an investigation to confirm the diagnosis must always be made. Of the diseases listed in Table 1 the following are notifiable on such a basis: tuberculosis, brucellosis, rabies and salmonellosis. Other diseases or infections are notifiable at laboratory confirmation. Only the index case (primary case) in each herd or flock (epidemiological unit) is reported.

Food

In addition to the above mentioned notification in animals, finding of *Salmonella* in food of animal origin is also notifiable. In official sampling *Salmonella* is notifiable in all food items irrespectable of its origin. There is currently no reporting system in place, where the NFA automatically obtains results from the microbiological investigations of food and food items performed by the local municipalities.

Feed

Findings of *Salmonella* in feed or in the environment where feed is produced is notifiable.

Table 1. Notification of zoonotic diseases/infections in humans, animals, food and feed. Only zoonotic agents described in the present report are included in the table.

Disease/ Disease agent	Humans	Animals [1]	Slaughter-house inspection	Food
DISEASES CAUSED BY BACTERIA				
Salmonellosis/ *Salmonella* spp. [2]	C	S	N	N
Campylobacteriosis/ *Campylobacter* spp.	C	-	-	V
Infection with VTEC O157	C [3]	N [4]	-	V
Yersiniosis / *Yersinia enterocolitica*	C	-	-	V
Tularemia/ *Francisella tularensis*	C	N	-	-
Tuberculosis/ *Mycobacterium bovis* [5]	C	S	N [6]	-
Brucellosis /*Brucella* spp.	V	S	-	-
Chlamydiosis (Psittacosis, Ornitosis)/*Chlamydia psittaci*	C	N [7]	N [7]	-
Listeriosis/ *Listeria monocytogenes*	C	N	N	V
Borreliosis *Borrelia burgdorferi* spp.	-	-	-	-
Granulocytic ehrlichiosis/*Granulocytic ehrlichia* spp.	-	-	-	-
DISEASES CAUSED BY PARASITES				
Giardiasis/ *Giardia duodenalis*	C	-	-	-
Cryptosporidiosis/ *Cryptosporidium* spp.	V	-	-	-
Toxoplasmosis /*Toxoplasma gondii*	C	-	-	-
Trichinosis/ *Trichinella* spp.	C	N	N	-
Echinococcosis/ *Echinococcus* spp.	V	N	N	-
DISEASES CAUSED BY VIRUSES				
Nefropatia epidemica/ Puumala virus	C	-	-	-
Rabies/ Rabies virus	C	S	-	-
Infection with TBE-virus	V	-	-	-

C Notified by physicians and at laboratory confirmation.
S Notified on suspicion and at laboratory confirmation.
N Notifiable at laboratory confirmation.
V Voluntary notification at laboratory confirmation.

1 Only the index (primary) case is notified.
2 Findings of *Salmonella* is notifiable in feed.
3 Infection with Enterohemhorragic *E. coli* notifiable since January 1996.
4 Only cases where connection with clinical infection with VTEC O157 in humans exists. Notifiable since October 1996.
5 *M. tuberculosis* is also notifiable.
6 Notifiable in ruminants, pigs and horses.
7 Notifiable in birds.

Salmonellosis

Introduction

Salmonellosis is caused by the *Salmonella* bacterium. More than 2400 serotypes have been identified. A few serotypes are host adapted; for example *S.* Typhi infects only man and *S.* Pullorum and *S.* Gallinarum only fowls. However, any one of the identified serotypes can cause illness in humans. While many *Salmonella* serotypes may cause invasive disease, some are more likely to do so in man. The habitat of the *Salmonella* bacterium seems to be limited to the digestive tract of animals and humans. Especially animals and humans with mild or unrecognised symptoms, are reservoirs for the bacterium. Therefor the occurrence of *Salmonella* in other habitats (water, food, environment) is due to faecal contamination. Salmonellosis is one of the most frequently reported food borne diseases world-wide. The most important mode of transmission is ingestion of food contaminated with *Salmonella*.

During the last three decades different serotypes of *Salmonella* have increased in the intensive animal production in several countries. For example, the evolution of the *S.* Enteritidis pandemic beginning in the 1980s, reaching its maximum in 1992, led to increased foodborne illness in many countries, associated with consumption of poultry, eggs or products thereof. *S.* Typhimurium DT104 is the latest but probably not the last emerging serotype spreading over the world. However, due to the Swedish *Salmonella* control programme, these serotypes have never spread in the Swedish animal population.

The prevalence of *Salmonella* in feed, live animals and animal products, produced in Sweden is very low, less than 0.05% in beef and pork and 0.1% in poultry at slaughter (Figures 12–17). This unique position has been achieved by a national control strategy from feed to food which was initiated more than 40 years ago. A severe domestic *Salmonella* epidemic during 1953, involving more than 9000 people, demonstrated the need for a more comprehensive control program.

The overall aim of the Swedish *Salmonella* control programme is that animals sent for slaughter shall be free from *Salmonella*, thereby animal products for human consumption will be free from *Salmonella*.

The strategies to reach this aim are as follows (Figure 1):

- To prevent *Salmonella* contamination in all parts of the production chain
- To monitor the whole production chain. Surveillance programmes for feed, live animals, carcasses, meat and food of animal origin are in place
- At any finding of *Salmonella*, necessary actions are taken to eliminate the *Salmonella* infection/contamination. Any food item contaminated with *Salmonella* is deemed unfit for human consumption.

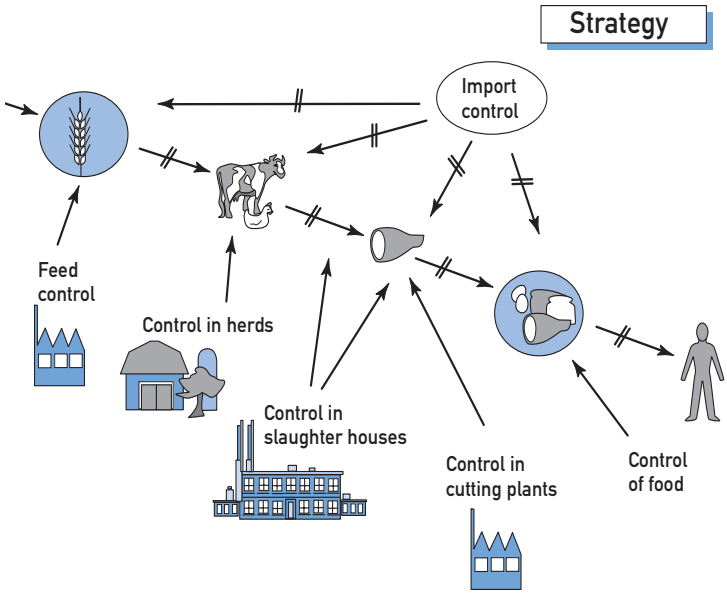


Figure 1. National control strategy of *Salmonella* in the production chain, from feed to fork.

The *Salmonella* control is regulated by parliamentary law and covers animals, feed, food and human health. It regulates the power of the authorities, the routines in case of *Salmonella* infections in domestic animals, the obligations of the animal owner and the financial support to animal owners in cases of *Salmonella* infections in domestic animals. It also regulates domestic feed production and gives rules and recommendations regarding production, hygiene and monitoring of *Salmonella*. Food production is also regulated and rules include both the production and the health status of people working in the food production chain.The import of certain animals and animal products as well as some feed ingredients is also regulated.

All isolations of *Salmonella* in humans, animals, feed and food of animal origin are notifiable. In addition, findings of *Salmonella* in official sampling of food of any origin is notifiable. All primary isolates of *Salmonella* are characterized by sero- and phagetyping the strains and isolates of animal origin are also tested for antibiotic resistance.

Salmonella in humans

Salmonellosis is a bacterial disease that typically causes gastro-enteritis, characterised by diarrhoea, abdominal cramps and dehydration. Patients usually recover within a week without medical treatment. It is estimated that more than 90% of persons with salmonellosis do not seek health care. In many cases the infection may be asymptomatic. A small percent may develop more severe illness with mortality or develop long term syndromes as reactive arthritis or Reiters syndrome.
Salmonella infection in humans is notifiable in Sweden. Since 1980 the annual incidence (cases per 100 000 inhabitants) of notified domestic *Salmonella* cases has varied between 3 and 14 (Figure 2).

A case is considered to be of domestic origin if the person has not been abroad during the incubation period for *Salmonella*. Thereby cases caused by contaminated imported foods as well as secondary cases to people infected abroad are also defined as domestic cases. It should be noted that all culture-positive cases, even subclinical cases, are also included in the notification system. In addition frequency of sampling is rather high compared with other countries; sampling of contact persons both with and without clinical symptoms occur in connection with outbreaks and people with certain "risk professions" may voluntarily be sampled after visits abroad. The increase in domestic incidence observed in 1991 was due to an infection with S. Enteritidis PT 4 in a farm with egg-laying hens causing more than 10 outbreaks in humans and an increased number of sporadic human cases. In addition, a large outbreak on a ferry, due to several serotypes, mainly *S.* Agona occurred in 1991. In 1999, an increased domestic incidence was also observed. This increase was due to several outbreaks mainly occurring in summer months. Although seasonal variation is not observed in Sweden, a peak was observed in 1999 (Figure 3). This was mainly due to one large outbreak in August involving about 200 persons.

Figure 2. Incidence (cases per 100 000 inhabitants) of domestic human *Salmonella* cases 1980–1999 (n=12301).

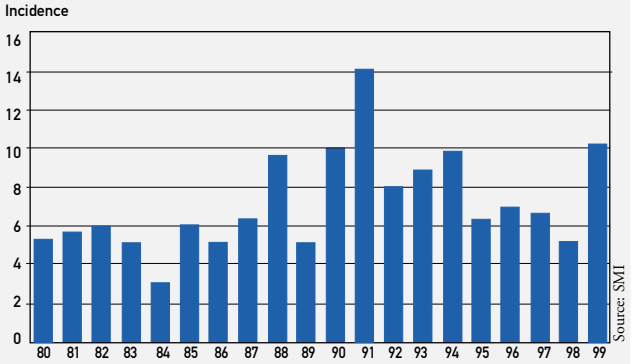
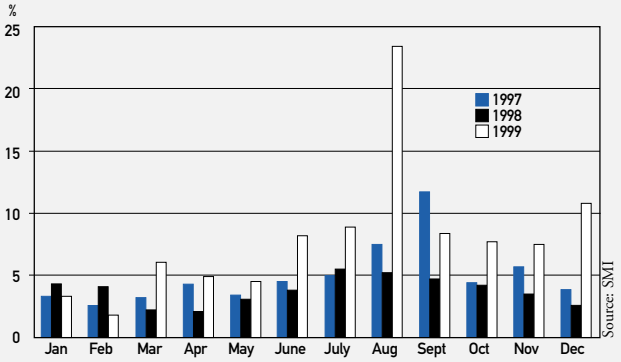
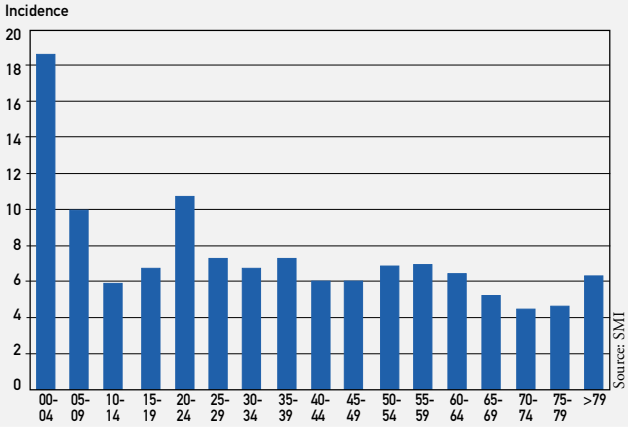


Figure 3. Distribution (%) of domestic human *Salmonella* cases per month 1997, 1998 and 1999 (n=1994).



As in other countries the incidence is higher in young children (Figure 4).

Figure 4. Average incidence (cases per 100 000 inhabitants) of domestic human *Salmonella* cases per age group 1997–1999 (n=1994).



The incidence seems to be lower in northern parts of Sweden, but this might also reflect a difference in care-seeking behaviour.

Salmonella outbreaks

Only a minor part of reported food or waterborne outbreaks are due to *Salmonella* (Table 2). In 1998, a study was performed in a large Swedish community (Uppsala). A total of 268 incidents of suspected food-poisning was recorded involving more than 515 persons. Of these, only one person (<0.05%) was diagnosed as having salmonellosis.

The 10 most common, out of more than 103 identified serotypes in humans, 1997–1999, are detailed in Table 3.

Table 3. The 10 most common *Salmonella* serotypes isolated from domestic human cases 1997–1999 (n=1920).

Salmonella serotype	1997	1998	1999	Number of isolates	%
S. Enteritidis	119	131	331	581	30
S. Typhimurium	124	98	310	532	28
S. Hadar	65	8	12	85	4
S. Stanley	10	11	15	36	2
S. Newport	17	8	9	34	2
S. Blockley	1	3	25	29	2
S. Agona	7	2	17	26	1
S. Virchow	8	5	10	23	1
S. Montevideo	12	6	4	22	1
S. Saintpaul	1	9	11	21	1
10 most common serotypes				1389	72

Source: SMI

S. Typhimurium and *S.* Enteritidis were responsible for approximately 60% of all findings. In 1996–1999, 12, 45, 28 and 98 domestic human cases of *S.* Typhimurium DT104 were reported. A significant part of these were probably due to consumption of imported food. For example, in 1999 at least 64 (65%) *S.* Typhimurium DT104 cases notified as domestic cases were shown to be due to imported contaminated meat (turkey meat and beef). A limited number of these strains have been tested for antibiotic resistance showing that the major part of DT104 had increased antibiotic resistance.

Table 2. Reported *Salmonella* outbreaks, serotypes and phage type of *Salmonella*, sources of infection and number of persons involved in 1997–1999.

Year	Serotype/phage type	Suspected food item	No. of cases in outbreak
1997	*S.* Enteritidis PT6a	Chicken salad	8
	S. Hadar	Several dishes at restaurant	60
	S. Typhimurium DT2	Baked potatoes with mayonnaise	4
	S. Oritamerin	Unknown	16
1998	*S.* Infantis	Unknown	4
	S. Enteritidis	Milk product, private import	3
	S. Typhimurium DT40	Pancake mixture	3
	S. not typed	Several dishes	4
1999	*S.* Typhimurium DT104	Roast beef, imported	31
	S. Typhimurium DT104	Smoked turkey, imported	33
	S. Typhimurium DT104	Unknown	5
	S. Typhimurium DT3	Kebab	5
	S. Typhimurium DT41	Salmon	11
	S. Enteritidis PT8	Several dishes	>200
	S. Enteritidis PT8	Dish served at school	12
	S. Blockley	Bean sprouts	20
	S. Saintpaul	Several dishes	5
	S. Enteritidis NST	Bernaise sauce	87

Source: SMI

* The majority of cases in the large outbreaks are only epidemiologically verified and thereby not included in the number of notified cases.

S. Typhimurium DT40 and DT41 are phage types that are more common among domestic cases than in people infected abroad, thereby indicating a domestic source of infection. During this three-year period, the five most commonly reported serotypes (*S.* Typhimurium, *S.* Enteritidis, *S.* Hadar, *S.* Stanley and *S.* Newport) also were among the 10 most common in each of these years.

Salmonella in animals

Salmonella infection in any animal species is notifiable.

Poultry

Apart from infection with the serotypes *S.* Gallinarum and *S.* Pullorum, which do not occur in Sweden, infection with *Salmonella* in poultry does not normally cause any clinical signs.

The frequency of *Salmonella* isolation in Swedish poultry flocks is very low. Most of today's measures in the *Salmonella* control programme are therefore of preventive nature. Vaccination of poultry against salmonellosis is not allowed in Sweden. Four factors are of major importance to maintain the favourable situation:

- The breeding pyramide is kept free from *Salmonella.* All grandparent animals are imported and all are quarantined and repeatedly tested negative for *Salmonella*.
- Feed free from *Salmonella*: The control consists of three parts; import control of feed raw materials, mandatory heat treatment of compound feeding-stuff for poultry and a HACCP based *Salmonella* control in the feed industry.
- High hygiene and biosecurity standards are in place, preventing introduction of *Salmonella*.
- Measures are always taken in case of *Salmonella* infection in poultry irrespective of serotype.

An extensive sampling programme continuously monitors the *Salmonella* situation in poultry. In addition to the sampling at flock level, samples are also collected at all poultry slaughterhouses to monitor the end product. The mandatory sampling scheme is summarised in Table 4.

Salmonella control programme in poultry

A voluntary and preventive *Salmonella* control programme for meat producing poultry, including all in – all out production, standard of houses and hygienic measures to prevent introduction of *Salmonella* by staff, equipment or feed, has been in force in Sweden for more than 30 years. Mandatory *Salmonella* sampling of poultry has been in force since 1984. Initially, this sampling comprised broilers and other poultry for meat production but in the 1990s sampling was successively extended. In 1993, a voluntary and preventive *Salmonella* control programme was also implemented for layers. Two years earlier an industry-led sampling programme for layers was initiated and in 1994 this sampling became mandatory. The sampling was further intensified in 1995 when Sweden joined the European Union. In 1998, sampling also became mandatory for ratites and quails.

The voluntary and preventive *Salmonella* control programme is administered by the SBA. At present, 100 % of Swedish breeding poultry, 98 % of broilers and about 30 % of the laying hens are affiliated to the voluntary programme.

Table 4. Mandatory *Salmonella* sampling scheme in poultry. Total number of sampling occasions and (frequency of sampling occasions) in poultry.

Category of poultry	Production period				
	Rearing	Eggproduction	Hatchery	Sampling prior to slaughter	Slaughter
Grand parents	5 [a)]	~ 10 [b,c)]	~ 20 [b,d)]	1	[e)]
Parents	3 [a)]	~ 10 [b,c)]	~ 20 [b,d)]	1	[e)]
Layers	1	3	n.r.	1	[e)]
Meat producing poultry [f)]	n.r.	n.r.	n.r.	1	[e)]
Ratites, breeders	4 sampling occasions per year	n.r.	n.r.	1	
Quails, eggproducing	n.s.	2 sampling occasions per year	n.s.	n.s.	

a Extended sampling compared with the zoonosisdirective (92/117/EEC)
b Sampling according to the zoonosis directive
c One sampling per month
d One sampling every second week
e Monitoring at slaughter. Annually about 4000 neck skin samples are collected. From ratites faecal samples are collected.
f Broilers, turkeys, ducks, geese, ratites
n.r. not relevant
n.s. no sampling

If *Salmonella* is isolated from a poultry flock an official veterinarian immediately places restrictions on the whole farm. An investigation to trace the source of the infection or any spread of infection is performed and official samples are collected. The *Salmonella* infected flock is destroyed. After destruction of the flock, the empty poultry house is thoroughly cleaned and disinfected under supervision of the official veterinarian. Environmental samples are collected after disinfection and they have to be *Salmonella* negative before restrictions are lifted and new birds are allowed into the house.

According to present legislation, sanitary slaughter and heat treatment is allowed for flocks infected with non-invasive serotypes but this option is not used. Thus, any poultry flock infected with *Salmonella* irrespective of serotype will be destroyed.

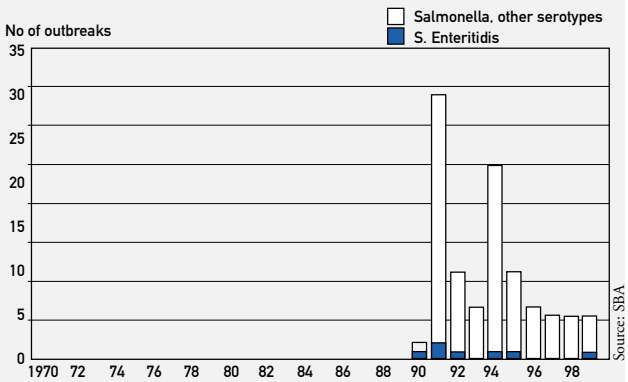
Before joining the EU in 1995, clean, unwashed and undamaged eggs from flocks infected with non-invasive serotypes were allowed to enter the Swedish market for human consumption. However, since 1995, eggs from *Salmonella* infected flocks are not allowed to be used for direct human consumption. Eggs originating from flocks infected with a non-invasive serotype may be used after pasteurisation.

