EUROPEAN COMMISSION
HEALTH & CONSUMER PROTECTION DIRECTORATE-GENERAL

Directorate B - Scientific Health Opinions
**Unit B3 - Management of scientific committees II**

**OPINION OF THE**

**SCIENTIFIC COMMITTEE ON VETERINARY MEASURES RELATING TO PUBLIC HEALTH**

**ON**

# FOOD-BORNE ZOONOSES

(12 April 2000)

## 1. TERMS OF REFERENCE

The Scientific Committee on Veterinary Measures relating to Public Health is requested to express an opinion on the basis of zoonoses[1] control policies. Special attention should be paid to the assessment of risks related to zoonotic diseases causing major concern to public health. The Scientific Committee is invited to present a qualitative and where possible a quantitative risk assessment. The risk assessment should provide for analysis of different pathogens in relation to specific animal species and the type of their production. Based on this analysis, the Committee is requested to identify various practical options which could be considered to control the presence of zoonotic agents at primary animal production level and throughout the rest of the production system in order to decrease the incidence of food-borne diseases in humans.

## 2. BACKGROUND

A two step approach to control zoonoses was provided in the so-called "Zoonoses Directive" (Directive 92/117/EEC[2]). Firstly it provides for the collection of information on the epidemiology of various zoonoses and secondly, based on that information, proposals for the appropriate control measures are foreseen.

At present the control measures focus on certain serotypes of *Salmonella* in poultry breeding flocks. A top-down approach was introduced by firstly providing for the measures to eradicate *S. enteritidis* and *S. typhimurium* in breeding flocks in order to reduce the vertical transmission to commercial flocks. Measures in commercial flocks were foreseen in the future.

The collection of epidemiological data has improved steadily but it is not yet sufficient to carry out comparative studies of the incidence and prevalence of zoonoses. Currently all 15 Member States submit their annual reports on trends and sources of zoonoses. However, the quality of the data still suffers from non-harmonised monitoring and surveillance systems.

The statistical trends on zoonoses in the EU reveal that the current situation with regard to foodborne zoonotic infections is not satisfactory. Although progress has been made in the control of several zoonotic infections, the incidence of others is still high (salmonellosis) or continue to rise (campylobacteriosis and VTEC infections).

Measures against zoonotic organisms (mainly *Salmonella* in poultry) have been initiated in most of the Member States. However, differences exist in relation to

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1 The term "zoonoses" refers here to diseases transmissible from animals to man, but excluding transmissible spongiform encephalopathies. Opinions on the latter, issued by the SSC, are available on http://europa.eu.int/comm/dg24/health/sc/ssc/outcome_en.html).

2 Council Directive 92/117/EEC of 17 December 1992 concerning measures for the protection against specified zoonoses and specified zoonotic agents in animals and products of animal origin in order to prevent outbreaks of food-borne infections and intoxications, OJ L 62, 15.3.93, p. 38

measures taken when positive animals / flocks are detected as well as regarding the acceptability of certain control measures, such as the use of antibiotics, vaccination and competitive exclusion. Presently not all Member States apply a *Salmonella* control program in poultry breeding flocks, which is in conformity with the minimum requirements provided for in the Zoonosis Directive.

It is envisaged that in the future, Community legislation on food safety will cover the entire food production chain “from farm to table” in order to reinforce the safeguard on human health from food-borne infections. The Zoonoses Directive is an existing tool, which provides for control measures for certain zoonoses in specific animal species. In order to reflect the need for an integrated approach in food safety, it is considered to enlarge the scope of the Zoonoses Directive and to cover other animal species and/or other zoonotic agents.

## 3. ZOONOSES

### 3.1. Definition

Article 2 of Council Directive 92/117/EEC ("Zoonoses directive") defines as

- a *zoonosis* any disease and/or infection which is likely to be naturally transmitted from animals to man.

- a *zoonotic agent* any bacterium, virus or parasite which is likely to cause a zoonosis.

### 3.2. The objective and the scope of the report

Considering the above wide-ranging definition and taking into account the deadline for the revision of the zoonoses directive (31 March 2000) and the specific mandate given, the Committee decided to focus only on foodborne zoonoses of major public health concern and to consider only infections that are relevant for Europe.

The Committee assessed the wide and diverse range of zoonotic pathogens and focused on those of major public health significance. The risk reduction initiatives that were suggested for those pathogens assessed, might also have an impact for those not covered by this report.

The Committee did not address environment borne zoonoses, toxins or poisons acquired from animals or animal products or pathogens existing in certain environments which can become a common source of infection for man and animals. This excludes a large group of water- and food-borne infections having humans as reservoirs**.** However the Committee draws the attention of the Commission to the public health threats posed by these pathogens and to the need to assess their risks to public health.

Risk related to zoonotic diseases is only a part of the more general risk of food-borne infection, which is itself a part of the general domain of food safety. The distinguishing feature of a zoonosis is the infection of the living animal. This should lead to a specific approach directed to the source, to prevent or minimise animal infection and thereby the contamination of the

food chain in order to protect humans from subsequent infection. Human to animal transmission of infection can occur via direct contact or indirectly when animals have access to human faecal material. For example, the practice of using human sewage sludge as fertiliser on farms poses a risk of transmission of disease if codes of good practice are not adhered to.

Some food-borne zoonoses may occur independently of the commercial circuits either because zoonotic cycles involve wild animals or because the food contaminated by animal-derived organisms comes from family production or is collected in nature. In particular aspects of human behaviour such as hunting or hiking can result in an increased risk for such zoonotic infections.

Some food-borne zoonoses may occur independently of the consumption of meat and of products of animal origin, since in some cases animal-derived (micro) organisms contaminate other types of food (*e.g.* vegetables or fruits), for instance through contaminated irrigation waters or biological fertiliser. These zoonoses have also been taken into account in this report when they were considered of public health relevance.

Antimicrobial resistance is also of relevance in relation to zoonoses. Zoonotic bacteria may develop acquired resistance to various antimicrobial agents and thereby represent a health concern in addition to their virulence properties. Furthermore, bacteria that are not zoonotic themselves, *e.g.* indicator bacteria, may harbour transferable resistance genes that can be transferred to pathogenic bacteria and through this mechanism contribute to compromised therapy in the patients. Such transfer of resistance genes can occur across ecological and phylogenetical borders, and man and animals share a common genetic pool of resistance genes. The dynamic exchange of resistance genes in the microbial world implies that the problem of antimicrobial resistance should also be addressed in a zoonotic perspective. Hence, the approach in relation to prevention and control measure as well as in relation to monitoring and surveillance should be holistic covering both humans, food and animals. Based on this, the Committee supports the Scientific Steering Committee opinion of 28 May 1999 that future monitoring of antimicrobial resistance in selected bacteria would be scientifically justified. In this report however, due to time constraints and to the fact that the question of antimicrobial resistance has already been addressed by the Scientific Steering Committee, the issue is not further developed.

Travel within the EU, and more particularly travel to the developing countries could represent an important factor of zoonotic agent acquisition. While all EU recommendations should apply to all imported foodstuffs, it is hardly foreseeable that the EU standards or equivalent will be followed in third country on a global scale. EU citizens should therefore be informed of potential risks of zoonotic disease when travelling. Moreover, surveillance and monitoring systems should be open or extended to the third countries to provide the travellers, their physicians and the regulatory authorities with comprehensive information. Hence, the risks of acquiring zoonotic disease by travelling abroad must be considered significant.

The true economic losses associated with a disease may be described as a function of its constraint for economic (such as trade, production and consumption) and human (such as travel) activities. One proxy measurement for these total costs under certain assumptions (perfect information about the disease and its control) is the costs of clinical cases together with the current expenditure on control measures (Howe, 1997 and McInerney, 1997). The control of zoonotic diseases could also be seen as a necessary condition for the good functioning of a modern society and in particular for the markets in foodstuffs.

Morbidity, mortality due to zoonotic infection as well as the chronicity of some of these diseases, may have an economic impact on the welfare of the society. In addition, indirect costs such as crisis management or prevention of diseases need to be evaluated. The economic impact however, of chronic diseases or infections that have unfortunate sequellae, might be much higher than what is indicated by the numeric incidence. The benefits and costs of zoonotic disease control should be therefore assessed having regard to these aspects.

The Committee however, did not analyse the travel and economic issues further, since these were outside the terms of reference for this report.

### 3.3. A potential for improvement in food safety

The significant increase of food-borne disease caused by zoonotic bacteria seen in all EU countries during the period from 1980 to 2000 does suggest that improvements are needed. However, the uncertainties of disease incidence measurements at present and in the past should result in a cautious approach when using this line of arguments. Therefore the need and potential of improvement should not relate to whether it is possible to return to the disease level of 1980 through appropriate measures. The most important factors to guide actions to improve the zoonotic food safety situation should be a) the magnitude of the problem, b) the nature of the problem and c) the potential for change.

The magnitude of the human health problems related to the most important zoonotic food-borne hazards is difficult to assess accurately, but European Union statistics for the most important diseases do exist (see Chapter 5). It is likely that many human infections go unrecorded with patients failing to present to health services, or if they do, no laboratory diagnosis is made or the laboratory diagnosis is not reported centrally. The cases reported may only represent the severe end of the spectrum of the disease. Therefore, the Committee concludes that there is an underreporting of human disease incidence with regard to food borne zoonoses. Despite this underreporting, it appears that the magnitude of these human health problems is significant.