Occurrence of *Salmonella*

Layers

During the last 10 years, the *Salmonella* situation has improved considerably and the number of notified cases have decreased (Figure 5). On average six positive farms have been identified annually since 1995. No cases have been notified in breeders.

Figure 5. Number of notified outbreaks (infected farms) of *Salmonella* in layers 1970–1999.



In 1991, the number of notified *Salmonella* infected laying farms increased sharply. This increase reflects an increased surveillance i.e. the start of the industry led sampling programme in layers.

The dominating serotype has been *S.* Livingstone which accounts for about 67 % of the cases in the 1990s. The total number of herds infected with *S.* Enteritidis is limited to seven farms. This supports the fact that Sweden has not been involved in the world wide spread of this serotype in the beginning of 1990s.

Table 5. *Salmonella* serotypes isolated in layers 1995–1999.

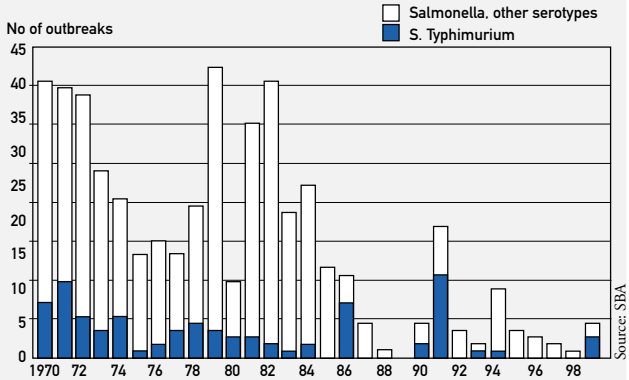
SEROTYPE	No. of infected farms
S. Livingstone	17
S. Mbandaka	4
S. Infantis	3
S. Enteritidis	2
S. Yoruba	1
S. Anatum	1
S. Cubana	1
S. subspecies I	1
S. subspecies IIIb	1
Total	31

Source: SBA

Broilers

The situation in Swedish broilers is very favourable and has improved considerably since the 1970s (Figure 6).

Figure 6. Number of notified outbreaks (infected farms) of *Salmonella* in broilers 1970–1999.



Since the beginning of the 1970s only two breeder flocks (broiler parents) have been found infected with *Salmonella*. *S.* Typhimurium was found in one flock in 1991 and *S.* Typhimurium DT 41 in one flock in 1999.

The peak seen in broilers in 1991 was due to *Salmonella* infected chickens delivered from a hatchery contaminated by the above mentioned *S.* Typhimurium infected breeder flock. An increase in cases was also observed in the early 1980s, the increase was probably due to *Salmonella* contaminated feed. Due to this, sampling of broilers became mandatory in 1984 and heat treatment of feed for broilers became mandatory in 1986. Since then the situation has improved. In 1995–1999, less than five infected broiler flocks a year have been detected. The serotypes isolated are detailed in Table 6.

Table 6. *Salmonella* serotypes isolated in broilers 1995–1999.

SEROTYPE	No. of infected farms
S. Livingstone	4
S. Typhimurium*	3
S. Mbandaka	2
S. Hadar	1
S. Infantis	1
S. Bovismorbificans	1
S. Havana	1
S. Newport	1
S. Rissen	1
S. Tennessee	1
Total	16

Source: SBA

* Incl. one broiler parent farm

Other poultry

In 1995–1999, a total of 20 cases of *Salmonella* have been reported. Serotypes isolated are detailed in Table 7.

Table 7. *Salmonella* serotypes isolated in other poultry 1995–1999

SEROTYPE	No. of infected farms				
	Ducks	Geese	Turkeys	Ratites	Total
S. Typhimurium	1	7	1		9
S. Enteritidis	1	4			5
S. Muenster		3			3
S. Anatum				1	1
S. Mbandaka			1		1
S. Idikan				1	1
Total	2	14	2	2	20

Source: SBA

Livestock

Salmonella have a large variety of animal hosts, domestic as well as wild. The infection usually is subclinical. Infected animals may eliminate the infection but occasionally they may become chronical carriers and shed the bacteria intermittently with faeces. In Sweden infection with *Salmonella* is only rarely associated with clinical disease. Occasionally young calves may develop clinical symptoms, usually fever and diarrhoea.

The frequency of *Salmonella* isolation in Swedish livestock is very low. As in poultry, most of today's measures in the *Salmonella* control programme in livestock are of preventive nature. Factors of major importance are that feed shall be free from *Salmonella*, the breeding pyramide (pigs) shall be free from *Salmonella* and that action is always taken at any finding of *Salmonella*.

Salmonella control programme in livestock

The *Salmonella* surveillance programme in livestock consists of:

- Surveillance of herds/breeding herds at farm level (pigs). Faecal samples (n=60) are collected annually in elite breeding herds, gilt-producing herds and twice annually in so-called sow pools. In addition, all weaner pig producing/integrated herds affiliated to a voluntary health control programme, are tested by annual faecal samples (10 pen samples). At present, approximately 45% of the production of fattening pigs is included in this programme. Annually approximately 8000 samples are collected and analysed.
- Testing for *Salmonella* of all sanitary slaughtered animals as well as any suspected animal at normal slaughter.
- Surveillance at all slaughterhouses: Sampling of slaughtered cattle and pigs (intestinal lymph nodes and swab samples) is carried out in all abattoirs. Annually approximately 20 000 samples are collected and analysed.
- Epidemiological investigations are performed upon finding of *Salmonella*, for example trace back/forward investigations from *Salmonella* infected herds, at findings of *Salmonella* at autopsy or investigations in connection with human cases of *Salmonella*.
- General surveillance by clinical checks made by veterinarians. Samples for culture of *Salmonella* may also be taken at autopsies.

- Action is always taken at suspected or confirmed cases of *Salmonella*

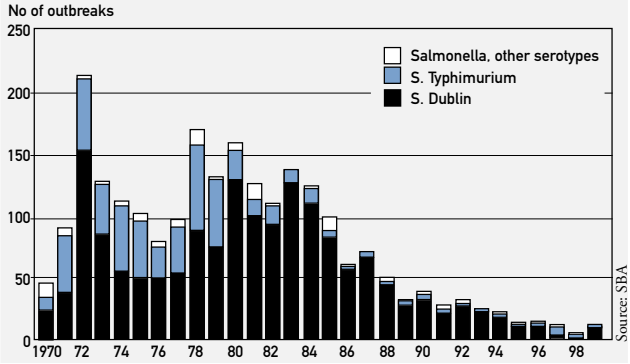
Any suspicion of *Salmonella* leads to investigation in the herd of origin. If *Salmonella* is isolated the herd is put under restriction. Animal movements are not allowed except for sanitary slaughter. Unauthorised persons are not allowed to enter the farm. An investigation is performed by veterinary and public health authorities. Trace-back investigations aiming at identifying the source of infection as well as any spread of infection are instituted. A sanitation plan is instituted and approved by the SBA. The strategy is to identify and eliminate chronically infected animals and prevent spread of infection by instituting hygienic measures in the herd. The whole herd is repeatedly examined for *Salmonella*. After all infected animals are eliminated, the whole herd has to be negative on two consecutive bacteriological examinations for *Salmonella* with a one month interval and the premises have to be cleaned and disinfected according to standards put up by authorities in each case. It may take several months and in some cases more than a year before the restrictions are lifted.

Occurrence of *Salmonella*

Cattle

The *Salmonella* situation in cattle is very favourable, an average of 20 infected herds have been notified annually since 1990. As can be seen in Figure 7, the number of notified infected herds have decreased. During the last five years an average of 10 infected herds have been reported annually.

Figure 7. Number of notified cases (infected herds) with *Salmonella* in cattle 1970–1999.



The most commonly reported serotype was *S.* Dublin, a serotype adapted to cattle. More than 75% of all reported cases were due to *S.* Dublin and 15% were due to *S.* Typhimurium. Serotypes isolated from cattle herds in 1995–1999 are shown in Table 8.

Table 8. *Salmonella* serotypes isolated in cattle herds 1995–1999.

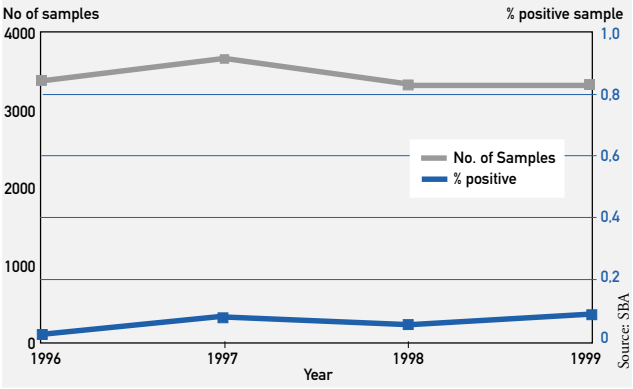
SEROTYPE	No. of infected herds
S. Dublin	36
S. Typhimurium	11
S. Duesseldorf	1
S. Livingstone	1
S. Montevideo	1
S. San-diego	1
S. Stanley	1
S. subspecies I	1
Total	53

Source: SBA

A total of four cattle herds have been found infected with *S.* Typhimurium DT104. The first herd was identified in 1995. All DT104 isolates were penta resistent. All herds have been cleared from *Salmonella.*

Routine surveillance of normally slaughtered cattle was initiated when Sweden joined the EU. Annually about 3000 lymph node samples are collected at slaughter houses. In 1996–1999 between 1–3 (<0.1%) positive samples were found annually (Figure 8).

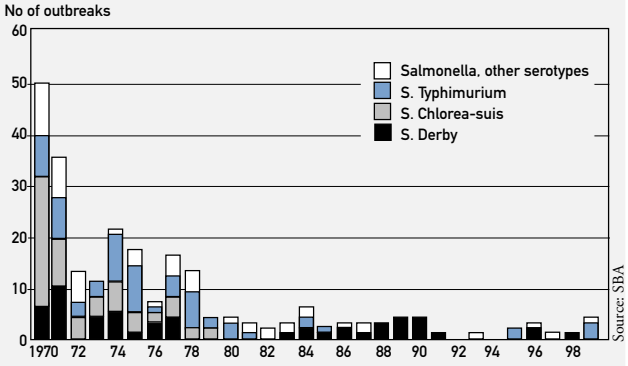
Figure 8. Number of lymph node samples and % positive samples collected from cattle at slaughter 1996–1999.



Pigs

In pigs only sporadic cases of *Salmonella* are notified. *S.* Cholerasuis does not occur in Sweden, the last case was reported in 1979. Infection with *S.* Typhimurium DT104 in a pig herd has never been reported. During the last 10 years an average of 1.7 infected herds have been reported annually (Figure 9).

Figure 9. Number of notified cases (infected herds) with *Salmonella* in pigs 1970–1999.



In 1990–1999 *S.* Derby was reported in 8 cases (47%) and *S.* Typhimurium in 5 cases (29%). *S.* Derby, the most commonly isolated serotype, seems to be adapted to pigs and might be more difficult to eliminate from herds and/or the environment of the herd. By subtyping it has been shown that the same strain of *S.* Derby was isolated repeatedly from one herd for more than 15 years before it was finally eliminated. In 1995–1999 *S.* Typhimurium was the most commonly isolated serotype (Table 9).

Table 9. *Salmonella* serotypes isolated in pig herds 1995–1999.

SEROTYPE	No. of infected herds
S. Typhimurium	6
S. Derby	3
S. Cubana	1
S. Java	1
S. subspecies IIIa	1
Total	12

Source: SBA

When Sweden joined the EU, routine surveillance of normally slaughtered fattening pigs as well as adult pigs was initiated. Annually about 6000 lymph node samples are collected at slaughter houses. In 1996–1999 between 2–7 (<0.3%) positive samples were found in adult pigs and between 1–9 (<0.3%) positive samples were found in fattening pigs annually (Figures 10 and 11).

Figure 10. Number of lymph node samples and % positive samples collected from fattening pigs at slaughter 1996–1999.

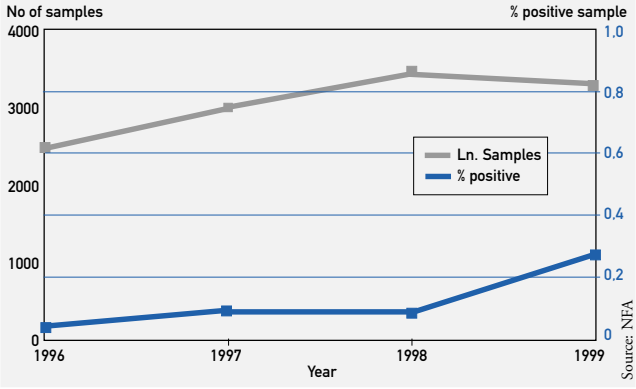
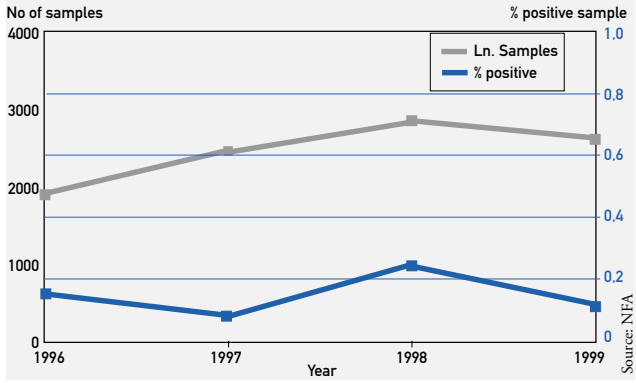


Figure 11. Number of lymph node samples and % positive samples collected from adult pigs at slaughter 1996–1999.



In the *Salmonella* control in pig herds (Table 10) three herds have been identified since 1997.

Table 10. Number of positive findings in the annual *Salmonella* control in pig herds.

Type of herd	1997	1998	1999
Elite breeding herds	0	0	0
Gilt producing herds	1	0	1a)
Sow pools b)	0	0	0
Voluntary sampling c)	0	0	1

a) *Salmonella* could not be re-isolated in the herd
b) Sampling twice annually
c) Sampling in weaner pig producing- integrated- fattening- herds affiliated to a health control programme.

Sheep and goats

Salmonella is very rarely isolated from sheep and goats. In 1995–1999, 3 cases were found in sheep, all being *S.* subspecies IIIb 61:[k]:1,5,(7). In a survey performed in 1998, this serotype could be isolated in the faeces from about 0.5% of slaughtered sheep. Apart from one case in a zoological park, no *Salmonella* were reported in goats.

Companion animals

Salmonella is very rarely isolated from dogs and cats. During the last five years on average 3 cases (a case is defined as a single dog or several dogs in a household or other epidemiological unit) have been reported annually in dogs. Serotypes isolated from dogs and cats in 1995–1999 are shown in Table 11.
S. Typhimurium DT104 has been reported in one occasion, where a bitch and some of her puppies were subclinically infected. In cats, usually less than two cases are reported annually. However, in the winter-spring of 1999 an increase was observed in "out-door" cats. All but one case were due to *S.* Typhimurium DT40 or a specific type of *S.* Typhimurium NST, identified by PHLS, Collindale, United Kingdom as phage type U277. The increased number of *Salmonella* infected cats was probably due to an increase in salmonellosis of the same serotype in passerine birds that occurred during the same time period.

Table 11. *Salmonella* serotypes isolated in dogs and cats (epidemiological units) 1995–1999.

SEROTYPE	No. of infected animals	
	Dogs	Cats
S. Typhimurium	8	85
S. Virchow		1
S. Welikade	1	
S. Livingstone	1	
S. Newyork	1	
S. Newport	1	
S. Putten.	1	
S. Senftenberg	1	
S. Tennessee	1	
S. Alachua.	1	
Total	16	86

Horses

Salmonella infection in horses is seldom reported. In 1995–1999, a total of 14 cases (infected flocks) were reported. All except one were due to *S.* Typhimurium (Table 12).

Table 12. *Salmonella* serotypes isolated in horses 1995–1999.

SEROTYPE	No. of infected herds
S. Typhimurium	13
S. Duesseldorf	1
Total	14

Several of the notified cases have occurred in imported horses. *S.* Typhimurium DT104 have been identified in one horse, a chronically infected foal. The source of infection was probably an imported mare. Increased antibiotic resistance has been found in two *S.* Typhimurium isolates, the previously mentioned DT104 and one DT195, respectively.

Reptiles

Salmonella is commonly isolated from reptiles. Cold-blooded animals are of special interest as small turtles and other reptiles may be kept as house pets. Reptiles are usually symptomless carriers of *Salmonella* species and it is possible that the bacterium exists as a commensal flora in the animal's gut. In 1995–1999, a total of 190 *Salmonella* isolates from reptiles have been recorded. Sixty-five different serotypes have been isolated. The 10 most common are detailed in Table 13.

Table 13. The 10 most common *Salmonella* serotypes isolated in reptiles 1995–1999.

SEROTYPE	No. of infected reptiles
S. subspecies IIIb	35
S. subspecies II	19
S. subspecies IV	11
S. subspecies III	9
S. subspecies IIIa	8
S. Newport	8
S. Abony	5
S. Bardo	5
S. subspecies I	5
S. Adelaide	4
S. Fresno	4
S. Enteritidis	4
Total	117

As close contact may occur between children and reptiles, this is a significant source of domestic *Salmonella* infections in humans.

Wild life

Salmonella is rarely isolated from wild animals. Annually about 1400 wild animals are necropsied at the NVI. In approximately 30% of these cases a bacteriological examination including *Salmonella* is performed. In 1995–1999, two cases of *Salmonella* were reported in wild mammals, a hedgehog and a fox. However, in wild birds 94 cases were notified.

In 1999, a survey was performed where faecal samples from 480 mammals (deers, hares, moose and wild boars) and 216 Canada geese and 111 gulls were collected and analysed for *Salmonella*. All were negative, except gulls, where about 4% of samples were positive. Passerine birds seem to be a reservoir of *S.* Typhimurium DT40 and phage type U277. *Salmonella* infections in these species are reported to vary between different years with a peak in winter-spring. In 1999 an increase was observed in passerine birds and a total of 54 cases were reported, all *S.* Typhimurium DT 40 or phage type U277.

Salmonella in feeding stuffs

The *Salmonella* control of feed has a long tradition in Sweden and has been carried out since the late 1940s. The reason for this early interest was due to the finding of a great number of *Salmonella* serotypes in imported meat- and bone meal. The first initiative to improve the hygienic quality of feed raw materials initially came from the feed industry.

The feed control is supervised by the SBA. In Swedish feed legislation it is stated that *Salmonella* may not be present in animal feeding stuffs. Findings of *Salmonella* spp. in the feed sector are notifiable.

All compound feedingstuffs for poultry has to be heat-treated whereas there is no such requirement stated for feedingstuffs for pig or cattle. Despite this, about 90% of compound feedingstuffs for pigs and 100% of the compound feedingstuffs for cattle is heat-treated. At present about 2.2 million tons of feeding stuffs are produced annually. More than 99% of the feed consumed by livestock is produced domestically.

The production of feed materials of animal origin is controlled according to the present EU legislation, where each batch produced is kept under quarantine until the results of *Salmonella* investigations have been completed. Environmental samples also have to be taken at the plant. Import of meat- and bonemeal for livestock does not occur and has not done so for many years. (At present it is not allowed to use processed animal protein for animals that are kept fattened or bred for the production of food.) Importation of meat- and bonemeal is limited to products for the petfood industry. This material has to be tested for the presence of *Salmonella* before import, in the country of origin. Imported certain feed materials of vegetable origin (considered to constitute a higher risk for *Salmonella*) are investigated for *Salmonella* and major domestic producers of feed materials are also required to monitor their production for the presence of *Salmonella.* In 1995–1999 between 41 and 63 positive samples have been found annually in the import control of feed material of vegetable origin. Positive findings have most commonly been found in soy bean meal, rape seed meal and palm kernel meal.