The focus on food safety, and especially the microbiological food safety, is not a new issue for the European food producers or food control authorities. The focus on good hygiene through all the production and preparation stages and the efficient, science-based use of physical (pasteurisation or cold-chain) or chemical (food additives) principles to kill or prevent growth of pathogenic micro-organisms have contributed significantly to food safety in general. However, most of these food safety management tools were general in nature, and therefore a

more focused control of the relevant hazards was introduced through systems such as HACCP.

Despite these improvements in production methods and general hygiene the food safety problems have increased. There are very little data on the true incidence of zoonotic diseases in humans in the Member States. For most zoonotic diseases, only a small fraction of the cases are diagnosed and/or reported, and this with large differences between the Member States. However the trends recorded as well as the growth rate of the problems seem to be within the same range in all EU Member States. The presumed large differences in food control efforts between different Member States do not seem to have resulted in measurable differences in the human health outcome (See Chapter 5). The effect of the traditional food control measures seems to have been insufficient in relation to the recent food-borne disease increases.

A chronology of food scares, including *Salmonella*, verotoxigenic *E coli* O 157, *Trichinella*, has damaged consumer confidence in the safety of the food supply and the ability and commitment of both the food industry and the regulatory authorities to protect that supply. To protect public health and restore confidence it is necessary to assess the risks of zoonotic pathogens, and to introduce appropriate risk elimination, or if not possible risk reduction strategies. Before these can be introduced, it is important to identify the factors and practices contributing to the spread of zoonotic agents so that interventions can be targeted appropriately. New production systems in the primary production as well as in the manufacturing sector are likely to have had an influence. Other changes in the food production chain from farm to table, including changes in kitchen habits at the consumer level have also been mentioned in this context, as have increases in food trade and tourism.

There is a clear realisation that these problems should be seen in the context of the full farm to table continuum. Likewise there is a new emphasis on the human health outcome, *i.e.* the risk, as the operative descriptor of the food safety problems. These conceptual changes could be used to orientate the management or control efforts in new directions. This would include:

(i) allocating primary effort as close to the source as possible,

(ii) redirecting and if necessary revising, old inspection and control routines to focus on the relevant pathogens of major public health importance,

(iii) using risk assessment to ensure the best scientific basis for risk management decisions,

(iv) monitoring and surveillance and correlating food prevalence and disease incidence to guide and review risk management efforts.

The potential for significant improvement in the present control and inspection procedures exists, and a number of countries outside the EU are already initiating some of these changes. It is likely that such changes are important prerequisites for the control of zoonotic food-borne diseases within the EU.

### 3.4. Selection of zoonotic agents

Since it was not possible to review all zoonotic diseases, the Committee concentrated on a subset of all zoonoses. Factors influencing the inclusion of diseases in this report included public health priority, relevance to most Member States, sufficient data for assessment and emerging threats to consumers' health. The Committee focused on agents responsible for the majority of foodborne zoonotic diseases.

The following factors were considered to select the most important agents:

- human incidence based on the Community reports on trends and sources of zoonoses in the EU (1994-1998)
- severity of illness (based on expert judgements)[3]
- epidemiological trend: the long term changes in disease incidence in humans or pathogen prevalence in food or animals (based on expert judgements)
- emergence: new or reappearing potential threat to public health (based on expert judgements)

On the basis of these criteria and the consensus opinion of the working group, the Committee identified the agents mentioned below which will be addressed in this report:

Bacteria: *Campylobacter sp., Listeria monocytogenes, Salmonella sp., Verotoxigenic Escherichia coli* (VTEC)

Parasites: *Cryptosporidium sp., Echinococcus granulosus / multilocularis, Trichinella spiralis*

For most viruses, the current evidence indicates a person to person transmission, directly or indirectly through food and water, but without animal reservoirs. For this reason, viral food-borne diseases are considered not to be zoonoses and therefore outside of the current mandate of the Committee. Food and water borne pathogenic viruses are therefore not addressed in this report. However, the Committee draws the attention of the Commission to the public health problem of viral food and water borne infections and proposes that the risks to public health be assessed. It should be noted that caliciviruses have been the most frequently diagnosed food borne viral infections (Vinje *et al*, 1997 and Codex Alimentarius document CX/FH/99/11) in some EU Member States.

A summary for each of the selected zoonotic agents is presented in the report (Chapter 6).

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[3] It should be noted that a zoonotic disease in an immuno-compromised person might be much more severe than the course of the same disease in an immuno-competent person.

A more detailed description and assessment of the zoonotic agents is presented in the annex. However, for *L. monocytogenes* the Scientific Committee on Veterinary measures relating to Public Health has already adopted an opinion on 23 September 1999, and therefore only reference to this opinion will be made. For *T. spiralis* a report is being prepared by an *ad hoc* working group of the Scientific Committee on Veterinary measures relating to Public Health and therefore it will not be developed further in annex.

## 4. NEW STRATEGIES IN FOOD SAFETY

The supply of safe food is a necessary condition for a functioning modern society (Bloom and Canning 1999, Bloom and Mahal 1997, Schwabe, 1984) and for economic development. Increasingly the diets of EU residents include ingredients from all over the world. The need to protect public health has made the control of food-borne pathogens a persistent topic on the public agenda in both developed and developing countries. During the last 150 years the introduction of pasteurisation of milk and compulsory meat inspection has represented milestones in the advancing food safety and public health improvements. Pasteurisation exemplifies a risk management intervention that has a high efficacy of removing most pathogens from milk and milk products without any prior identification of pathogens. Provided the pasteurisation process is working, no recontamination occurs, and the food is kept cool, pasteurised food should be generally safe.

Meat inspection is a risk management measure based on identification of contaminated carcasses and removal of those identified. The meat inspection procedure has a relatively high diagnostic sensitivity for trichinellosis, echinococcosis and tuberculosis and a high efficacy in reducing the incidence of these diseases. For other food borne pathogens such as *Salmonella, Campylobacter, Listeria monocytogenes* or verotoxigenic *E. coli* (VTEC) the traditional meat inspection procedures has an insignificant diagnostic sensitivity. Thus, these procedures do not contribute to a significant lowering of risks related to these pathogens.

The traditional microbiological procedures applied on a sample of products or animals cannot verify the absence of pathogens in a food batch or an animal population. However, the microbiological procedures do enable probabilistic statements concerning the prevalence of food borne pathogens in an animal population or a food batch. For each pathogen additional pieces of information about the epidemiology, the detection limits for the diagnostic procedures used, the ability of the agent to grow in the food under given temperature and storage conditions and the infectious dose (Haas, 1983) are available. The infectious dose concept means that the number of bacteria in food and the probability of this number causing disease in humans can be correlated. In addition, information about the intended use of the foodstuff, its storage temperature and period throughout the food chain is also available. The infectious dose varies with the different pathogens. In particular, the margin of error is reduced for those such as VTEC with a low infectious dose. Other factors including host susceptibility and virulence of the pathogen, will in addition to the actual number of pathogens ingested determine whether a person becomes ill or not.

It is possible to combine these pieces of information to obtain a probability of a foodstuff containing less than the infectious dose of a pathogen at the point of consumption. This is the basis of the new strategy in food safety represented by the concepts of Food Safety Objectives (FSO), Hazard Analysis Critical Control Points (HACCP) and Epidemiological Intelligence (EI).

A food safety objective (FSO) is a novel risk management concept where an acceptable level of pathogens in a foodstuff at the point of consumption is set, possibly derived from the infectious dose and a safety margin. A FSO could be given a probabilistic interpretation *e.g.* 99% of the foodstuffs should have less than the FSO stated.

HACCP is a structured systemic approach that can be used to achieve the food safety objectives by identifying hazards and measures for their control. A HACCP procedure is implemented on each production or processing establishments. WHO has published guidelines for HACCP (WHO, 1995 and WHO 1998). HACCP is based on 7 principles:

– hazard analysis
– identification of critical control points
– establishment of critical limits at each control point
– corrective action
– record keeping
– monitoring
– verification.

HACCP can be applied across the entire food chain, however usually these plans are applied independently at individual parts of the chain, *e.g.* the food processing plant or the food retailer. The objectives of the HACCP approach are either determined by statutory requirements such as FSO or due diligence considerations of the operator. It follows from the laws of probability, that everything else being equal, a HACCP with attention to the critical control points along the food chain will afford equal or better protection of the consumer. On the other hand, fewer critical control points increase the likelihood that the plan is implemented. Very simplified and assuming that all CCP have similar efficacy (e), this concept can be summarised in the formula:

$$\text{Probability food unit unsafe} = P \times (1-e)^{n}$$

Where P denotes the probability of a food unit being unsafe without any HACCP applied and n denotes the number of critical control points along the food chain.

An optimal HACCP would cover continuously the food chain from the feed and farm to the point of consumption, and thus the HACCP approach should apply to all stages such as slaughter, food-processing, retail and catering. An important CCP is the nature of raw ingredients entering the food chain covered by a HACCP program, hence the need for epidemiological intelligence related to the raw material such as both animal feed and animals.