In the feed mills, great effort is put on the control of raw materials, the heat treatment and preventing recontamination of the heat-treated feed. In 1991, testing of the final product was replaced by a Hazard Analysis Critical Control Point (HACCP) approach where samples from critical control points are analysed for *Salmonella*. This type of surveillance has proven to be more sensitive than the previous testing of the final product. The idea of the surveillance system in feed mills is to enable the earliest possible detection of any *Salmonella* that has entered the mill and also to verify that the feed is not recontaminated. In feed mills producing compound feedingstuffs for poultry at least five samples have to be investigated every week and in feed mills producing only compound feedingstuffs for pig or cattle at least two samples have to be investigated per week. Approximately 7000 samples are analysed at feed mills annually, of which about 5500 are within the compulsory sampling. About 40% of the sampling are performed before heat treatment. On average, five positive samples were isolated annually during 1995–1999 at critical control points after heat treatment. *S.* Typhimurium DT104 has been detected once in an environmental sample. Any positive sample after heat treatment will generate an extensive sampling of the feed mill as well as analysis of feed samples. So far no positive samples have been detected in these investigations.

Table 14. The 10 most common *Salmonella* serotypes identified at import control of feed material of vegetable origin 1995–1999.

Salmonella serotype	No. of isolations
S. Senftenberg	36
S. Mbandaka	31
S. Havana	23
S. Cubana	20
S. subsp. I=4, 12:b:-	19
S. Derby	19
S. Agona	18
S. Tennessee	17
S. Anatum	7
S. Yoruba	6
S. Infantis	5

Dog chews imported from third countries are controlled for *Salmonella*. In addition, importers of petfood are visited annually by official inspectors and samples for *Salmonella* are collected. As this routine sampling indicated that *Salmonella* contamination was a problem, a systematic investigation was performed at a major border inspection post in Sweden during 1998. The investigation showed that 23 (67%) out of 48 investigated consignments with dog chews from third countries were contaminated with *Salmonella*. This highlights the importance of thorough control of imported dog chews.

Pet food, imported from third countries, is controlled for *Salmonella* by random sampling. *Salmonella* has occasionally been found but no systematic sampling has been performed to clarify the true prevalence. Pet foods from the EU are not officially controlled for *Salmonella*.

Salmonella in food

Due to the Swedish *Salmonella* control covering the whole production chain, food of Swedish origin is practically free from *Salmonella*. Compared with most other countries the *Salmonella* situation in food is very favourable.

Extensive surveillance programmes for food are in place and imported food of animal origin is controlled for *Salmonella*. Any finding of *Salmonella*, irrespective of serotype, in food of animal origin is notifiable. However, in official sampling *Salmonella* is notifiable in all food items irrespective of its origin. Any food contaminated with *Salmonella* is deemed unfit for human consumption. If *Salmonella* of any serotype is isolated in food or if any food item is identified as the source of infection for human cases, the origin of the contamination is traced back whenever possible. Consignments originating from EU countries that are found contaminated with *Salmonella* (at spot checks) are traced back if possible and destroyed or returned to the sender. Consignments originating from other countries where *Salmonella* is found at border inspection points are not allowed to enter Sweden. People working in the food production chain, found to be *Salmonella* infected, will be allocated to another working task, where they do not handle food stuffs, or if that is not possible, suspended from work during the infection. The Swedish Food Act states that "persons who have, or can be assumed to have, a disease or infection, wound or other injury that can render food unfit for human consumption may not be engaged in food handling."

Continuous surveillance for *Salmonella* on carcasses of pigs and cattle is performed at slaughter. Annually, approximately 9000 samples are collected; 3000 from fattening pigs, 3000 from adult pigs and 3000 from cattle carcasses. Directly after slaughter, an area of 1400cm^2 is swabbed at places where risk of faecal contamination is considered greatest. As seen in Figures 12–14 the finding of *Salmonella* is exceptional.

Figure 12. Number of swab samples and % positive samples collected from adult pigs at slaughter 1996–1999.

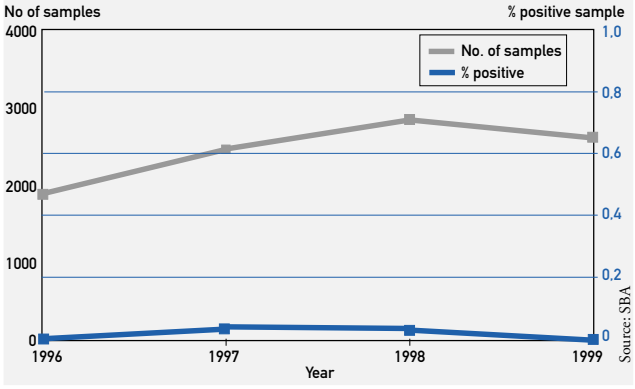


Figure 13. Number of swab samples and % positive samples collected from fattening pigs at slaughter 1996–1999.

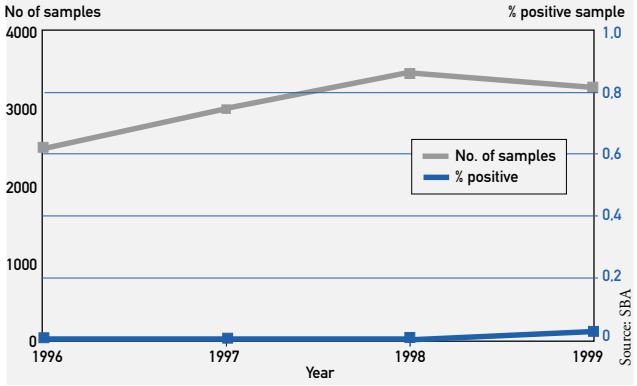
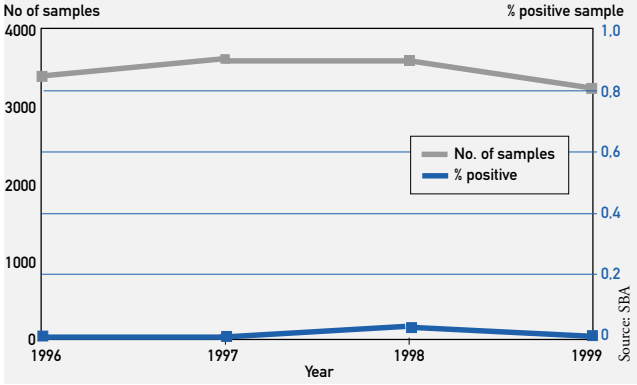
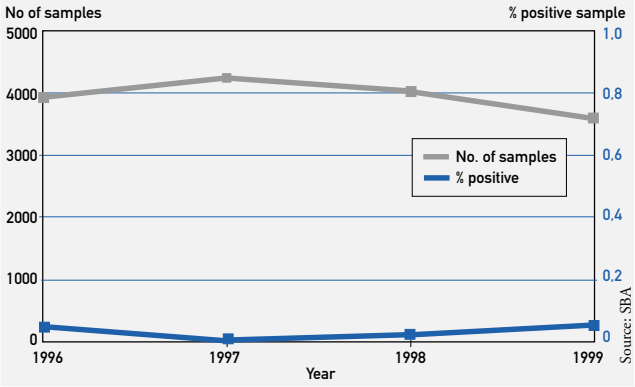


Figure 14. Number of swab samples and % positive samples collected from cattle at slaughter 1996–1999.



Surveillance of poultry is also performed at all slaughterhouses. Annually about 3–4000 samples from neck skins are collected and analysed for *Salmonella*. As seen in Figure 15, findings of *Salmonella* are very rare.

Figure 15. Number of neck skin samples and % positive samples collected from poultry at slaughter 1996–1999.



Surveillance of food of animal origin is also performed at cutting plants and at slaughterhouses. Since 1995, about 6000 samples (about 4–5000 from beef/pork and more than 1000 from poultry) are collected annually from cutting plants supervised by NFA. In addition, about 2000 samples are collected at cutting plants handling beef, pork and poultry, supervised by local municipalities. Frequency of sampling is correlated to the capacity of the establishment. Depending on the production capacity, sampling is performed daily, weekly, monthly or twice annually. Samples consist of crushed meat, trimmings etc. As seen in Figures 16 and 17 the finding of *Salmonella* is exceptional.

Figure 16. Number of samples (meat scrapings) and % positive samples at cutting plants handling pork and beef, supervised by NFA 1996–1999.

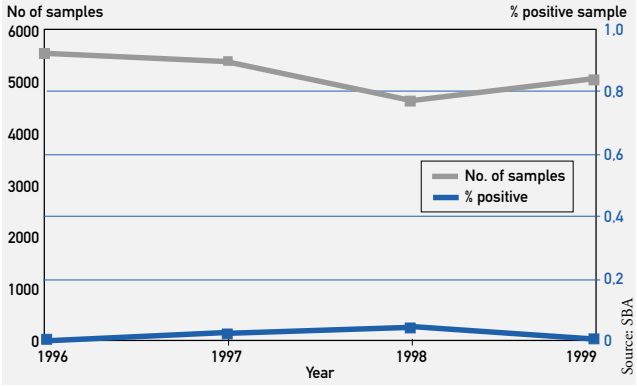
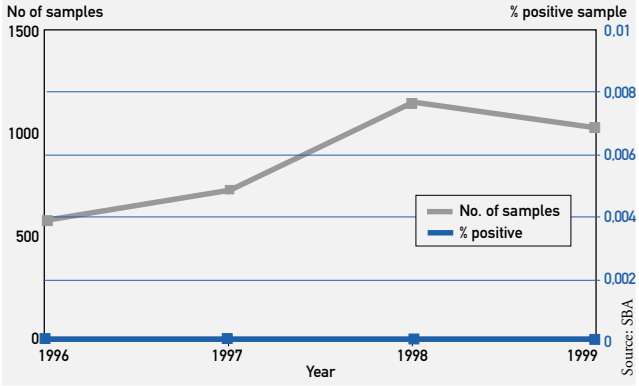


Figure 17. Number of samples (meat scrapings) and % positive samples at cutting plants handling poultry, supervised by NFA 1996–1999.



Swedish municipalities continuously perform official controls of various food items for various pathogens. Altogether 196 (of 289 municipalities) reported that a total of 54 459 *Salmonella* analyses were performed in 1995–1999. Of these 93 (0.17%) were positive. As imported food is also included in this sampling, the figure represents the *Salmonella* situation in food in Sweden and may not entirely reflect food of Swedish origin. Minor evaluations performed by some municipalities showed that the positive samples more often are obtained from imported food items than from food of domestic origin.

Campylobacteriosis

Introduction

Since the mid 1970s, medical interest in human *Campylobacter* infections has increased. Today *Campylobacter* is considered one of the principal bacterial agents causing enteritis and diarrhoea in man. In Sweden, *Campylobacter* infection is the major cause of bacterial enteric disease in humans. The genus *Campylobacter* contains several species of importance for both public and animal health. From a zoonotic point of view, *Campylobacter jejuni* and *C. coli* are the most important but also other species may cause disease in humans. Birds and mammals, both wild and domesticated, are the principal reservoirs for *Campylobacter*. The bacterium is excreted in faeces from infected animals/humans.

Most human cases are sporadic. The source of infection is usually faecally contaminated food or water. *Campylobacter* is often found in natural water sources where it can survive for weeks or months at low temperatures. Humans can also become infected by direct contact with infected animals. Transmission from person to person may also take place. The bacterium will usually not multiply in food as it is micro-aerophilic and requires temperatures above room temperature for growth.

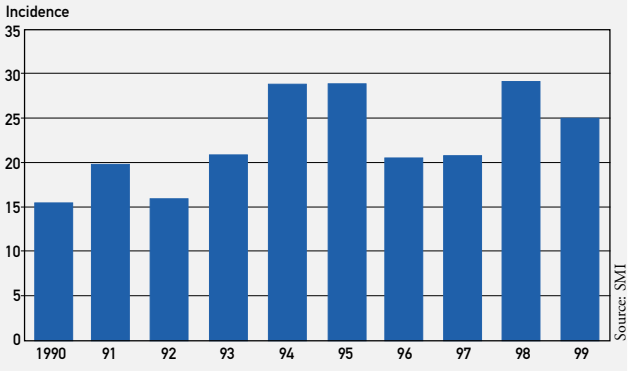
Much is still unknown concerning the epidemiology of campylobacteriosis. Subtyping by molecular methods can be used for identification of sources of infection and routes of transmission. There is, however, no simple standard method for such work today.

Campylobacteriosis in humans

Campylobacteriosis is an acute enteric disease of variable severity, characterised by diarrhoea, fever, malaise, abdominal pain and more seldom vomiting. In addition, headache, muscle and joint pain may occur. The patient usually recovers spontaneously within 10 days. A small percent may develop complications such as reactive arthritis and autoimmune syndroms.

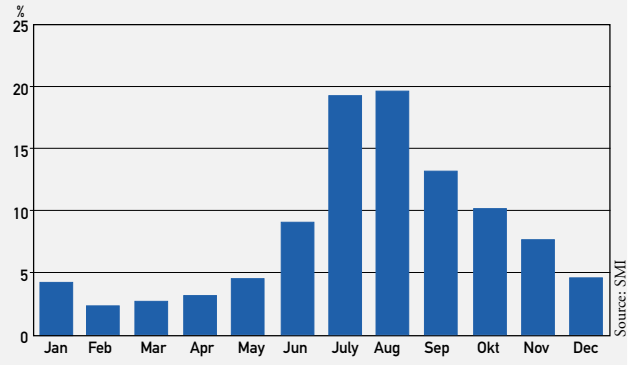
Human infection with *Campylobacter* became notifiable in Sweden in 1989. The number of infected cases is probably much higher than the number of reported cases. In Sweden, it has been estimated that only about 10% of all cases are diagnosed and thereby notified. Since 1990, on average 22.5 domestic cases per 100 000 inhabitants have been notified annually. During 1994–1995 and 1998–1999 an increased incidence (both domestic and imported cases) was observed (Figure 18).

Figure 18. Incidence (cases per 100 000 inhabitants) of domestic human *Campylobacter* cases 1990–1999 (n=19759).



The reasons for these increases are not known. A seasonal variation, with increased number of cases during summer (July to September) is observed (Figure 19).

Figure 19. Distribution of domestic human *Campylobacter* cases per month 1997–1999 (n= 6676).

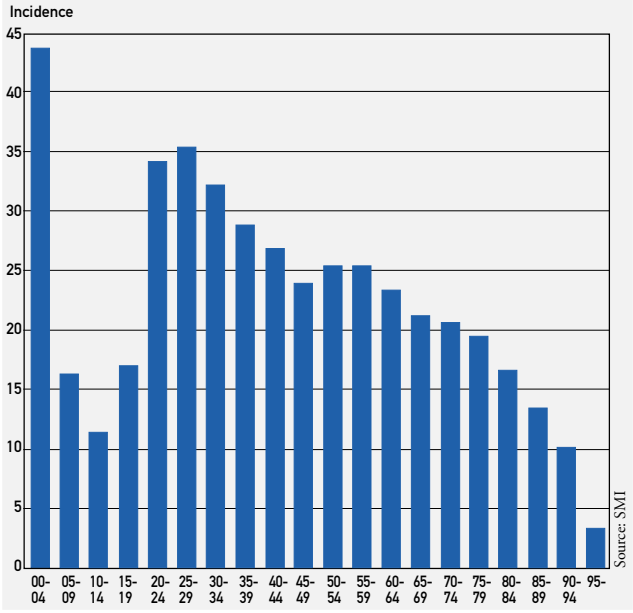


This increase may be due to changes in eating habits (more barbeques), more out-door activities, including drinking of surface water. As a similar

increase is observed in broilers it might also reflect that occurrence of *Campylobacter* in the environment probably is more common in the summer than in winter. A variation is also observed between counties. During 1997–1999 the incidence varied between 15–46 cases per 100 000, with most cases reported from the counties of Gotland, Blekinge and Skåne. If this reflects a real difference or is due to other factors such as frequency of testing is unknown. The incidence also varies with age, the highest incidence beeing seen in age groups 0–4 and 20–30 years (Figure 20).

Since 1990 between 0 and 12 *Campylobacter* outbreaks have been identified annually. Unpasteurised milk and under-cooked chicken are the most common sources of reported foodborne outbreaks. Waterborne outbreaks due to contaminated private wells, use of untreated surface water as drinking water or contaminated community water supplies also occur. Although the number of waterborne outbreaks are fewer compared with foodborne outbreaks, the number of persons involved in waterborne outbreaks may be 10 or 100 times more (Table 15).

Figure 20. Average incidence (cases per 100 000 inhabitants) of domestic human *Campylobacter* cases per age group 1997–1999 (n= 6676).



Campylobacter in animals

Infection with *Campylobacter* (of significance to human health) is not notifiable in animals. Birds and mammals constitute a large reservoir of *Campylobacter*, but it is often difficult to correlate this pathogen to diarrhoeic disease in animals since there is a high carrier rate in clinically healthy animals. In calves and puppies, infection may cause diarrhoea. Reptiles are rarely infected with *Campylobacter*. Findings of *Campylobacter* among wild and domesticated birds are common.

In Swedish domesticated birds (commercial broilers) the prevalence varies between farms and some farms seem to be totally free. More than 50% of farms are free from *Campylobacter* all year round and the majority of these farms have been free for several years.

Compared to the situation in many other countries, the prevalence of *Campylobacter* in broiler is low. A voluntary control programme has been running since 1991, and the prevalence of *Campylobacter*-infected flocks has decreased to below 10% (Figure 21).

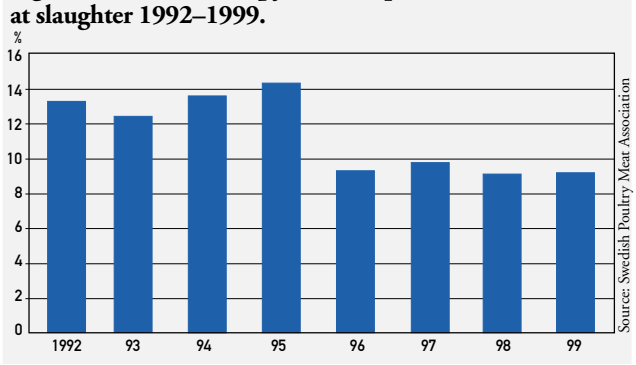
Table 15. Annual number of outbreaks and number of persons involved in outbreaks due to foodborne or waterborne infections in 1990–1999.

	FOODBORNE OUTBREAKS		WATERBORNE OUTBREAKS	
Year	Number of outbreaks	Number of cases in outbreaks*	Number of outbreaks	Number of cases in outbreaks*
1990	1	11	0	0
1991	1	11	1	600
1992	0	0	0	0
1993	3	39	1	2
1994	4	36	2	2900
1995	5	19	2	3011
1996	6	143	1	2
1997	10	96	2	19
1998	6	27	1	7
1999	4	15	0	0

Source: SMI

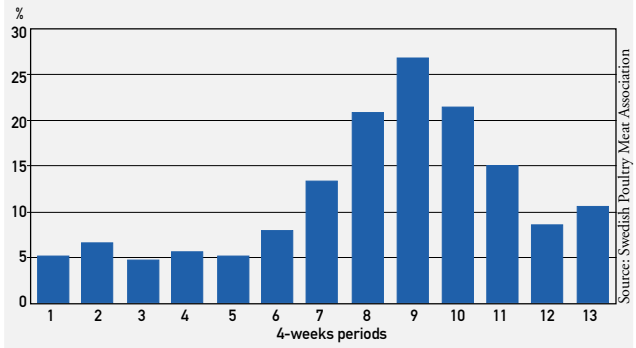
* The majority of cases in the large outbreaks are only epidemiologically verified and thereby not included in the number of notified cases.

Figure 21. Percent *Campylobacter* positive broiler flocks at slaughter 1992–1999.



All flocks are sampled at slaughter and the results are given to the farmer as a feed-back of the hygiene and prevention routines. As in humans, seasonal variation occurs with the highest prevalence occurring during late summer (Figure 22).

Figure 22. Average percent *Campylobacter* positive broiler flocks per 4-week periods at slaughter 1992–1999.