EI (Schwabe, 1984) could be described as activities aimed at one or more of 4 objectives:

– to collect and analyse information with the purpose to detect relevant changes in disease incidence (in humans) or prevalence (in food and animals);

– to provide the baseline information for risk assessments;

– to enable decision makers to make informed risk management decisions;

– to evaluate the effects of risk management interventions.

It follows from these objectives that EI should be an integral part of any disease control or risk management effort. Important tools for EI are monitoring and surveillance[4] also referred to as MOSS activities (Nordhuizen *et al.*, 1997). To provide the desired EI, the monitoring and surveillance activities should analyse the disease occurrence with regard to time, place, individual and other putative risk factors.

Moreover, when designing an EI system it would be useful to distinguish between emerging and classical zoonoses since the monitoring and surveillance activities will differ. In the first case the emphasis will be on human disease, while for the classical zoonoses one should consider the whole food chain. For the emerging zoonoses the monitoring and surveillance system should be co-ordinated at the Community level since the emergence will appear more clearly at the highest level of aggregation. In addition the number of human cases may be low and it may only be by monitoring and surveillance across all Member States that a detectable incidence will be identified. For the classical zoonoses one should in addition monitor the prevalences at each segment of the food chain where trade occurs.

Definitive laboratory diagnosis using standard methods and protocols is essential if the results of monitoring and surveillance in animals, food and humans would have to be comparable across Member States. It would also facilitate the timely identification of problems and assist with monitoring and surveillance of the effectiveness of interventions.

Ensuring a continuous epidemiological intelligence is an integral part of the risk management of zoonoses.

## 5. DATA SOURCES

For exposure assessment within the risk assessment process a qualitative and/or quantitative evaluation of the likely intake of zoonotic agents via food should be made. For this purpose, the Committee has evaluated data on the prevalence of the selected zoonotic agents in animals and their products as well as consumption data. These data originate from different sources and include prevalence of micro-organisms in food and the effect of processing and food handling operations on them, data on food production and consumption patterns as well as the incidence of human

[4] Monitoring and surveillance are used interchangeably and sometimes as synonyms and the definitions appear to have changed over time. Moreover, some authors talk about active (surveillance or monitoring) versus passive monitoring. Because of this overlap, the Committee decided to use the words "monitoring and surveillance" in its report.

diseases. Limited information is available on the severity of human cases and dose-response data.

In this report emphasis was put on the data to determine if foodborne transmission plays an important role in the aetiology of disease and which foods are implicated. Due to a lack of data, the level of microorganisms in the food at the time of consumption was not evaluated. Prevalence and incidence data were used as far as possible based on the reporting system provided for in Council Directive 92/117/EEC; however, data from this system do not sufficiently reflect present trends and there is a clear lack of comparability between data from different countries (see 5.1). Specific networks established as scientific projects on *Salmonella*, verotoxigenic *E. coli* and *Campylobacter* are described. Furthermore, some published literature has been included.

In many Member States there is no integrated approach within countries because monitoring and surveillance of feed, animal health, contamination in foodstuffs and human health is undertaken by different government agencies. In addition many of the zoonotic pathogens do not cause animal disease and therefore data on their prevalence is not collected in animal health programmes *e.g.* verotoxigenic *E. coli* O157 and *Campylobacter*. Furthermore, the investigations of human gastroenteritis to identify a pathogen and to determine the sources vary between Member States.

A short description of the data sources is given, including the main objective of the activity, the way of funding, the zoonotic agents covered, temporal and spatial coverage.

## 5.1. Data collected under the provisions of Council Directive 92/117/EEC

Council Directive 92/117/EEC (Zoonoses Directive) provides for the yearly reporting from all Member States of the EU on the epidemiology of various zoonoses, *e.g.* tuberculosis due to *Mycobacterium bovis*, brucellosis and the agents thereof, salmonellosis and the agents thereof, trichinellosis, campylobacteriosis, echinococcosis, listeriosis, rabies, toxoplasmosis, yersiniosis, and other zoonoses and the agents thereof, in the Community. Furthermore, on a voluntary basis, verotoxigenic *E. coli* O157 has been included into this reporting system from the beginning. No information is collected on *Cryptosporidium*, viral zoonoses apart from rabies nor viral food borne infections.

These national reports cover information on the occurrence of the zoonotic agents in animals, food and feed since 1994. Furthermore, data on human incidence of zoonoses is collected routinely.

The reporting network ought to enable the authorities to evaluate the reasons for sporadic human cases and outbreaks, to compare the development of zoonoses, to develop regional strategies for the prevention of diseases spreading to other regions, as well as to determine the need for control activities in specified regions.

For most aspects some data are available from the Member States. The main problem is that the data provided are not comparable as the system lacks harmonised monitoring and surveillance schemes as well as standardised

methods for diagnosis and characterisation. Furthermore, although the Community Reference Laboratory on the Epidemiology of Zoonoses (CRL-E) provides for the formats in which the data should be reported, not all Member States comply.

Another problem is the timeliness of the reports. Member States have to report until the end of May of the following year, but most reports are provided later.

Data given in the Annex II.2 should mainly be used as an indication for the presence of the pathogens in animal species and foodstuffs and for temporal trends within a Member State instead of means for comparing prevalences between Member States.

One should distinguish between different notification systems when interpreting the data on human incidence given in Annex II.3. For most zoonotic agents the information provided by the Member States may not detect relevant changes at each step in the food production chain on the prevalence of zoonotic agents, due to lack of precision and possible biases. Emerging zoonoses can not easily be detected since the sentinel systems are few and the data is not collated on European level. Therefore risk management decisions within the EU cannot be fully based on sound scientific knowledge and the effect of implemented control measures can not be evaluated precisely.

### 5.2. The EU human communicable disease network

Decision N° 2119/98/EC[5] sets up a network for the epidemiological surveillance and control of communicable diseases in the Community entering into force in the beginning of 2000. The objective of this Decision is to promote co-operation and co-ordination between Member States with a view to improving the prevention and control of communicable diseases specified. The network should be used for the epidemiological monitoring and surveillance of these diseases and an early warning and response system for the prevention and control of these diseases. Two decisions 2000/57/EC[6] and 2000/96/EC[7] have been taken concerning the implementation of a rapid alert system and covering surveillance matters. Hence, collation and analysis concerning the incidence of communicable diseases in the European Union should commence during 2001.

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5 Decision N° 2119/98/EC of the European Parliament and of the Council of 24 September 1998 setting up a network for the epidemiological surveillance and control of communicable diseases in the Community (OJCE L268 of 3.10.98, p1)

6 Commission Decision N° 2000/57/EC of 22 December 1999 on the early warning and response system for the prevention and control of communicable diseases under Decision N° 2119/98/EC of the European Parliament and of the Council (OJCE L21 of 26.1.2000, p32)

7 Commission Decision N° 2000/96/EC of 22 December 1999 on the communicable diseases to be progressively covered by the Community network under Decision N° 2119/98/EC of the European Parliament and of the Council (OJCE L28 of 3.2.2000 p50)

## 5.3. Enter-net

Enter-net is an EU wide network for the surveillance of human *Salmonella* and verotoxigenic *Escherichia coli* (VTEC) infections. The key professionals directly responsible in every EU country are participating, usually the microbiologist in charge of national reference laboratory services and the epidemiologist conducting public health surveillance at a national centre, while there is limited communication with the food and veterinary authorities.

The network has been funded by DG XII (now Research DG) as a Research Concerted Action since it began in 1994. For the first three years the collaboration concentrated upon improving *Salmonella* surveillance and was called Salm-net. In 1997 with a further grant from DG XII the network was extended to include surveillance of VTEC infections. From 2000 the Health and Consumer Protection DG funds the core international surveillance activity of Enter-net as a part of the Commission's response to the communicable disease Network Decision.

Enter-net has created international databases for both salmonellosis and VTEC infection in man. However, the VTEC surveillance is in its embryonic state.

The database on human *Salmonella* isolates has some biases and limitations. The proportion of the total of laboratory-confirmed human *Salmonella* isolates that has been reported to Enter-net varies enormously between countries. Most cases reported to the National Reference Centre (NRC) are incorporated into the international database. Broadly speaking two categories of countries can be described, those from which most laboratory-confirmed infections are reported to the NRC and those from which a minority of the infections are reported. Moreover, it should be recognised that the number of laboratory-confirmed isolates does not indicate the full impact of human gastro-intestinal infections within a country as only a fraction of cases have stool specimens examined in a laboratory.

In the recent past, outbreak recognition and the efficiency of investigations in the EU have improved, and national monitoring and surveillance has been strengthened. The network functions as an alert system through rapid enquiries to all participants when an unexplained outbreak is recognised in one of the Member States. Concerted research has produced a European phage-typing scheme for the principal *Salmonella* serotypes.

The Enter-Net project has potential to contribute to control initiatives if the data collected is integrated with the monitoring and surveillance undertaken in both animals and food.

Further details are given in annex III.

## 5.4. Campynet

The network Campynet was established on the 1st October 1998 to harmonise and standardise molecular typing techniques for *C. jejuni /coli*. The network is funded by the European Commission for 3 years and formally comprises

eleven countries. A reference set of strains will be established, standard operating procedures and data handling will be recommended. In a second step these technologies will be transferred to all participating laboratories.