Investigations performed on Swedish wild and domesticated animals indicate that infection with *Campylobacter* is not uncommon, especially in wild birds. A recent survey showed that 15% of Canada geese and 22% of gulls were excreting *Campylobacter* in faeces.

Campylobacter in food

There is no official surveillance system of *Campylobacter* in food. Monitoring is achieved by various projects initiated by municipalities, the NFA, Swedish Meats R&D and other research institutes. If *Campylobacter* is found in heat-treated meat products they are deemed unfit for human consumption. Should an outbreak occur, the NFA decides on a case-by-case basis what action to take.

Current investigations on different types of meat indicate that approximately 13% of broiler, 1% of lamb, 0.5% of pork and less than 0.1% of beef is contaminated with *Campylobacter*. This is a favourable situation compared with many other countries, where for example 40 to 50% of broilers may be contaminated.

Verotoxigenic *Escherichia coli* O157

Introduction

In 1983, an epidemiological investigation revealed a link between two outbreaks of haemorrhagic colitis and consumption of under-cooked hamburgers from a fast-food outlet in the USA. Microbiological examinations identified a verotoxigenic *Escherichia coli* strain of the ”rare” O157 serotype. The same type of bacterium was isolated in patients as well as in meat samples from the batch that was originally implicated in the outbreaks. Since then many outbreaks associated with verotoxigenic *E. coli* (VTEC) O157 have been reported and VTEC-infection has become widely recognized as a major zoonotic infection in humans.

The term enterohaemorrhagic *E. coli* (EHEC) is used interchangeably with VTEC and shiga toxin producing *E. coli* (STEC). In this report, VTEC will be used. Although over 150 different serotypes of VTEC have been associated with human illness, the vast majority of outbreaks among humans have been associated with serotype O157. Other common serotypes causing disease in humans are serotypes O26, O103, O111 and O145. VTEC O157 does

not seem to cause disease in animals. As most research has focused on O157, less is known about other serotypes. The present report will focus on serotype O157.

VTEC O157 appears to have ruminants as reservoir, but it has also been isolated from many other species, e.g. pigs, dogs, cats, horses, sea-gulls and geese. The bacterium is excreted in faeces from infected animals and humans. Four routes of infection for humans have been identified; faecally contaminated food or water (under-cooked meat, unpasteurised milk, contaminated drinking water), person-to-person transmission, transmission through the environment (swimming in a faecally contaminated lake or swimming pool) and direct contact with infected farm animals. The bacterium can survive for long periods on straw, wood surfaces and in water. In food, O157 strains are killed by ordinary cooking and frying. The pasteurisation of milk will also kill the bacteria. VTEC O157 is acid tolerant, therefore foods preserved by acidification, e.g. salami, fermented meat sausages and fresh apple cider, must be considered as putative vehicles for the bacterium.

As the epidemiology of VTEC O157 is not fully known it is unclear how and where in the production chain (from farm to table) that relevant measures can be instituted to prevent transmission to humans.

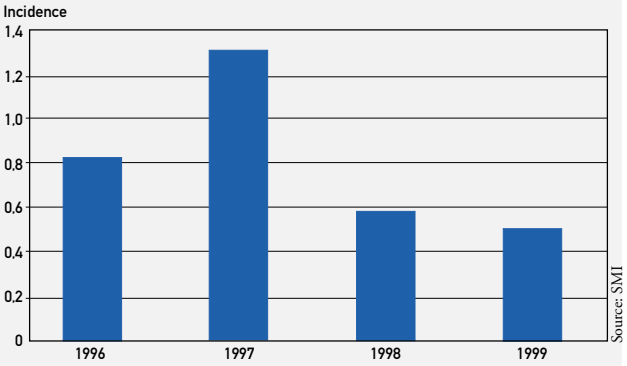
VTEC O157 in humans

The clinical signs associated with infection with VTEC O157 may vary from symptom-free infections to diarrhoea and haemorrhagic enteritis. More seldom, haemolytic uremic syndrome (HUS) may occur. Most patients recover within a week. Especially among children, but also among elderly, the disease may progress to HUS where a few percent die and survivors may develop permanent renal failure. After recovery, VTEC O157 may be excreted in faeces for several weeks. The number of bacteria that will cause infection in humans is very low compared with for example, *Salmonella* and *Campylobacter*. The infectious dose may be as low as 10 bacteria. Thus, the bacteria do not have to multiply on the food item to cause infection, as the original contamination may be sufficient.

Findings of VTEC O157 in humans are notifiable since January 1996. But the occurrence of HUS and infections due to serotypes other than O157 are not notifiable and therefore less well known. The first known Swedish case of human infection with VTEC (due to serotype O121) occurred in 1988 and the first case due to serotype O157 occurred in 1989. Both cases were diagnosed retrospectively. Investigations indicate that approximately 50% of VTEC infections are due to serotype O157. The second most common serotype being O121.

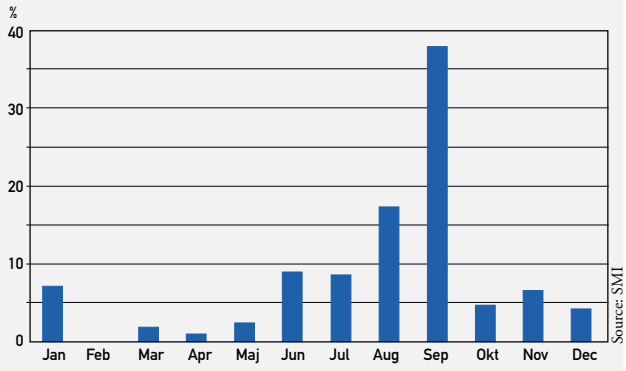
During 1996–1999, a total of 387 human infections with VTEC O157 have been notified, 286 (74%) of these were infected in Sweden. The annual domestic incidence has varied between 0.5 and 1.2 cases per 100 000 inhabitants (Figure 23).

Figure 23. Annual incidence (cases per 100 000 inhabitants) of domestic human cases of VTEC O157 1996–1999 (n=286).



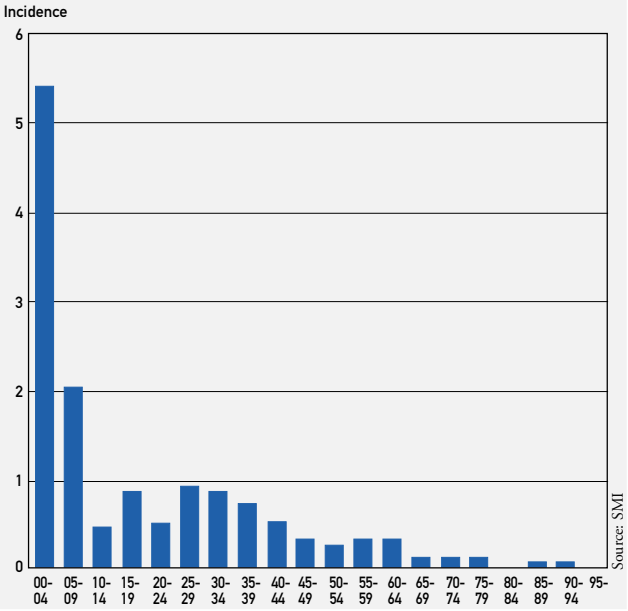
During the autumn and winter of 1995/1996 the first reported outbreak due to VTEC O157 occurred in Sweden, involving about 120 persons. This increased the awareness and today people, especially children, with bloody diarrhoea will probably be examined for the presence of VTEC. In the summer of 1997 an increased number of cases was reported from the west coast of Sweden (the counties of Halland och Västra Götaland). Epidemiological investigations revealed several possible sources of infection; bathing in a small lake, person-to-person spread and direct or indirect contact with cattle. Most domestic cases are reported during late summer (Figure 24).

Figure 24. Average distribution (%) of domestic humans cases of infection with VTEC O157 per month 1997–1999 (n=214).



This may reflect more environmental contact with the bacterium, more direct or indirect contact with infected cattle and perhaps also increased consumption of under-cooked meat (barbeque) and unpasteurised milk. As reported from other countries, infection with VTEC O157 is also more frequent among children (Figure 25).

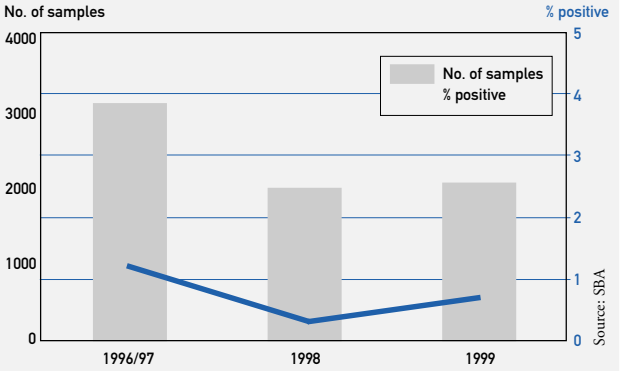
Figure 25. Average incidence (cases per 100 000 inhabitants) of domestic human cases of infection with VTEC O157 per age group 1997–1999 (n=214).



VTEC O157 in animals

Findings of VTEC O157 in animals became notifiable in October 1996. In 1999, notification was restricted to findings of serotype O157 that had a connection with a human clinical infection with VTEC O157. VTEC O157 was first isolated in Swedish cattle in 1996. The same year, infection with VTEC O157 in humans in Sweden was, for the first time, traced back to a VTEC O157-infected cattle herd. Surveillance was initiated to establish the prevalence of the pathogen in Swedish livestock. Since 1996/97 approximately 2000 faecal samples have been collected annually from normally slaughtered cattle. The annual prevalence has varied between 0.4 and 1.2% (Figure 26).

Figure 26. Number examined and percent VTEC O157 positive faecal samples collected from cattle at slaughter 1996–99.



1996/97: Samples consist of 10 gram faeces, 1998 and 1999 samples consist of 1 gram faeces

Young cattle (< one year) were more often infected than older cattle and dairy cattle seemed to be infected more often than cattle of beef breeds. Similar studies have shown that 0.8% of lambs, 0.9% of sheep and 0.1% of pigs were infected with VTEC O157. In all herds where VTEC O157 infected pigs were found, a ruminant contact could be established. In 1998/99 a study based on sampling in herds showed that approximately 10% (23 of 249) of dairy herds were infected with VTEC O157 and the individual prevalence of calves and heifers was 1–2%. It also showed that the prevalence was lower in northern parts of Sweden. Surveys have also been performed in wild life (125 hares, 90 deer, 68 wild boars, 84 moose, 195 roe deer, 105 geese and 111 gulls). VTEC O157 was only found in faeces from one wild boar. It can be concluded that cattle is the major reservoir for VTEC O157 in Sweden and that no major changes in prevalence seem to have occurred since 1996. Other livestock may also harbour the bacterium, but so far there is no indication that wild life

plays a significant role in the epidemiology of VTEC O157. The occurrence of other serotypes is not known.

As VTEC O157 is not uncommon among Swedish livestock, especially cattle, general prophylactic recommendations aiming at reducing the risk of direct transmission to humans have been issued and updated annually by competent authorities in cooperation with the industry since 1998 (see box). In addition, all notified herds will receive "guidelines for handling of infected herds" including hygiene recommendations, sampling at slaughter and sampling of live animals before sale.

Recommendations for herds with ruminants during summer 1999

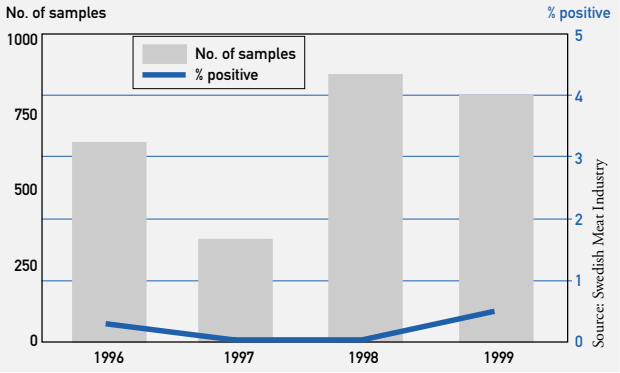
- Groups of children under 5 years should not visit buildings where ruminants are housed.
- Other visitors shall exercise good hygiene, including hand-wash after direct or indirect contact with animals.
- Unpasteurised milk or products thereof shall not be offered to anybody.
- Animals sent for slaughter shall be clean.
- Animals shall not be housed or their manure handled in such a way that would promote spread of infection; avoid spreading of manure on growing vegetables and berries or near shores/lakes where people go swimming; separate grazing animals from bathers.
- General hygiene recommendations, including annual cleaning and disinfection of cowhouses.

VTEC O157 in food

Apart from the voluntary monitoring of cattle carcasses performed on the initiative of, and by, the meat industry, there is no specific routine surveillance or monitoring system for VTEC O157 in food. If VTEC O157 is found in food the NFA will, on a case-by-case basis, take the action necessary to ensure that contaminated food will not reach the consumer.

Since January 1998, approximately 900 cattle carcasses are being examined annually for VTEC O157 on a voluntary basis. Similar investigations were also performed in 1996 and 1997. Results indicate that between 0 and 0.5% of carcasses may be contaminated (Figure 27).

Figure 27. Number examined and percent VTEC O 157 positive cattle carcasses, examined at slaughter, 1996–99.



All cattle and sheep originating from infected herds are examined at slaughter on a voluntary basis.

In connection with the first outbreak of human infections with VTEC O157, the NFA initiated investigations in different foods. Imported meat (482 samples), Swedish meat (543 samples), fermented products (60 samples), cheese (14 samples) and 600 samples from fresh vegetables were examined for VTEC O157. All were negative except one positive sample from imported meat.

Yersiniosis

Introduction

The genus *Yersinia* includes three pathogenic species. Of these, *Y. enterocolitica* and to a lesser extent *Y. pseudotuberculosis* cause yersiniosis in humans. This report will focus on *Y. enterocolitica*. More than 50 serotypes of *Y. enterocolitica* have been identified and certain serotypes appear to be more frequent in certain geographical areas. Yersiniosis in humans is usually caused by *Y. enterocolitica* (serotype O3, O5/27, O8 and O9) and less often by *Y. pseudotuberculosis*.

Before the 1970s, very little attention was paid to this bacterium. The interest increased in the 1980s when cases of diarrhoea with appendicitis-like syndromes, caused by Yersinia, were reported mainly from Scandinavia but also from other European countries, the USA and Canada.

The major sources of infection are considered to be ingestion of contaminated food, for example pork and unpasteurised milk, or water. Direct contact with animals or man is less common. The ability of the organism to grow at 4°C means that multiplication can occur at low temperatures, for example in cold-stored food with long shelf life.

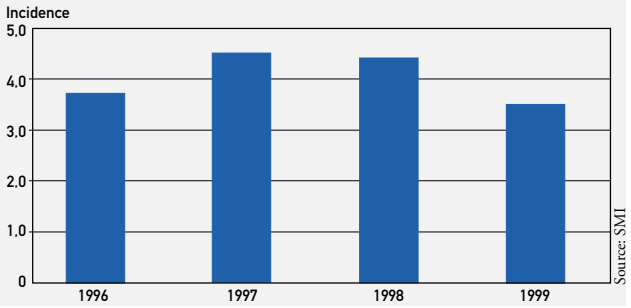
Effective control measures to minimise *Y. enterocolitica* infections in humans are still missing. However, improved slaughter techniques may reduce contamination at slaughter; avoidance of long refrigeration periods and adequate cooking procedures are other ways of reducing occurrence of *Y. enterocolitica*.

Yersiniosis in humans

After an incubation period of 4 to 7 days, clinical signs such as fever, diarrhoea and abdominal pain, similar to those of appendicitis, may occur. Clinical signs may last up to 3 weeks. Data from Scandinavian patients have shown that reactive arthritis develops in approximately 10% of clinical cases. However, most of these patients recover within a year. Skin lesions (erytema nodosum) may also occur. Septicemia is less common and usually occurs in patients with other underlying diseases.

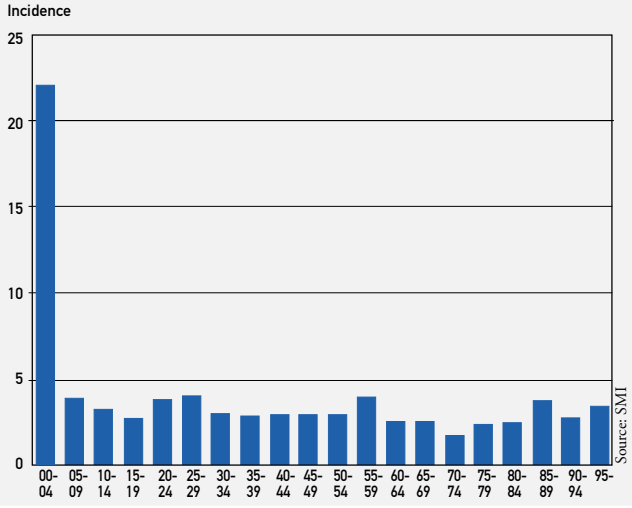
In Sweden, infection with *Yersinia* in humans is notifiable since 1996. The annual incidence of domestic cases has been approximately 4 (3.5–4.5) cases per 100 000 inhabitants (Figure 28).

Figure 28. Incidence (cases per 100 000 inhabitants) of domestic human cases of *Yersinia* 1996–1999.



Approximately 70% of all reported cases are of domestic origin. Children under five years of age are most often affected. (Figure 29).

Figure 29. Average incidence (cases per 100 000 inhabitants) of domestic human cases of *Yersinia* per age group 1997–1999 (n=1099).



In Sweden, human infections are usually caused by serotype 3. In 1997–1999 approximately 80% of cases were due to this serotype, 2 % were due to serotype 9 and in 18% the seroptype was not specified. Less than one case annually was due to *Y. pseudotuberculosis*.

Most cases are sporadic and the source of infection usually remains unknown. Major outbreaks are very rare; altogether five outbreaks have been reported since 1980. The latest, in 1994, was probably due to a ready-to-eat pork product, brawn. It involved 13 persons. In 1988, a larger outbreak including 61 persons was reported. Contaminated milk, due to deficiencies in a dairy, was the probable source of infection. In the remaining three outbreaks the source of infection was not identified.

It has been observed that eating habits of people in Sweden (people avoiding pork products) will affect the incidence of yersiniosis. A decrease in the number of human cases (based on voluntary reporting) was observed in 1994–1995, possibly due to changes in slaughter techniques improving hygiene at slaughter of pigs. It is, therefore, probable that pork is a major source of *Y. enterocolitica* infection in Sweden.

Y. enterocolitica in animals

A variety of domestic and wild animals are reservoir hosts for *Y. enterocolitica*, such as pigs, rodents, sheep, goat, cattle, horses, dogs and cats. The bacterium is passed in faeces, thereby allowing faecal-oral transmission among animals. In pigs it has been shown that the bacterium persists longer in tonsils than in faeces which is of importance when reducing contamination at slaughter. Generally, animals are considered to be subclinical carriers of *Y. enterocolitica*. In dogs and sheep, illness due to *Yersinia* has been reported. It is probable that stress or immunosuppression could promote disease.

Tularemia

Introduction

In Europe and Russia, existence of human disease outbreaks, associated with times of rodent abundace, has long been recognised. In 1532, it was described how lemmings crowded together and died of epidemic disease and then caused disease among humans. Tularemia is caused by infection with the bacterium, *Francisella tularensis*, a disease where the reservoir is found among wildlife. Two different strains of the bacterium have been identified, a more virulent type A strain occurring naturally in North America and a type B strain that occurs throughout the Northern hemisphere. The epidemiology of human tularemia is complex and varies with subspecies and virulence, ecosystem and geographical region. The disease is mainly observed among wild rodents, but cases may also occur in other mammals and birds.