As *Campylobacter* infections are one of the most frequent causes of bacterial diarrhoea in humans in the European Union, it is necessary to have standardised diagnostic and typing techniques to develop effective monitoring and surveillance, and to understand the epidemiology of this pathogen. Moreover, this would contribute to comparable data on *campylobacter* incidence and prevalence.

Further details are given in annex III.

### 5.5. Eurechinoreg/EchinoRisk

A concerted European approach to the study of alveolar echinococcosis (AE), a relatively rare but very severe zoonotic disease present in most countries of northern Europe, seemed typically adapted to add value to any action in this field. The European Commission (DG V) thus supported in 1998 an appropriate pilot project to set up a formal network from teams, otherwise informally linked by bilateral projects and occasional meetings. The aims of the pilot project were:

(1) To collect reliable epidemiological and clinical data on AE cases in humans, in countries of the EU where the disease is endemic or suspected to become endemic.

(2) To collect reliable epidemiological data on adult stages of the parasite in definitive animal hosts, and of the larval stage in intermediate hosts in the same countries.

(3) To set up a network for epidemiological surveillance and elaborate an agreed European system for case definition and staging.

(4) To promote a better information on the disease, its prevention and its treatment

(5) To facilitate international staff exchanges and training of physicians, surgeons, veterinarians, PhD students and post-doctoral researchers.

The network involves 10 teams from 8 EU countries and sentinel centres in countries of central Europe at the border of the EU and in Turkey. The network is multidisciplinary in nature and associates teams dealing with human as well as animal epidemiology.

The pilot programme has achieved a series of goals:
(1) the infrastructure of a network has been established,
(2) actions have been taken to set up national reference centres,
(3) a common definition of AE cases and a common staging system (PNM) have been elaborated and evaluated,
(4) updated maps of endemic areas have been drawn, and
(5) new trends in the incidence of human cases and animal infection have been clearly disclosed.

After the pilot project, the teams involved in the network and some additional teams have set up a common project of research (EchinoRisk) within the 5th Framework Programme "Quality of life and management of living resources" to go on registering cases and studying environmental, genetic and behavioural risk factors.

### 5.6. Consumption data

(1) In the Annex II.1 an estimate of the amount of animal derived food consumption in the European Union is given. EUROSTAT (Statistical Office of the European Communities) uses uniform rules to collect all statistical data from the National Statistical Institutes of each of the 15 Member States.

(2) Additionally, as an example, data are given from one country. A German study conducted ten years ago provides some detailed information on the frequency of intake of the main animal derived foods and daily intake (mean value) of the amount of these items. This study is based on a representative sample of the population. Information on the frequency of food intake was collected by a standardised questionnaire answered by one person per household. Additionally all members of the household had to record their food intake in detail for a period of 7 consecutive days.

(3) Finally, although at present no food consumption data can be generated from this activity, the COST Action 99 - a research action on Food Consumption and Composition Data - should be mentioned for his future importance**.** It is a continuation of EUROFOODS (established in 1982) and the EUROFOODS-ENFANT Project (1990-1994) of the FLAIR-Program of the European Union and is working towards improving quality and compatibility of data on food consumption and composition in COST countries.

A literature review showed that some consumption data are available from other Member States for several foods of animal origin too. In summary, consumption habits have changed over the years and are different for the Member States, regions of Member States, ethnic and/or vulnerable groups. Furthermore, they are influenced by other factors such as age, socio-economic class, urban/rural profile, religion or fashion, or, at short term, by specific events, like the BSE crisis. Consumption data are necessary for formal risk assessments of food borne diseases.

## 6. SUMMARIES OF THE 7 PATHOGENS/ZOONOSES SELECTED

A thorough description, including risk assessment data and references is presented for *Campylobacter*, *Salmonella*, VTEC, *Cryptosporidium* and *Echinococcus* in Annex I. For *Listeria monocytogenes* and *Trichinella spiralis* see Chapter 3.1.

## 6.1. Campylobacter

### *6.1.1. The pathogen and the animal hosts*

*Campylobacter* are Gram-negative rods. However, actively dividing cells have a characteristic slender, curved or spiral shape and are highly motile. In older cultures the spiral forms may change into coccoid forms. In general, *Campylobacter* sp. do not grow in conventional culture systems, but require specific supplements and an atmosphere containing 5-10% oxygen. *Campylobacter jejuni* (*C. jejuni*) and *Campylobacter coli* (*C. coli*) are distinguished from most other campylobacters by their high optimum growth temperature (42°C), hence the term "thermophilic". *Campylobacter* will hereafter refer to thermophilic *Campylobacter*.