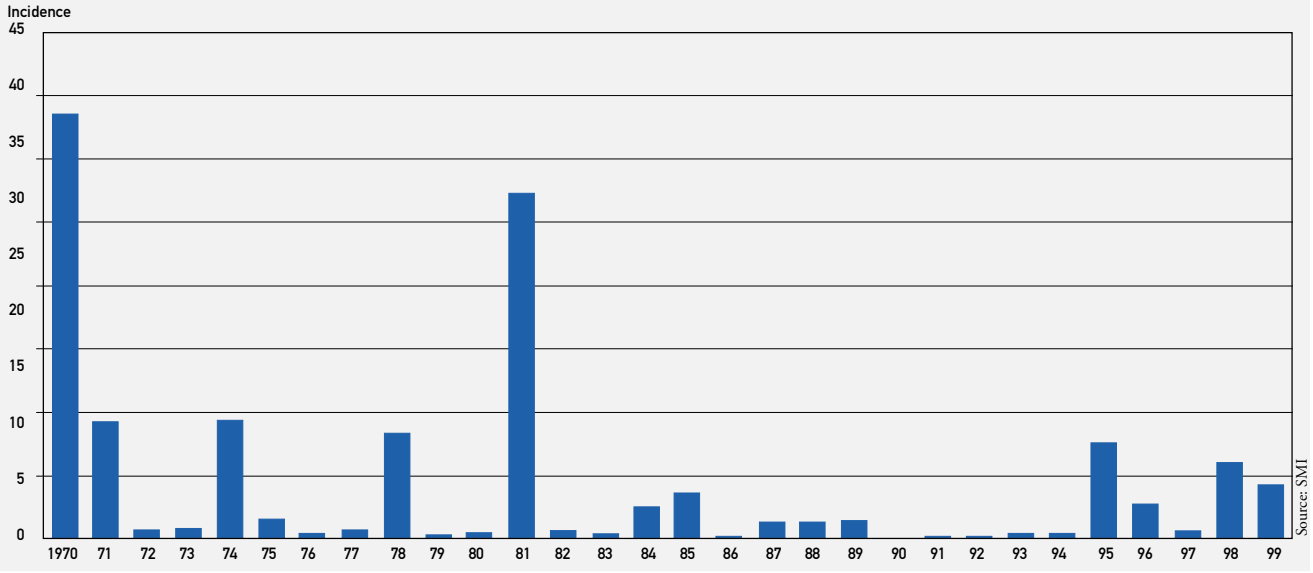
The disease in Sweden is most often transmitted by mosquitoes, but transmission through direct contact with infected animals, by bites from other vectors such as ticks and tabanides, by inhalation of dust contaminated with urine or faeces from diseased animals, or by drinking contaminated water also occurs. Laboratory-acquired infections have also been reported. Human-to-human transmission is not considered to occur.

Tularemia in humans

The clinical manifestation is related to the route of infection. An indolent ulcer, accompanied by swelling of the regional lymph node is the most common clinical symptom. Inhalation of the bacteria may cause pneumonia. The general symptoms may include fever, headache and nausea.

Tularemia is a notifiable disease in humans. Since 1970, between 0 and 538 cases have been reported annually in Sweden (Figure 30). The incidence of disease varies between years. The largest outbreak that has been recorded in Sweden occurred in 1966/67 in Jämtland county, where more than 2700 people were reported infected. The latest outbreaks occurred in 1970 and 1982, respectively, when 450 and 538 cases were reported. The majority of human and animal cases occur in northern parts of

Figure 30. Incidence (cases per 100 000 inhabitants) of tularemia in humans (n=2076) during 1970–1999.



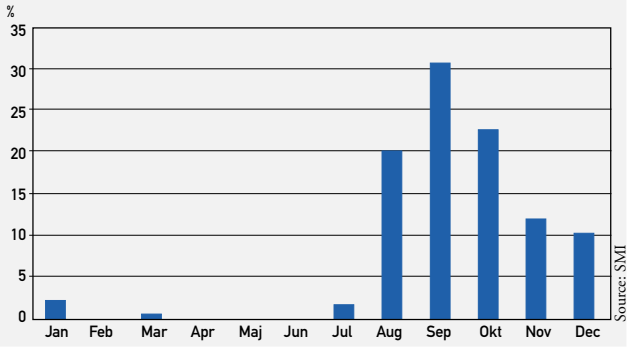
Sweden and during late summer/autumn, reflecting the transmission of disease by mosquitoes (Figure 31).

Tularemia in animals

Severity of disease varies and susceptible species may be divided into three groups. Many species of rodents and lagomorphs belong to the group with most susceptible animals and generally suffer a fatal septicemic disease. Other mammals and birds are less susceptible and rarely suffer fatal disease. Carnivores like dogs, cats and mink may become infected, but this is very unusual with the type B strain of *Francisella tularensis.*

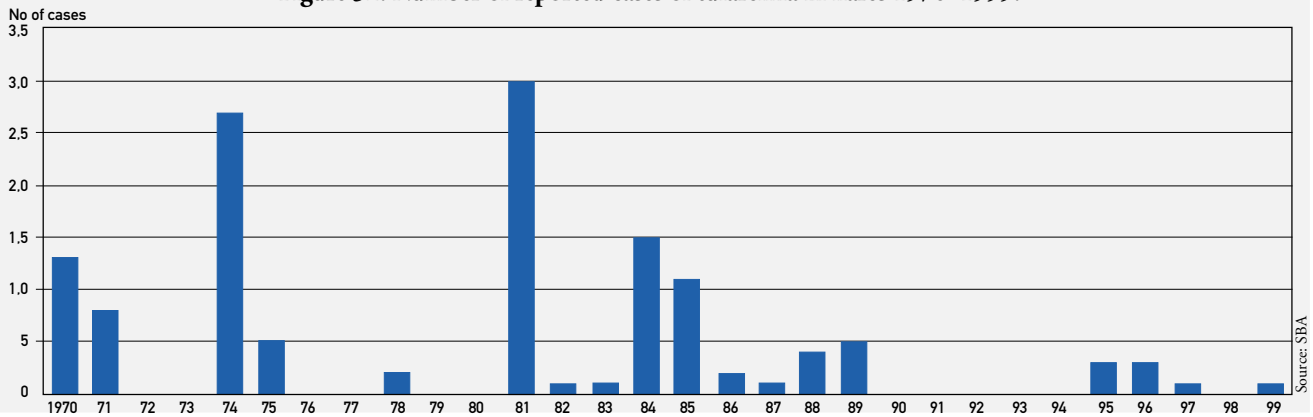
In Sweden, tularemia is mainly observed in mountain hares, but cases have been reported in other species such as muskrat, lemming, squirrel, voles, mice, raven, rough-legged buzzard and ural owl. Although knowledge of reservoirs is incomplete, rodents such as voles and possibly also beavers are considered the most important reservoirs in Sweden.

Figure 31. Average distribution (%) of humans cases of tularemia in per month 1997–1999 (n= 185).



Tularemia in animals is a notifiable disease. As in humans, the number of reported cases of tularemia varies greatly between years (Figure 32). Outbreaks occur mainly among mountain hares.

Figure 32. Number of reported cases of tularemia in hares 1970–1999.



Bovine tuberculosis

Introduction

Bovine tuberculosis is caused by a bacterium, *Mycobacterium bovis*. *M. bovis* has one of the widest host ranges that has been reported for a pathogen. Cattle are the most important reservoir but during the 1970s new reservoir species such as badgers, brush-tailed possum and farmed deer, have been identified.

The disease is common in developing countries. In most industrialised countries the disease is under control but some countries still have substantial problems with *M. bovis* in food-producing animals, mainly cattle.

As one of the first countries in the world, Sweden declared itself free from *M. bovis* in 1958 and is declared officially free from tuberculosis in bovine herds according EU-legislation.

The control programme in cattle in Sweden was based on routine tuberculin testing and meat inspection at slaughter. In 1970, the routine tuberculin testing was abolished and today the surveillance is mainly based on meat inspection at slaughter.

Tuberculosis in humans

Infection with *M. bovis* in humans causes symptoms similar to those in classical human tuberculosis (caused by *M. tuberculosis*). The incubation period may be 1–2 months or clinical symptoms may not develop until years or decades after the infection. Clinical signs depend on the location of the infection. As the lungs are most commonly affected, cough is the most common symptom. Man contracts the infection primarily by ingestion of unpasteurised contaminated milk or products thereof, secondarily by aerosol transmission from infected animals. Human to human transmission is possible. Meat consumption is not considered as a relevant source of infection. Ordinary cooking procedures and pasteurisation kill the bacterium. Control of bovine tuberculosis in humans is based on the control in animals, including pasteurisation of milk in combination with treatment of infected people.

In Sweden, during the 1990s the number of reported cases of tuberculosis due to *M. bovis* was, on average, 7 per year. Cases usually occur in elderly people, infected in their youth before bovine tuberculosis was eradicated in Sweden, or in immigrants coming from areas where bovine tuberculosis is still common.

Tuberculosis in animals

Tuberculosis is a chronic disease in animals, the time between infection and development of clinical signs may be very long, from a few weeks to many years. The susceptibility varies between species. In cattle, tuberculous lesions are usually found in the lungs, the lymph nodes of the lungs and in the head region. Tuberculous mastitis, which is of great importance in the epidemiology of the disease, may be found in less than 1 % of cases. The disease is transmitted by respiratory or gastrointestinal route and also by unpasteurized milk. As the survival of the bacterium in the environment may be long (weeks to months), a contaminated environment may also act as a source of infection.

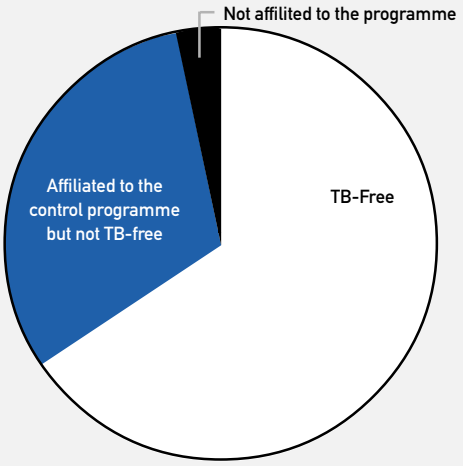
In Sweden finding or suspicion of *M.bovis* in any species is notifiable. According to EU-legislation, Sweden is free from bovine tuberculosis. The last case in cattle was reported in 1978. In dogs, no cases have been reported for more than 20 years. In 1990, one infected cat was reported. The source of infection was the infected owner. Except for two cases of *M. bovis* in moose in 1940, no cases have been reported in wild-life.

In 1991, *M. bovis* was reported for the first time in farmed deer in Sweden. An epidemiological investigation identified a common source of infection, a consignment of fallow deer imported in 1987. Tracing revealed an additional 12 infected herds of farmed deer, the last in 1997. All herds

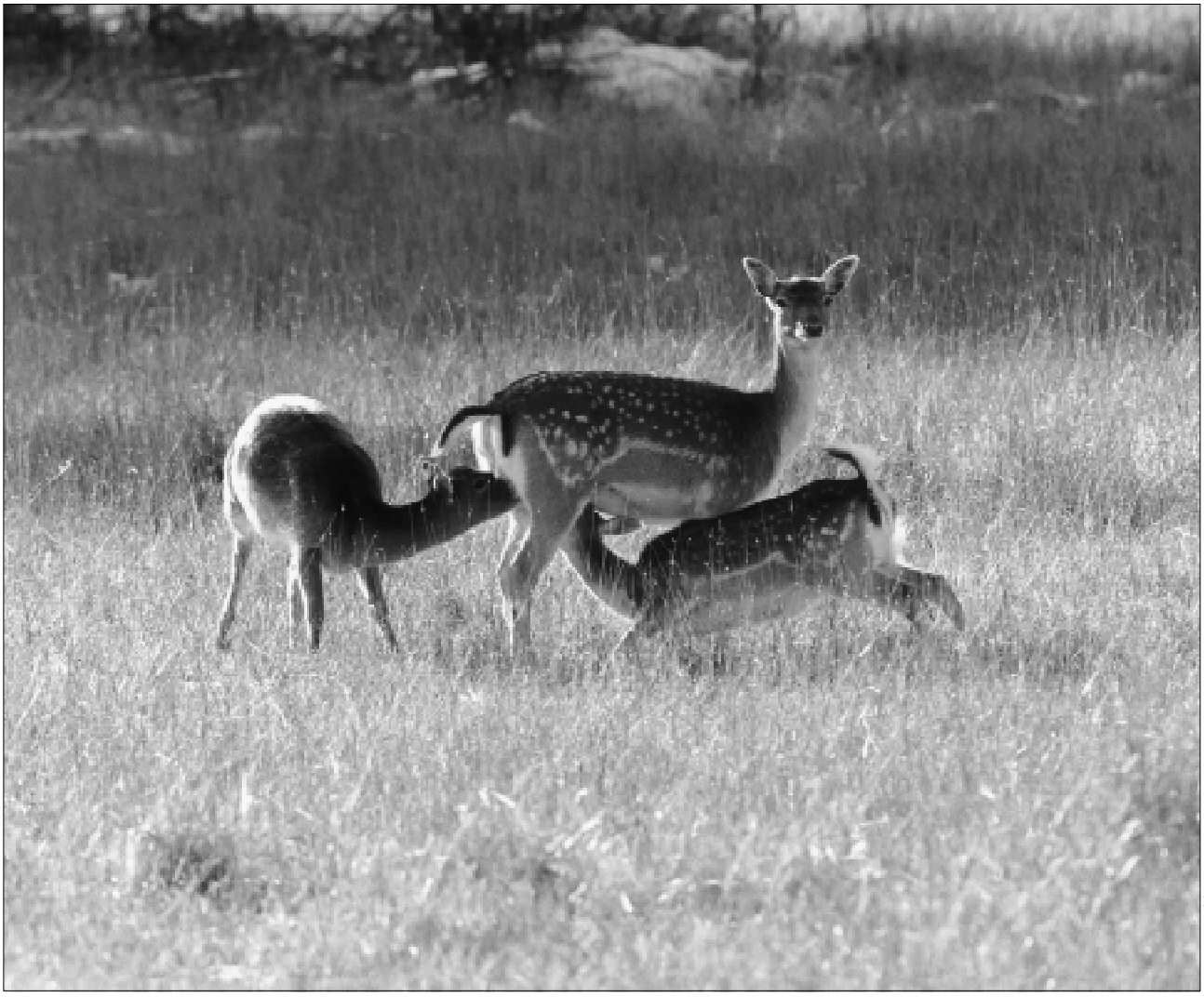
have been depopulated. The compulsory meat inspection for farmed deer was extended in 1994 to include free-living fallow and red deer. A voluntary control programme based on repeated tuberculin testing was initiated in 1994 and all live animal movement was prohibited except from herds with tuberculosis-free status. In December 1999, 550 (96%) out of the 569 farmed deer herds were affiliated to the control programme. A total of 374 herds (66%) had obtained tuberculosis-free status, 89 as a result of at least three whole herd tuberculin tests, 254 by slaughter and meat inspection of the whole herd, and 31 new herds had been established with deer from tuberculosis-free herds (Figure 33).

Another 176 herds (32%) were affiliated to the control programme but had not obtained tuberculosis-free status. Of these herds, 23 had begun to tuberculin test their deer and 19 had begun to depopulate their herd. A total of 19 herds (3%) were not affiliated to the control programme.

Figure 33. Tuberculosis status of Swedish farmed deer herds in 1999 (n=569).



Source: Swedish Animal Health Service



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Brucellosis

Introduction

The genus *Brucella* includes five main species of which three (*B. abortus, B. suis* and *B. melitensis*) are of major zoonotic importance. The *Brucella* bacterium (*B. melitensis*) was first isolated in 1887 from a British soldier who had died in Malta fever (syn. Brucellosis, Bang's disease or Mediterranean fever). The etiology of the disease (due to *B. melitensis*) was discovered several years later when goats were identified as the source of human infection and that apparently healthy goats could excrete *B. melitensis* in milk. By avoiding consumption of unboiled goat milk the incidence of Malta fever decreased dramatically. The *Brucella species* that is the major cause of brucellosis in cattle, *B. abortus,* was discovered in Denmark in 1897.

In animals, *Brucella* may provoke abortion and genital excretion of the bacteria may contaminate the environment where they can survive for months. The bacterium may also be excreted in milk. Man is usually infected by contact with infected animals or indirectly by consumption of unpasteurised milk or products thereof. The bacterium is killed by pasteurisation.

Brucellosis in humans

The incubation period in man may vary from a few days to several weeks or months. The infection is characterised by fever that may be intermittent and of variable duration. Swetting, head ache, weakness and generalised aching may also be observed. If untreated, the disease may last for several months, occasionally for more than a year. Orchitis and affection of joints and the central nervous system can also occur. Antibiotic treatment will, however, provide cure in most patients.

Brucellosis in humans is not notifiable in Sweden. Available figures are based on voluntary reports from laboratories. During the last 10 years no domestic cases have been identified. Between 0–6 cases infected abroad, usually in the Mediterranean countries, have been reported each year.

Brucellosis in animals

The major symptoms of brucellosis in all animal species is abortion or premature expulsion of the foetus. *Brucella* can also be excreted in milk. In cattle, brucellosis is usually caused by *B. abortus*. In pigs, the most common cause is *B. suis* and in sheep and goat *B.melitensis* is the most common cause of brucellosis.

Infection with *Brucella* is notifiable in all animals on the basis of clinical suspicion. Sweden is considered free from brucellosis. The last case of bovine brucellosis in Sweden was reported in 1957. Brucellosis in other species has never been found. Sweden has been declared free from brucellosis in bovines, sheep and goats according to EU-legislation. Since Sweden joined the EU, serological surveys are performed annually in sheep and goats (approximately 9000 samples). Annual serological surveys are also performed in cattle (3000 bulk milk samples) and pigs (3000 blood samples). No positive samples have been found.

Chlamydiosis

Introduction

Human chlamydiosis, due to *Chlamydia psittaci* is a disease with world-wide distribution, mainly affecting people exposed to infected psittacines and to a lesser extent also other birds. It is caused by a bacterium, *C. psittaci,* an intracytoplasmatic obligate bacterium. The disease was first described in 1879 in patients who had been exposed to tropical pet birds and had developed an unusual type of pneumonia. The disease was named after the Greek word for parrot "psittakos". Psittacosis and ornithosis are used interchangeably with chlamydiosis. In the present report, chlamydiosis will be used.

Infected psittacine birds are the most common source of human infection. Wild birds and, especially in processing plants, ducks and turkeys, have also been associated with human disease. Infection is usually acquired by inhalation of dust contaminated with avian excreta or fomities. The bacteria survive well in the dust. Person-to-person transmission rarely occurs. Contact with sheep, especially lambing or aborting ewes, can also cause infection in humans. The strain isolated in sheep differs slightly from strains isolated from birds and has recently been re-named *Chlamydophila abortus*.

Chlamydiosis in humans

Infection with *C. psittaci* causes a human disease ranging from asymptomatic infection to severe pneumonia and death. In pregnant women, abortion may occur. Serological surveys indicate that subclinical infections are not uncommon. Sporadic cases occur where cage birds often are reported as the source of infection.

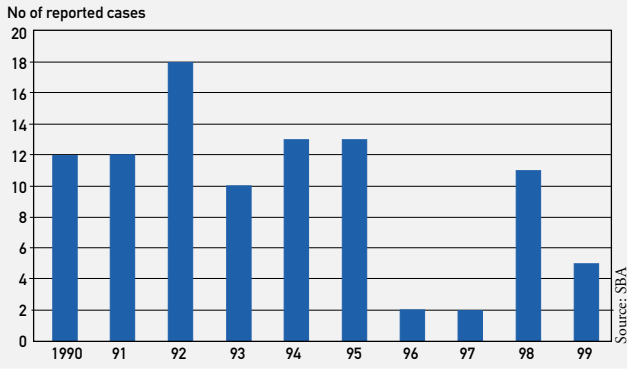
C. psittaci is notifiable in Sweden, however, number of reported cases does not reflect the true incidence since diagnostics for *C. psittaci* is seldom performed in cases of pneumonia.

Chlamydiosis in animals

Psittacine birds, pigeons and seabirds are common reservoirs for human infection. Within the poultry industry, turkeys, geese and ducks are also natural hosts. In birds, infection with *C. psittaci* is usually asymptomatic, but conjunctivitis and also generalised infection can occur. *C. psittaci* is also of economic importance in farm animals. In ruminants, infection can cause disease in the lungs, diarrhoea, abortion or arthritis. Many animals may harbour the bacterium without showing any clinical signs.

In Sweden, infection with *C. psittaci* is notifiable in birds. Since 1990 between 2 and 18 cases (infected epidemiological units) have been reported annually (Figure 34). However, the number of reported cases mainly reflects the frequency of sampling.

Figure 34. Number of reported cases (infected epidemiological units) of *Chlamydia psittaci* 1990–1999.



C. psittaci is not considered to occur in Swedish commercial layers and broilers. The bacterium has been isolated from cage birds, especially psittacines and to a lesser extent also among wild birds.
In game birds, sporadic cases of clinical disease due to infection with *C. psittaci* have been reported.
Enzootic abortion in sheep, caused by *Chlamydophilus abortus* has not been observed in Sweden.

Listeriosis

Introduction

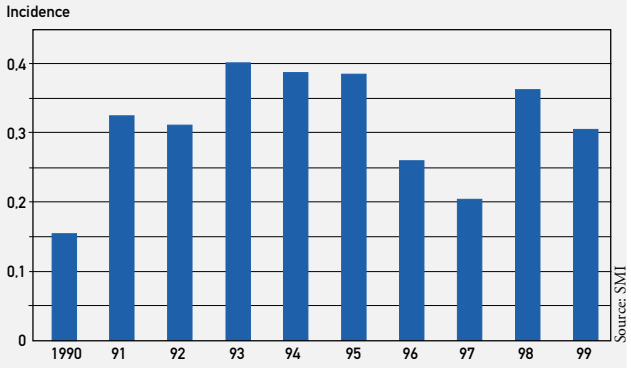
The bacterium *Listeria moncytogenes* was fully described in the early 1920s after an epizootic outbreak among rabbits and guinea pigs. The disease listeriosis had earlier been recognised in sheep, although the microorganism had not been identified. The first case of human listeriosis was reported in 1929 and the first perinatal case in 1936. The bacterium has been reported to cause disease in a wide range of wild and domesticated animals. It was not until the 1980s that the significance of food as a source of infection for human listeriosis was recognised. Although other modes of transmission occur, such as vertical (mother to child), contact with infected animals, nosocomial (hospital acquired) it is generally considered that food-borne transmission is the major route of infection for human listeriosis. *L. monocytogenes* is widely distributed in faecally contaminated environments and has been isolated from soil, vegetation, silage, sewage and water. The bacterium is resistant to environmental conditions which allows it to survive under adverse conditions and even grow under refrigerated conditions. The bacterium has often been isolated from food processing environments especially those that are cool and wet where it can cause post-processing contamination of food. New approaches of food preparation, for example food with long refrigerated shelf-life or raw or semi-raw meat or fish dishes may increase the risk of listeriosis in humans. Even though the contamination rate initially may be low, the ability of *Listeria* to grow during refrigerated storage means that the levels can increase. As ingestion of low levels of *Listeria* is probably not uncommon, there is an ongoing debate on the public health significance of ingestion of low levels of the bacterium for individuals other than the immunocompromised.

Listeriosis in humans

A high proportion (on average 2–10%) of healthy people are estimated to be carriers of the bacterium without any apparent adverse consequences. Clinical signs are usually seen in people at the extremes of age, during pregnancy or among immunocompromised individuals. The bacterium most often affects the pregnant uterus, the central nervous system or the blood stream; thereby causing abortion or severe disease in newborn babies, meningoencephalitis, or septicemia in adults. Infection with *Listeria* can also cause gastrointestinal symptoms. The infection can be treated with antibiotics, but between 20 and 40% of reported clinical cases are likely to be fatal. Most cases of clinical listeriosis appear to be sporadic although an unknown proportion of these may be due to an unrecognised common source of infection. Listeriosis is a relatively rare disease, on average 0.4–0.8 cases per 100 000 inhabitants are reported annually in North America and Western Europe.

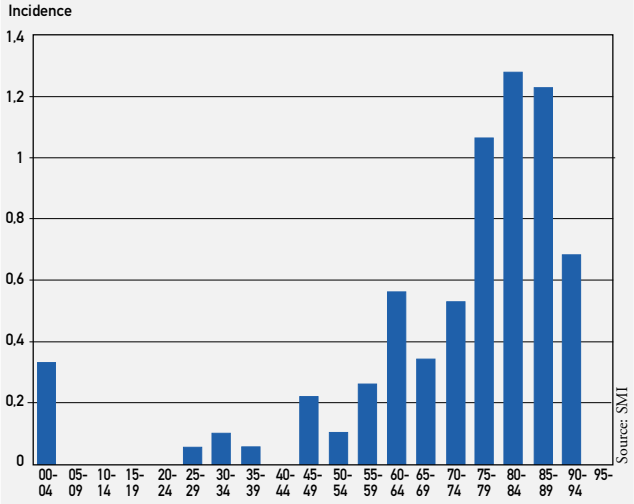
The situation is similar in Sweden, the average incidence during 1990–1999 being 0.3 (0.15–0.4) per 100 000 inhabitants (Figure 35).

Figure 35. Incidence (cases per 100 000 inhabitants) of listeriosis in humans 1990–1999 (n=271).



In Sweden, normally all cases are observed within the risk groups mentioned above. During 1997–1999, the highest incidence was observed in people over 75 years and in children under 1 month age (Figure 36).

Figure 36. Average incidence (cases per 100 000 inhabitants) of listeriosis in humans in different age groups 1997–1999 (n=62).



Listeria in food

There is no officially co-ordinated surveillance system for *L. monocytogenes* in food. Surveillance is achieved by various projects initiated by the authorities as well as the industry. In addition, *L. monocytogenes* is often included in own check programmes run in relevant sectors of the industry. In the near future, however, a stricter control of *L. monocytogenes* in ready-to-eat foods is to be expected in the EU.

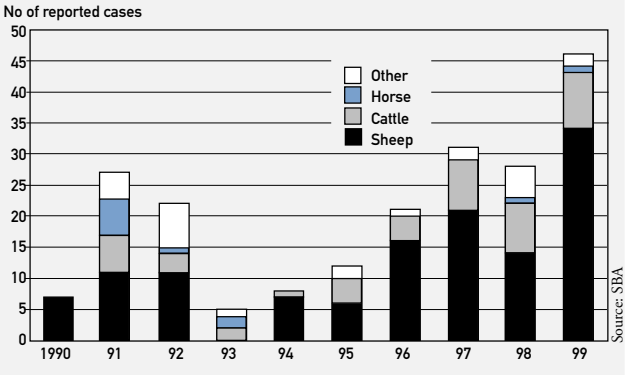
Results of the surveillance in Sweden, show that approximately 1–2% of pig and cattle carcasses, 3–10% of meat/meat products/fish products and a few percent of vegetables are contaminated with *L. monocytogenes*. The bacterium has also been found in approximately 1% of heat-processed meat products. Findings of *L. monocytogenes* in food are not notifiable. If the bacterium is found in food not intended to be further heat treated, the food will be deemed unfit for human consumption if the number of bacteria exceed a specified cut-off point. One *Listeria* outbreak has been reported in Sweden. It occurred in 1994/95 and involved eight persons. The source of infection was vacuum-packed cold smoked or "gravad" rainbow trout/salmon. Since the incubation period (time from consumption of contaminated food to clinical manifestation) may be several weeks, it is often hard to determine the source of infection in human cases.

Listeriosis in animals

Listeria has been reported to cause disease in a wide range of animals. The most susceptible species are sheep followed by goats and cattle. The bacterium has been isolated from many species of mammals, birds, amphibians and reptiles. Listeriosis manifests itself in ruminants as encephalitis, neonatal mortality and septicemia. The most common clinical form is encephalitis. *L. monocytogenes* has also been reported to cause mastitis in cattle and as the agent can be excreted in milk over a long period this may have an effect on public health if unpasteurised milk or products thereof are consumed. The role of animals in the epidemiology of listeriosis is not well known. Although some researchers consider listeriosis a disease common to man and animals and not as a zoonosis *per se*, it is probable that animals contribute to maintenance of the bacterium in the environment.

In Sweden, listeriosis is a notifiable disease in animals. Since 1990 between 5 and 46 cases (infected herds) have been reported annually, the majority of cases occurring in sheep and other domestic ruminants. Single cases have also been diagnosed in other species such as horses, cats, chinchillas, hares and wild birds. (Figure 37).

Figure 37. Number of notified cases of (infected herds with) listeriosis in animals 1990–1999.



Borreliosis

Introduction

Lyme borreliosis is a tick-borne disease caused by the bacteria of the *Borrelia burgdorferi* sensu lato (s.l.) group. The first agent to be associated with the disease was identified in 1982 in the USA and named *B. burgdorferi* (later *B. burgdorferi* sensu stricto). Also other borreliae were later found to cause Lyme borreliosis, e.g. *B. afzelii* and *B. garinii*. Wild rodents are the main reservoir for infection with *B. burgdorferi* s.l., which are transmitted to humans and other animals through tick bites. The most common tick in Sweden, like in the rest of Scandinavia, is *Ixodes ricinus*. The ticks are most abundant in the southern and central parts of Sweden, but can be found in the northern part along the coast of the Baltic sea.

B. burgdorferi s.l. in humans

Lyme borreliosis is a multisystemic disorder that can affect several organ systems, primarily the skin, nervous system, heart and joints. The predominant manifestation is the skin "erythema migrans", which is considered to be pathognomonic. The disease is not notifiable in Sweden. The number of clinical cases with Lyme borreliosis is estimated to be around 10 000 per year. In a prospective study performed in southern Sweden in 1992–1993, the annual incidence was shown to vary between 26 and 160 cases per 100 000 inhabitants with a mean of 69 cases/ 100 000 inhabitants.

B. burgdorferi s.l. in animals

Serologic evidence of infection with *B. burgdorferi* s.l. is found in animals. There is no clear case definition, although a variety of symptoms have been reported as associated with infection. The most commonly reported signs are lameness, swollen joints, stiffness, myalgia, and fever. In Sweden, a study performed in horse in 1997–98, showed an overall seroprevalence of 16.8%. Another study in 1991–94, performed in dogs, not suspected of having Lyme borreliosis, showed that 3.9% were seropositive. In 1989, 21.9% of studied dogs with clinical signs suggesting Lyme borreliosis, were seropositive. Lyme borreliosis is not a notifiable disease in animals in Sweden.

Granulocytic ehrlichiosis

Introduction

Ehrlichia species of the *E. phagocytophila* genogroup belong to the granulocytic ehrlichiae, which are strictly intracellular bacteria, infecting blood granulocytes. So far, only this type of ehrlichiae has been identified in Sweden, but in southern Europe and in, e.g. the USA, monocytic ehrlichiae are known pathogens of both humans and animals. In Sweden, the granulocytic ehrlichiae are transmitted by *Ixodes ricinus* ticks and wild small rodents are assumed to be the reservoir of infection. For certain *Ehrlichia* species, also large mammals are identified as competent reservoirs. Granulocytic ehrlichiosis is characterized as an acute febrile illness with the ehrlichiae visible as microscopic inclusions within blood granulocytes in stained blood smears during the acute stage of the disease.

Granulocytic ehrlichiosis in humans

Human granulocytic ehrlichiosis (HGE) was first described in the USA in 1994. The clinical disease is generally nonspecific and characterized by fever, head-ache, myalgia and malaise. Systemic involve-

ment, such as gastrointestinal, respiratory or CNS manifestations may be severe, but only occur in few patients according to reports from the USA. In Sweden, a seroepidemiological study performed on blood samples taken from a population in the south-western part in 1994, showed that 11% of the samples contained antibodies reactive with *E. equi*. The first Swedish clinical HGE cases were reported in 1998, and a few cases have been identified in the country since then. The disease is not notifiable in Sweden.

Granulocytic ehrlichiosis in animals

The first granulocytic ehrlichiosis to be identified in animals was tick-borne fever in sheep in the 1930s. Later, it was also recognized in cattle as pasture fever. In sheep and cattle the causative agent is *E. phagocytophila.* In the 1970s, *E. equi* was found to infect horses in the USA, and in 1995 another granulocytic *Ehrlichia* was identified as causing the disease in Swedish dogs and horses. Based on 16S rRNA gene sequencing, the *Ehrlichia* species of Swedish dogs and horses is closely related to *E. phagocytophila* and *E. equi* and identical to the human granulocytic ehrlichisosis (HGE) agent. Typical clinical signs of acute granulocytic ehrlichiosis in animals are high fever, fatigue and inappetence. In addition, horses often develop distal limb edema, dogs gastrointestinal symptoms, lactating cows agalactia and pregnant dams may abort. In a Swedish study in 1997–1998, the overall seroprevalence in horses to granulocytic *Ehrlichia* sp was16.7%. The overall seroprevalence among dogs, not suspected of having granulocytic ehrlichiosis, was 17.7% in a study conducted during 1991–1994. The disease in animals is not notifiable in Sweden.

Giardiasis

Introduction

Giardia is a flagellated protozoan parasite which is one of the most common intestinal parasites of humans in temperate and tropical regions. *Giardia* was first described in 1859 but the organism was observed as early as in the 17th century. In the 1920s, clinical symptoms due to *Giardia* infection in humans were described for the first time, but it is only recently that the significance of *Giardia* as a cause of chronic diarrhoea in children in developing countries has been established. Today there is also increasing evidence that *Giardia* should be considered as a zoonotic agent.

Five species are currently recognised in the *Giardia* genus, although more than 50 species have been described. *Giardia duodenalis* (syn. *G. intestinalis* and *G. lamblia*), the species that occurs in humans, can also affect a wide range of other vertebrate animals such as rodents, livestock, cats, dogs, and possibly also birds and reptiles.

The parasite is mainly transmitted by faecally contaminated drinking water and food. Person-to-person spread also occurs by the faecal-oral route, especially among children at day-care centres. The source of infection is usually man but animals may provide an additional source of infection.

The infectious dose for *Giardia* is low, less than 100 parasites can cause infection. After ingestion, the parasite excysts and rapidly colonises the small intestine. After multiplication some of the organisms (trophozoites) will encyst and finally be shed by the faeces. The *Giardia* cyst is the main transmissible stage of infection. It is relatively resistant and can survive for at least two months in suitable temperature and moist environment. The ability of *Giardia* cysts to withstand chlorination of drinking water has made water an important vector of giardiasis in humans in countries with no other water purification than chlorination. In acute infections,

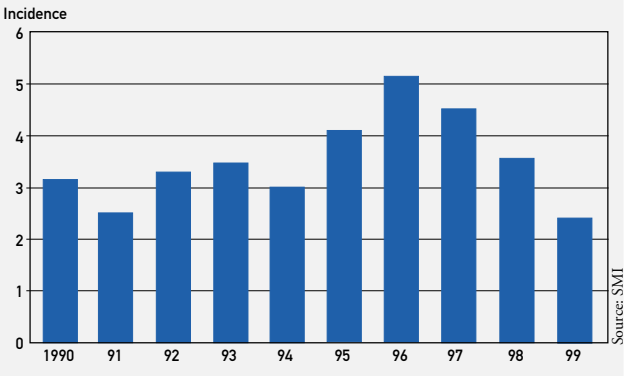
trophozoites may also be passed in faeces but the trophozoite stage is not particularly resistant to environmental changes. At present *Giardia* is considered to multiply only asexually as no sexual reproduction has been identified.

Giardiasis in humans

The clinical signs in human giardiasis vary greatly from subclinical to diarrhoea or chronic disease associated with nutritional disorders. The duration of infection is a few days to several months. In the majority of patients, symptoms disappear in a few weeks. The variability in symptoms may be due to immune as well as nutritional status but also due to variations in pathogenicity in different strains of *G. duodenalis*. Immunodeficient individuals are particularly at risk of developing a chronic disease. Children, especially in developing countries, appear to be more at risk for chronic disease and *Giardia* infection may cause malnutrition and retarded growth and development. The prevalence varies from between 2 and 7% in Europe, North America and Canada to more than 40% in developing countries. During recent decades *Giardia* has caused several waterborne outbreaks that have attracted much attention. The infections have been due to drinking of, or swimming in, contaminated water.

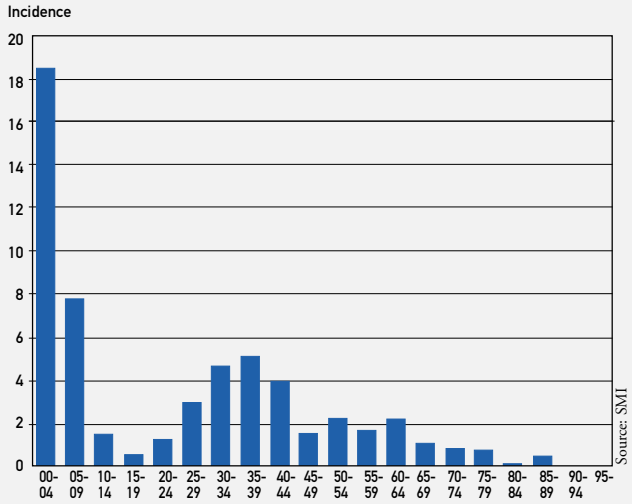
In Sweden, infection with *Giardia* in humans is notifiable. Since 1990 on average 3.5 (2.4–5.2) domestic cases per 100 000 inhabitants have been reported annually (Figure 38).

Figure 38. Incidence (cases per 100 000 inhabitants) of domestic human cases of *Giardia* 1990–1999.



The most important source of domestic infection is person-to-person spread at day-care centres, contaminated food or water.

Figure 39. Average incidence (cases per 100 000 inhabitants) of domestic human cases of *Giardia* per age group 1997–1999 (n=941).



Waterborne outbreaks of giardiasis are occasionally reported in the Nordic countries. In Sweden, two major outbreaks have been reported. In 1986, an outbreak involving 3600 persons occurred in Dalarna county and in 1982 another outbreak that involved 557 persons was reported in Blekinge county. The outbreaks were due to contamination of drinking water with sewage.

Giardiasis in animals

Giardia organisms are common findings in faeces in livestock, dogs and cats but they are rarely associated with clinical disease. If clinical signs due to *Giardia* occur they are similar to those seen in humans, e.g. diarrhoea and malabsorption. One reason why clinical giardiasis in animals is seldom reported might be that concurrent infections often occur which make it difficult to definitively establish the cause of symptoms. However, *Giardia* might be an underestimated cause of diarrhoea in young mammals.

In Sweden, infection with *Giardia* is not notifiable in animals. In regional studies performed on Swedish lambs older than seven weeks of age, *G. duodenalis* was a common finding. Since 1992 *Giardia* has also been found in many other mammal species such as dog, cattle, goat, several rodent species and wild animals such as moose and roe deer.

Giardia in the environment and water

Since *Giardia* as well as other parasites such as *Cryptosporidium* is rather chlorine resistant they will not be affected by normal chlorination at water works. Slow sand filters will, however, give sufficient protection against *Giardia*. In Sweden, most large waterworks with surface water sources will have such filters.

Investigations performed on surface water in Sweden showed that 13 (26%) samples contained *Giardia*. Although it is not known if these *Giardia* species were pathogenic for humans, it emphasises the importance of efficient water purification including several purification steps. In Sweden, it is mandatory to have several purification steps when surface water is used as source. That might explain why only waterborne outbreaks with intrusion of sewage-polluted water have been reported in Sweden.

Cryptosporidiosis

Introduction

Cryptosporidium is a genus of protozoan parasites that infect the gastrointestinal tract of vertebrates. At least six species of *Cryptosporidium* have been identified but only one species, *Cryptosporidium parvum* is of public health concern. *C. parvum* was considered as a commensal until the 1970s when an episode of diarrhoea in cattle due to *C. parvum* was described. Some years later human infection with *C. parvum* was also described. In the 1980s the parasite became widely acknowledged as a serious pathogen in immunocompromised persons, especially AIDS patients.

C. parvum has a world-wide distribution with an annual incidence in developed countries of <1 to 4.5 % and a much higher incidence in developing countries. In the United Kingdom, peaks in disease occurrence in humans have been observed reflecting rainfall or events such as calving or spreading of manure. This indicates that livestock may be an important reservoir of *C. parvum* for humans and that contaminated water is a key vector for the parasite. The largest waterborne outbreak so far reported occurred in the USA and involved more than 400 000 people, of whom 100, mostly AIDS patients, died. The investigations revealed some deficiencies in water treatment but the water produced during the incident complied with American water quality standards.

Infection with *Cryptosporidium* occurs via the faecal-oral route. The source of infection usually is direct contact with an infected man or animal, indirect contact by a faecally contaminated environment, or ingestion of uncooked contaminated food or contaminated water. Its small size as well as its tolerance to chlorine and other disinfectants may enable the parasite to pass the different steps of water purification in the waterworks. The parasite is unusually resistant and can survive for months under suitable conditions, for example in water. A man or animal is infected by *C. parvum* by ingestion of the encapsuled stage of the parasite (oocyst). In the intestine of the host, the oocyst excysts and undergoes a series of proliferative changes in the intestinal epithelium. During this process *C. parvum* rapidly multiplies in its host. After approximately 2–12 days, infective parasites (oocysts) are shed with faeces.

Cryptosporidiosis in humans

The infectious dose for *C. parvum* is very low. *C. parvum* usually causes a self-limiting gastrointestinal infection with watery diarrhoea, abdominal pain and cramps. Vomiting is less common. In some patients mild symptoms may persist for some weeks after the acute phase. In immunocompromised individuals, especially AIDS patients, the

infection is usually more severe and can be life-threatening.

In Sweden, infection with *Cryptosporidium* in humans is not notifiable. Since 1994, however, diagnostic laboratories report cases on a voluntary basis. Approximately 40 cases have been reported annually. However, as notification is not compulsory, this figure clearly does not reflect the true occurrence of disease. One waterborne outbreak, with three different etiological agents (*Campylobacter*, *Giardia* and *Cryptosporidium*) involving 600 persons, has been reported in Sweden. It occurred in 1991 and was due to intrusion of river water into the drinking water pipelines. As in many other countries, *C. parvum* has been identified in surface water supplies in Sweden.

Cryptosporidiosis in animals

C. parvum has been recorded in a wide range of mammals. In young ruminants, watery, profuse diarrhoea can be seen. In pigs, companion animals, horses and rodents clinical symptoms seldom occur.

In Sweden, finding of *Cryptosporidium* in animals is not notifiable. The parasite is common in Swedish cattle. A regional study performed on calves less than 3 months of age showed that *C. parvum* was the second most common pathogen found in diarrhoeic calves. The parasite has also been identified in other species such as rabbit, guinea pigs, pheasant and snakes.

Cryptosporidium in the environment and water

Cryptosporidium are not eliminated by normal chlorination at waterworks as it is rather resistant to disinfectants. Compared with *Giardia* cysts, *Cryptosporidium* oocysts are smaller and thereby more likely to pass filters in waterworks. However, slow sand filters will give sufficient protection and most large Swedish waterworks with surface water sources will have such filters.

In one investigation including 26 Swedish surface water supplies, *C. parvum* was detected in 32% (16/50) of the samples. One positive sample was also found in treated water originating from a contaminated water supply.

Toxoplasmosis

Introduction

Toxoplasmosis is caused by a monocellular parasite, *Toxoplasma gondii*. Although the parasite was discovered in 1908 in a rodent it was not until 1970 that *T. gondii* was found in feline faeces, thereby identifying cats and other felids as the definite host of the parasite.

The life cycle of *T. gondii* can be divided into two parts: sexual multiplication in the intestine of cats and asexual multiplication in different tissues of intermediate hosts. Infected cats excrete the parasite in faeces where it becomes infectious after 1–5 days and, under favourable conditions, can survive in the environment for more than a year. Many, probably all, warm-blooded animals can act as intermediate hosts. After ingestion of the parasite, it multiplies in the intermediate host and spreads via lymph- and blood-vessels through the body. Approximately three weeks after the infection, the parasite encysts in muscular and neural tissues where it can survive for several years, or possibly throughout the remaining lifetime of the host. Thereby two major sources of infection exist for humans and other intermediate hosts; ingestion of the parasite from the environment or ingestion of muscle tissue from infected animals containing infectious tissue cysts (Figure 40). Cats, the definitive host, usually get infected by eating a rodent with encysted parasites. The parasite multiplies in

the intestine and the cat begins to excrete parasites in faeces.

T. gondii has a world-wide distribution in humans and animals. Although infection is common, clinical disease in humans is not. The public health importance of the parasite lies in the potentially severe sequelae of congenital infection in humans. Apart from sheep, where infection with *T. gondii* is a major cause of abortion, toxoplasmosis in most animals usually is a subclinical disease. *T. gondii* is destroyed by heat (67°C) and deep freezing (-20°C).

Toxoplasmosis in humans

Most *T. gondii* infections in humans are asymptomatic and life-long immunity is acquired. The parasites are usually not eliminated from the body and remain in a latent form probably throughout the lifetime of the host. At times the parasite can produce devastating disease in the foetus when a nonimmune woman becomes infected during pregnancy. The symptoms at congenitally acquired toxoplasmosis vary greatly. Abortion, severe brain lesions with mental retardation may occur. When infection occurs later in pregnancy, mild or subclinical disease with delayed manifestation (ocular disease) may occur. Currently, increasing numbers of cases are seen in immunosuppressed people, usually due to reactivation of a previously latent infection. The disease in these persons is generally serious and often fatal.

Humans are usually infected by undercooked meat containing tissue cysts or faecally contaminated food. Direct or indirect contact with cat faeces containing cysts may also cause infection. Generally, consumption of undercooked meat is considered to be the major source of infection. As toxoplasma infection is more common among pigs and sheep than cattle, meat from the former species is considered as an important source of human infection. Although infective parasites have been recovered from poultry tissues, the risk of transmission is regarded as slight as poultry meat is usually well-cooked before consumption.

Infection with toxoplasmosis is notifiable in Sweden. During the last 10 years, between 4 to 22 cases were reported annually. This data is not reliable as the infection is almost always asymptomatic, and very rarely diagnosed. A few cases of probable congenital toxoplasmosis are notified each year, but they represent a fraction of the total infection pressure, and give no indication of overall incidence or risk factors. Limited studies performed in certain areas of Sweden indicate that the seroprevalence among pregnant women is decreasing. In the late 1950s approximately 50% were seropositive

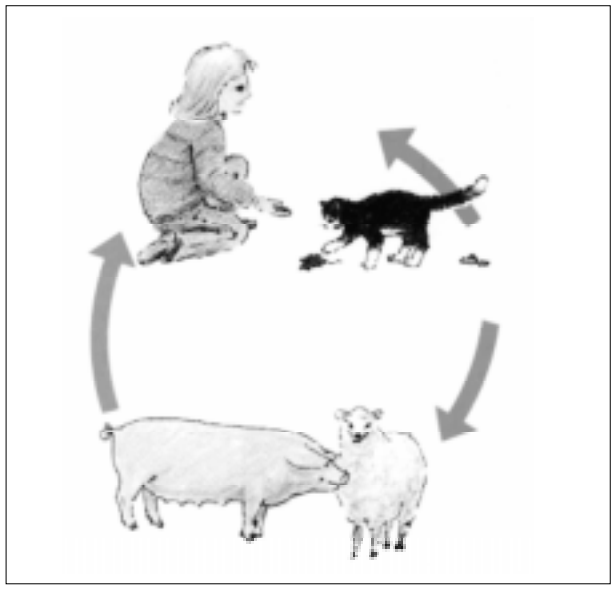


Figure 40. Life cycle of *T. gondii*.

compared to approximately 15% in the early 1990s. A study performed on newborn in 1997–1998, showed that the seroprevalence in fertile women in Stockholm city and Skåne county were 14% and 26% respectively. The reason for this regional difference is unknown. In pregnant nonimmune women the incidence was estimated to be less than 1 case per 1000 pregnant women and in their foetuses the incidence was lower than 0.1 per 1000. The study showed that the incidence of toxoplasmosis in pregnant women as well as in foetuses is low.

Toxoplasmosis in animals, *T. gondii* in food

The disease in animals is similar to that in man, i.e. the infection is generally asymptomatic. However, in sheep, goats and also pigs, toxoplasmosis may cause perinatal mortality. Australian marsupials and neotropical monkeys are highly susceptible and infection may often lead to death. The brown hare and mountain hare are two other species where acute fatal toxoplasmosis has been frequently described. Among livestock, pigs, sheep and goats relatively often harbour *T. gondii* in tissues. Cattle appear to be more resistant and capable of clearing the infection. Therefore, beef is generally not regarded as an important source of human infection.

Toxoplasmosis in animals is not notifiable in Sweden. Almost every year sporadic cases of abortion occur in sheep. Results of serological investigations performed in the 1980s showed that approximately 40% of the cats, 23% of the dogs, 20% of the sheep, 10% of the pigs, 1% of the horses and 0% of brown hares had antibodies against *Toxoplasma*. It also showed that the prevalence of seropositive sheep increased with increasing age and that sheep kept outdoors more often seroconverted than sheep kept indoors. In 1999 a survey was performed in pigs originating from different parts of Sweden. The overall prevalence of seropositive pigs was 5% with 3% in fattening pigs and 17% in adult pigs. These results are in agreement with results of investigations in several countries, including Denmark and Finland. Acute toxoplasmosis is not uncommon among Swedish wild hares. Evaluations of necropsies routinely performed on Swedish wildlife during recent decades showed that 8% of 3980 autopsied hares had acute toxoplasmosis.

Trichinellosis

Introduction

Trichinella species are intestinal roundworm parasites. In Sweden three species have been identified; *Trichinella spiralis* which has a cosmopolitian distribution and is the etiological agent of trichinellosis in domesticated animals; *T. nativa*, occurring among wild life in arctic and sub-arctic regions, and *T. britovi*, occurring in wild life in temperate areas of the palearctic region.

Trichinella spp. are unusual among nematode parasites as their whole life cycle, from larvae to adult parasite to larvae, is completed in a single host. Animals become infected when eating muscles from infected animals containing encapsuled *Trichinella* larvae. In a similar way, humans become infected when eating under-cooked or raw meat from infected animals. Ingested larvae mature to adult parasites, mate and the fertilised female begin to release new-born larvae in the intestine of the host. Larvae cross the intestinal wall and spread to other organs and tissues via the lymphatic and blood circulation. In the striated muscles, the larvae finally encapsulate. Larvae remaining in other organs and tissues die within a short time. When the life cycle is completed, the muscle of the infected animal contains larvae that can survive for long periods, in some hosts for many years. Larvae can also remain viable for considerable time (months) after death of the host, thereby allowing transmission by scavenging.

As a preventive measure, examination for *Trichinella* is usually performed at slaughter. In Sweden, all pigs (including wild boars), horses and bears are controlled for *Trichinella* at slaughter. Heat treatment of meat (at least 68°C) will kill the parasite and freezing will kill *T. spiralis.*

Trichinellosis in humans

Clinical signs are highly variable and can range from subclinical infection to fatal disease depending on the number of ingested viable larvae. The progression of the disease follows the development of the parasite. When new-born larvae are released in the intestine of the (human) host they may cause gastrointestinal symptoms. The symptoms become more general as the body of the host responds immunologically to the infection. Thirst, profuse swettings, chills and weakness may be observed. Finally when the larvae migrates out to the muscles they can cause muscle soreness and pain. Respiratory and neurological complications as well as death due to heart failure may occur after several weeks.

Trichinosis is a notifiable disease in Sweden. Since 1990 only one case has been reported, a person contracting the disease abroad after eating pork. In 1961 a major outbreak was reported in Blekinge county involving approximately 338 persons where the source of infection was smoked sausage including pork of domestic origin. The latest reported domestic outbreak occurred in 1969 in which a pig, slaughtered on the farm but not subjected to meat inspection (permitted for occasional animals bred on the same farm and which are to be used in the farmer's own household), was the source of infection.

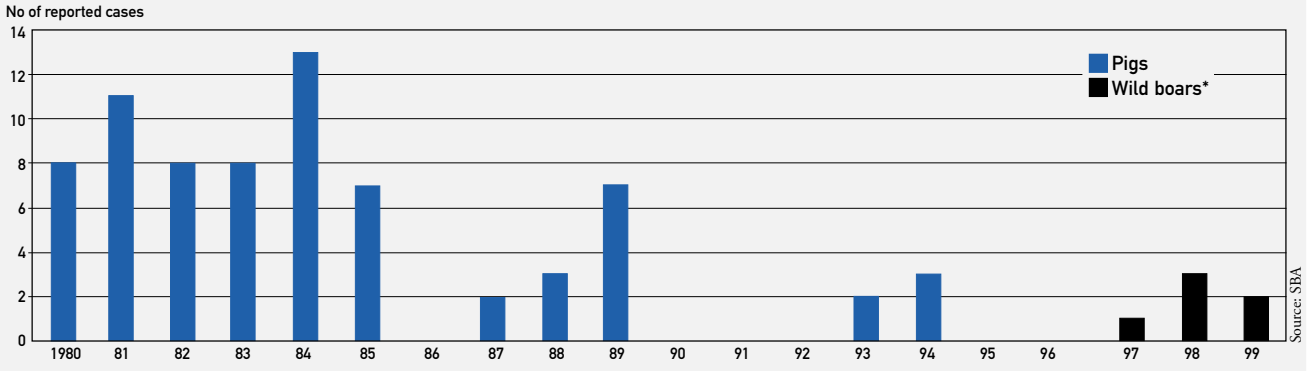
Trichinella in animals and food

Trichinella have a wide range of hosts among domestic and wild animals, the infection has been confirmed in more than 100 species of mammals. The main reservoirs are wild carnivores. In Sweden, the main reservoir is the red fox and approximately 10% of the population is estimated to be infected. All three species (*T. spiralis, T. nativa* and *T. britovi*) have been found in red fox.

Findings of *Trichinella* in animals are notifiable in Sweden. At meat inspection, a total of 5 cases (infected herds) have been reported in domestic pigs during 1990–1999 (Figure 41).

In the 1980s between 0 and 13 cases were reported annually. This figure dropped during the 1990s. The reason for the decrease is not known, but increased standards of stables and improved management might be the cause. The sources of infection were never established but rodents might be suspected. The situation in wild boars is less favourable with cases of *Trichinella* reported annually since 1997. This indicates that the risk that meat originating from wild boars contains *Trichinella* larvae is not negligible. Since 1990, *Trichinella* has also been found in foxes, lynx, a wild wolf, a dog and a cat.

Figure 41. Number of reported cases of (infected herds with) *Trichinella* in pigs 1980–1999.



* 1999: One farmed wild boar and one crossbred wild boar/domestic boar

Echinococcosis

Introduction

Echinococcosis has been known in humans and animals since ancient times. Hippocrates (379 BC) describes a patient "with the liver filled with water". In the 17th century it was recognised that the cysts originated from contacts with animals. In the 19th century sufficient was known about the life cycle of the parasite to initiate successful control of *Echinococcus* in Iceland, where the disease was very common.

Four species of the genus *Echinococcus* are described. Two of these, *E. granulosus* and *E. multilocularis* occur in Europe. This report will only deal with *E. granulosus* as *E. multilocularis,* which causes a severe disease in humans, has never been reported in Swedish animals.

Echinococcus spp. are helminth cestode parasites. The lifecycle requires two hosts, one definitive and one intermediate host (Figure 42). Canids, usually dogs, are definitive hosts of *E. granulosus.* The major intermediate hosts are ruminants. Most commonly domesticated ungulates (sheep, cattle, reindeer, pigs and horses) are intermediate hosts. In endemic areas up to 80% of sheep and 50% of dogs may be infected. In exceptional cases humans become infected.

Infection in the definitive host is acquired by ingestion of raw offal containing cysts (with protoscoloides/larvae). The parasite establishes and develops as tape worms and reproduces in the intestine of the definitive host. Six to eight weeks after the infection, eggs are dispersed with faeces. It is not known how long eggs are shed in faeces, a time period of one to two years has been suggested. The eggs are resistant and can survive for long periods (> 16 months in 4°C) but are killed by ordinary cooking procedures but not by freezing. Intermediate hosts are infected by ingestion of eggs. Larvae develop from the eggs and are transported by the blood to visceral organs where fluid-filled cysts, containing larvae, develop.

Control of the parasite is based on meat inspection and effective disposal of offal at abattoirs combined with prevention of feeding raw offal to dogs and treatment of infected dogs.

Two strains of *E. granulosus* occur. A northern and a European strain. The northern strain is indigenous to the boreal region of the holarctic, with wolf, reindeer and moose as the principal hosts. This type has been reported in reindeer in Sweden. Disease in humans caused by the northern strain causes a more benign disease in humans than the European strain.

Echinococcosis in humans

Infection in humans causes cystic echinococcosis (CE). Although CE is most commonly diagnosed in people aged 30–40 years, infection can occur at any age. The incubation period and clinical picture depends on the organ(s) affected. Both rapid development and long latency periods, up to several years, occur. The most commonly affected organ is the liver, followed by the lungs, and usually only single cysts occur. Cysts may rupture and, depending on the location, lead to severe consequences.

Humans are infected by ingestion of eggs. Transmission can occur directly from dog faeces or indirectly by drinking water or eating raw vegetables

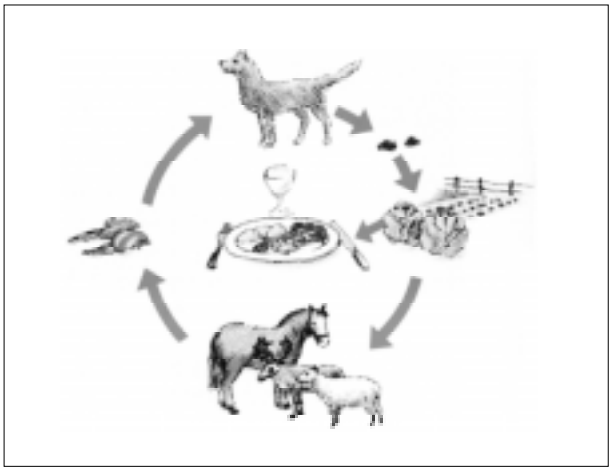


Figure 42. Life cycle of *E. granulosus.*

contaminated by dog faeces containing eggs. It is known that eggs from tape worms can spread over long distances, probably by dust particles or even with flies. The infectious dose is very low, theoretically a single egg may infect man.

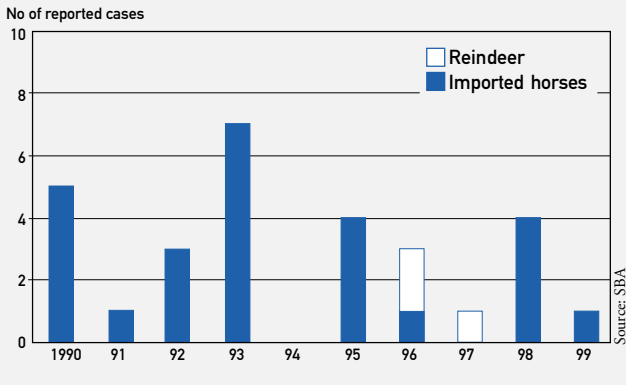
In Sweden, infection with *Echinococcus* spp. in humans is reported on a voluntary basis since 1994. In 1994–1999 between 3 to 11 cases were reported annually, all infected abroad.

Echinococcosis in animals

Infection with *Echinococcus* in animals is notifiable in Sweden. During 1990–1999 the number of cases reported annually has not exceeded seven. Apart from 3 cases in reindeer, all were in imported horses (Figure 43).

In a study performed in 1973 the parasite was found in approximately 2% of reindeer lungs originating from the northernmost part of Sweden. Actions to improve slaughter hygiene by introducing mobile slaughter houses were already initiated.

Figure 43. Number of notified cases of *Echinococcus granulosus* in animals 1990–1999.



By removing all offal the transmission of the parasite to dogs could be stopped. During the winter of 1996/97, *Echinococcus* was reported in three slaughtered reindeer. The source of infection was not identified. Action was taken and focused on treating dogs at risk and preventing them from eating raw offal. No further cases have been found in reindeer.

Nephropatia epidemica

Introduction

Puumala virus is one of several newly discovered agents belonging to the genus hantavirus. Each type has its own main rodent vector and thereby its own geographical distribution. Hantavirus can infect a variety of different species, both mammals and birds. Apart from rodents, the significance of infection in other species for human illness is unclear. Infected rodents remain apparently healthy but have, probably lifelong, capacity to shed infectious hantavirus in saliva, urine faeces and also possibly from the lungs. The survival time of hantavirus in the environment is not known. Transmission to man occurs mainly through aerosol infection of rodent excreta. Humans do not spread the infection further. Clinical signs in man vary between the different types of hantavirus.

The human disease caused by Puumala virus is named Nephropatia epidemica (NE). NE was first described in Sweden in 1934 and Puumala virus, the causative agent, was discovered in 1977 in a bank vole collected in Puumala in Finland. Incidence of NE is reported to reflect the bank vole population. However, variation of the prevalence of Puumala virus in the bank vole population in endemic areas is not well known.

In Sweden, the bank vole population is reported to vary in cycles of 3–4 years with two successive high density years. However, cycles in various areas within Sweden may not coincide. It has not been shown that the incidence of NE in humans in Sweden is correlated to the density of the vole population. There is little information on occurrence

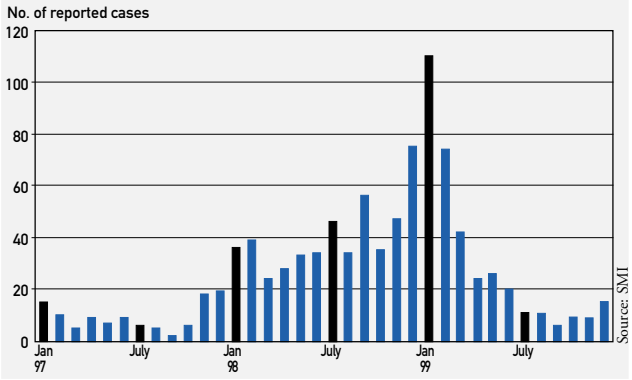
of Puumala virus in animals, other than rodents, and humans. A recent study performed in the northern part of Sweden showed that approximately 2% of investigated moose had antibodies to Puumala virus, indicating a past infection.

Nephropatia epidemica in humans

NE is usually transmitted by inhalation of dust particles contaminated with rodent excreta. The incubation period is estimated to be between 4 and 42 days. NE is a micro vascular disease mainly affecting the small blood vessels and causing "capillary leakage". It is probable that most infections with NE are mild or subclinical and that the diagnosis is not established. It has been estimated that only 10% of human cases are notified. In more severe cases, fever, muscle pain, back and abdominal pain and kidney affection can occur. Patients usually recover within 2 months.

NE is notifiable in humans in Sweden. During 1990–1999 on average 215 (67–518) cases were reported annually. The number of cases varied over time (Figure 44).

Figure 44. Number of reported human cases of Nephropatia epidemica per month 1997–1999 (n=954).



Although the reservoir host is present in the whole country, a regional variation occurs, with almost all cases reported north of limes norrlandicus (59°), mainly in the four northern counties. In 1997–1999, the annual incidence in these four counties varied between 8 and 65 cases per 100 000 inhabitants, with the highest figures observed in the two northernmost counties (Norrbotten och Västerbotten). It remains unclear why the disease does not occur in the southern part of Sweden but still occurs in countries south of Sweden such as Denmark, Belgium, the Netherlands and Germany. Most reported cases occur in middle-aged people, less in young and elderly. Approximately 62% of cases are reported to occur in males. A seasonal variation occurs, with highest incidence during winter months (December and January) (Figure 44). The variation is probably due to the approach of the voles to houses when frost arrives. In studies from Sweden, cases of NE have been associated with handling of firewood, cleaning of houses and to a lesser extent, handling of hay.

Rabies

Introduction

Rabies virus can infect all warm blooded animals and infection almost invariably results in death. The disease has a world-wide distribution. More than 90% of human cases are contracted in the tropics and the source of infection usually is a rabid dog. Various mammalian species may act as reservoirs. Within a certain area the rabies virus may adapt to a certain species. This may result in a major single host reservoir in a certain area and where infections in other animals are spill-over effects caused by sporadic contact with the major host. In Europe, fox is the major reservoir of rabies. Oral vaccination of wild life, successfully performed in the central and western Europe, has decreased the number of rabies cases. At present, rabies in terrestrial animals is on the brink of eradication. Rabies in European bats was first recognised in 1950s and has since then been reported from several European countries. The most frequently affected bat is *Eptesicus serotinus.*

Rabies in humans

Humans are usually infected by a bite or licking by an infected dog. The incubation period for rabies is more variable than in any other acute infection in humans. It is usually 3 to 6 weeks but may vary from 5 days to more than a year. The incubation period depends on the site of the bite, the severity of the bite, the quantity of virus transferred and the immune status of the host. Infection with rabies virus may cause symptoms such as headache, fever, tiredness. Parestesia and itching on the site of the bite may occur. Finally, neurological symptoms develop and coma and respiratory arrest will occur. Vaccination gives a good protection even as post-exposure treatment provided it is given soon after exposure.

Rabies is a notifiable disease in humans in Sweden. The most recent case of human rabies occurred in 2000 when a person contracted rabies after taking care of a puppy in Thailand.

Rabies in animals

Animals infected with rabies virus develop behavioural changes and paralysis. Clinical signs may vary. In the "dumb form" of rabies, animals become lethargic while in the "furious form" animals become uneasy, impatient and also aggressive. Death usually intervenes 2–7 days after development of clinical signs. Rabies virus may appear in the saliva of the rabid animal up to 10 days before onset of illness.

In Sweden, rabies in animals is notifiable. Sweden is free from rabies. The last cases of rabies occurred in 1886. Sweden has a strict import control, either dogs and cats shall be quarantined for four months or pet travel schemes involving identity control, vaccination and antibody titre control are required.

In Denmark, rabies has been reported in bats (*Eptesicus serotinus*). As this bat species also occurs in the southern part of Sweden, repeated surveys on bats are performed.

TBE-virus

Introduction

Tick-borne encephalitis, TBE, is caused by a flavivirus. Small rodents and insectivores are competent reservoirs for the virus, which is transmitted by *Ixodes* ticks to larger animals and man. In the Baltic countries and also in former Czechoslovakia, virus has been isolated from the milk of viremic lactating ruminants. Humans have been infected by consumption of unpasteurized milk or milk products. This route of transmission to humans has not been observed in Sweden.

The first case of TBE in Sweden was verified in 1954, and since then the disease in humans has been monitored.

In Sweden, TBE is considered endemic along the south-east coast of the Baltic and around the lake Mälaren.

TBE-virus in humans

Typically, the disease has a biphasic course. In the initial stage, uncharacteristic symptoms such as moderate fever, headache and myalgia occur. After an asymptomatic interval, 5–30% of those with clinical symptoms in the first stage, develop a second phase of the disease with symptoms of meningo-encephalitis which vary in severity. Hospitalisation in this stage is usually required, and about 10% develop cranial or peripheral paresis. Sequelae are seen in up to 25% of cases. In Sweden, 50–100 cases with TBE are reported each year (Figure 45). About 85% are infected in the Stockholm area. The situation has been stable during the 1990s. TBE is not notifiable in Sweden.

TBE-virus in animals

In Sweden, serological studies have shown that several animal species such as cattle, dogs, horses and wild mammals and birds have been exposed and infected with TBE-virus. In general, TBE in animals is thought to pass as a subclinical infection. Very few clinical cases among animals have been reported. In those cases, the clinical picture has been characterized by severe, often fatal, symptoms from the central nervous system. In Sweden, only one clinical confirmed case, in a dog, has been reported. Occasionally, suspected animal cases are tested and found seropositive. The finding is not notifiable.

Figure 45. Number of reported cases (voluntary reports from laboratories) of TBE in humans, 1980–1999.

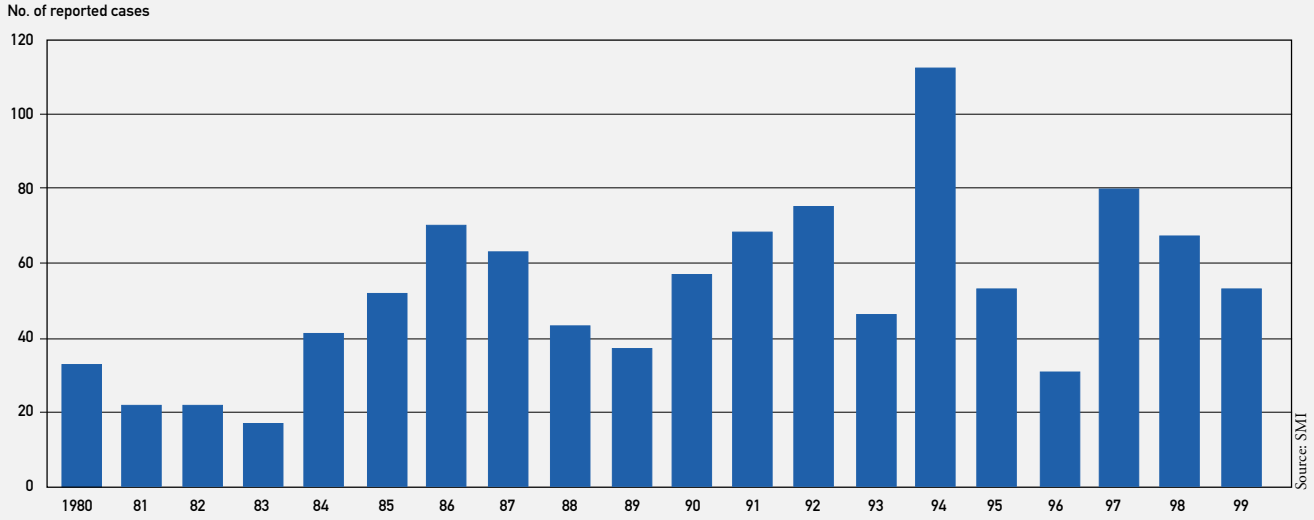


Table 16. Animal population, number of animals (in thousands), number of herds, number of slaughtered and sanitary slaughtered animals in Sweden 1999.

Animal species	Number of animals (in thousands)	Number of herds	Number Slaughtered[2]	Number Sanitary slaughtered[2]
Cattle > 1 year [1)]	1 213	n.a.	482 158	1 451
Calves < 1 year [1)]	499	n.a.	38 519	5
Total No. of cattle [1)]	1 712	33 990	520 677	1 456
Sows, gilts [1)]	220	4 108	n.a.	0
Boars [1)]	4	2 478	n.a.	0
Fattening pigs[1)]	1 239	4 907	n.a.	0
Piglets [1)]	650	3 657	n.a.	-
Total No. of pigs [1)]	2 114	6 012	3 814 534	0
Sheep[3)]	437	8 261	197 542	0
Goats, not kids	n.a.	n.a.	n.a.	0
Farmed deer [4)]	18	569	1 135	0
Horses [1, 5,6)]	80	14 324	5 509	726
Reindeer	n.a.	-	9 897	0
Wild boar (farmed and wild)	-	-	204	0
Moose	-	-	1 116	1
Total number of poultry [1)]	13 709	6 650	-	-
Turkeys	n.a.	n.a.	538 179	-
Ducks	n.a.	n.a.	30 221	-
Geese	n.a.	n.a.	36 193	-
Ratites	n.a.	n.a.	-	-
Broilers	-	-	66 607 582	-
Laying hens	-	-	3 069 600	-
Breeders	-	-	521 070	-

1) Source: No. animals /herds: National Board of Agriculture
2) Source: National Food Agency
3) Including 243 000 lambs
4) Source : No. animals /herds:Swedish Meats
5) Only including horses on agriculture companies
6) The total no. of horses is estimated by insurance companies to be 220 000.

Table 17. Human population (in thousands) by sex, in Sweden 1999.

Female	Men	Total
4 481 308	4 380 118	8 861 426

Source: Official Statistics of Sweden, Statistics Sweden

Figure 46. Number of cattle per county 1999.

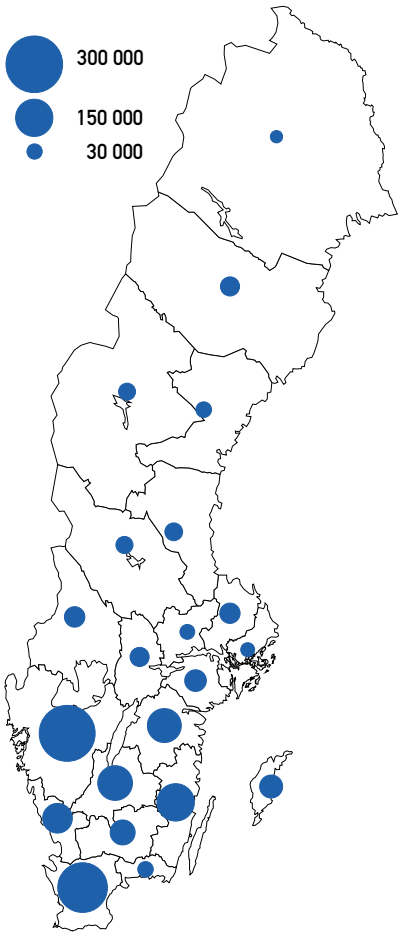


Figure 47. Number of dairy cattle per county 1999.

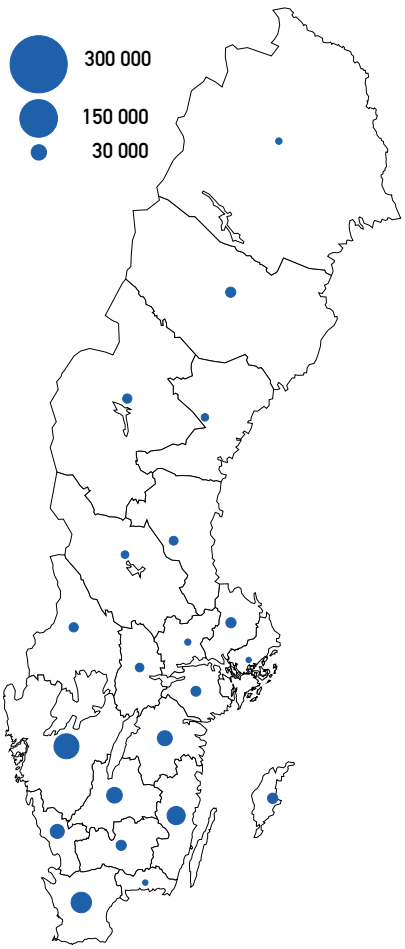
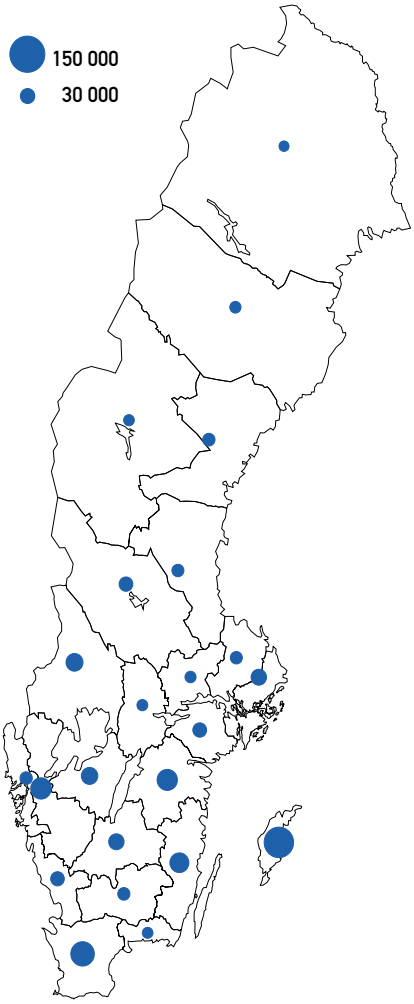


Figure 48. Number of sheep per county 1997.



Source: Year book of ASgricultural Statistics 2000.

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Figure 49. Number of sows and boars per county 1999.

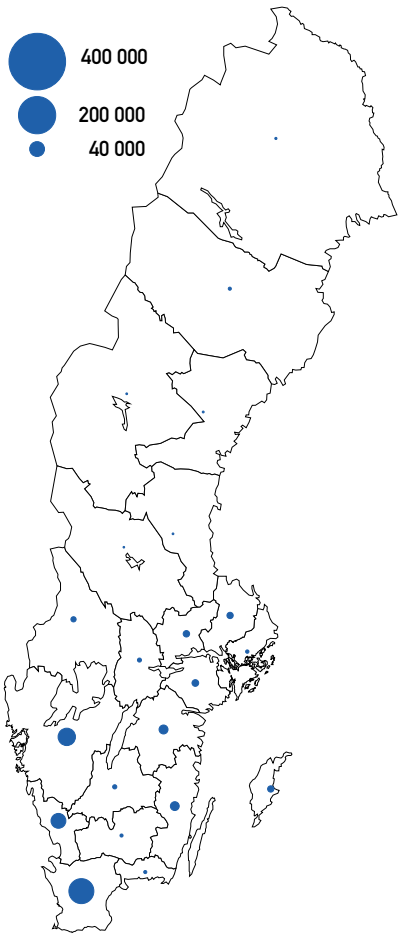


Figure 50. Number of fattening pigs per county 1999.

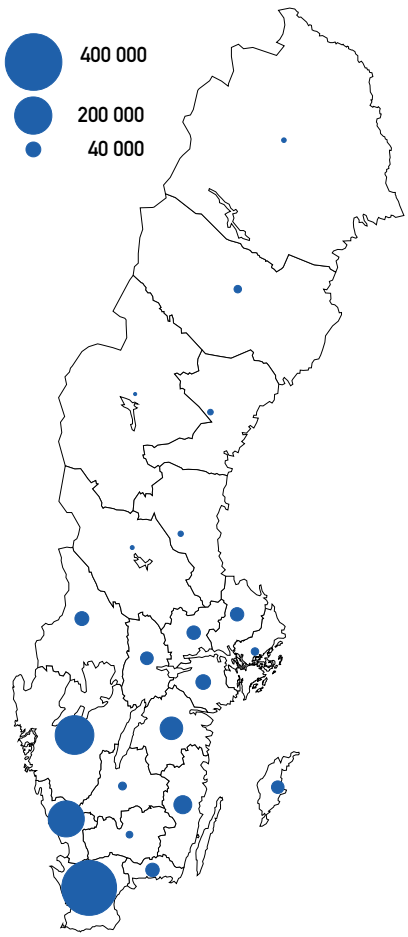
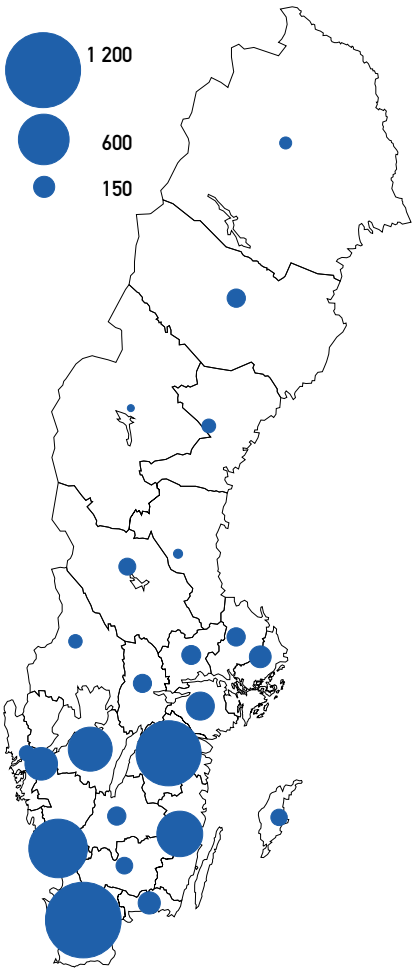


Figure 51. Number (in thousands) of fowls (chicken excluded) per county 1997.



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Counties in Sweden