The principal reservoir of pathogenic *Campylobacter* sp. is the alimentary tract of wild and domesticated animals and birds and *Campylobacter* is commonly found in broilers, fowls, cattle, pigs, wild animals and birds, and in dogs. *Campylobacter* has also been isolated frequently from surface water, rivers, and lakes, where it is introduced by sewage and faeces from wild animals and birds. In water and other environments with sub-optimal growth conditions, *Campylobacter* may convert into 'viable but non-culturable' forms, which seem to survive longer. It is still debated whether such forms are still virulent or if they can reverse into a culturable, virulent state after passage through a host. In animal husbandry *C. jejuni* and *C. coli* seem to have favoured reservoirs: *C. jejuni* is predominantly associated with poultry and *C. coli* is predominantly found in pigs. In animals *Campylobacter* sp. does not seem to cause disease problems.

### *6.1.2. Human disease and disease incidence*

*Campylobacter* causes an acute enterocolitis in humans, which can not clinically be distinguished from enteric illness caused by other pathogens. The incubation period may vary from 1 to 11 days, typically 1-3 days. In most cases the diarrhoea is self-limiting and may persist for up to a week, but relapses do frequently occur. In rare cases, *Campylobacter* has been shown to cause Guillain-Barré syndrome (GBS), a serious nerve disorder resulting in paralysis. A few percent of campylobacteriosis cases will also develop reactive arthritis. Few deaths are related to *Campylobacter* infections. Reduced susceptibility of *C. jejuni* to antimicrobial agents has emerged in human populations and especially fluoroquinolone resistance may cause severe problems in the future in cases where drug treatment is required.

*C. jejuni* and to a lesser extent *C. coli* is a major cause of diarrhoeal illness. Disease from thermophilic *Campylobacter*, primarily the two species mentioned above, has an incidence rate comparable to or even higher than *Salmonella* in many countries. The number of confirmed cases of human campylobacteriosis is registered in twelve EU Member States and the reported incidence rate per 100.000 inhabitants vary widely, *i.e.* from 9.5 in Spain to 108 in Scotland in 1997. Probably a major explanation is the differences in monitoring and surveillance systems, implementation of diagnostic methods and way of reporting.

Most *Campylobacter* infections occur as sporadic cases, but larger outbreaks have been described. The incidence of sporadic cases seems to have a seasonal variation with a peak in the summer. It might be noted that in the UK there is a peak in the middle of May, while in the Nordic countries the highest incidence is in July/August. Moreover, some regions can have a higher incidence than the rest of the country.

### *6.1.3. Prevalence and ecology in food*

Since faeces content will inevitably contaminate the meat during slaughter *Campylobacter* will be present on a fraction of slaughtered carcasses. For pork and beef there is a decline in the concentration/prevalence during the slaughter process, primarily due to dehydration from forced chilling procedures. In poultry the same dehydration does not seem to occur and a prevalence decline is not observed.

*C. jejuni* and *C. coli* have optimum for growth at 42-45°C and do not survive cooking or pasteurisation temperatures. They do not grow below 30°C and survive poorly at room temperature, *i.e.* they do not multiply in food stored at temperatures up to +30°C. Although their viability declines during chill and frozen storage, they may persist under these conditions for prolonged periods.

The reporting systems show that especially poultry meat is contaminated with *Campylobacter* (reported prevalences up to 85%). *Campylobacter* has also been found in beef, pork, other meat products, raw milk and milk products, and in fish and fish products at lower prevalences (typically a few percent). A seasonal variation has been observed in the prevalence in poultry meat at the retail level with the highest prevalences in summer and the lowest in winter. *Campylobacter* is also found in surface and raw drinking water.

### *6.1.4. Management options in place*

For poultry at farm level the establishment of hygiene barriers for each poultry house seems to be the only preventive option which has been shown to work in practice until now. The use of all in and all out is important. The success of this approach is indicated as in Sweden around 60% of the poultry farms consistently produce batches of broilers without *Campylobacter*. Furthermore, in Sweden the flock prevalence has been reduced from 50% to 10%. For other production animals no specific farm level risk management options are in place.

For poultry several options have been tried at slaughter-level in order to reduce the contamination level in scalding and chilling water and on the broiler carcasses, but none of these techniques have shown a satisfactory result. For other animals processes involved in slaughter and secondary production, and especially the use of forced air cooling, seem to reduce the level of contamination and the risk of cross contamination.

The maintenance of the cold chain throughout the production and retail system, as well as hygienic measures to prevent cross-contamination can at least contribute to not increase the problems throughout the chain.

### *6.1.5. Future Management options and potential for measuring their effect*

The occurrence of waterborne outbreaks seems to be important primarily in areas using surface waters as the primary drinking water source. The prevention of faecal (animal or human) contamination of such sources is important, however the importance of 'viable but non-culturable' forms with increased survival potential should be further investigated.

The efficiency of establishment of strict hygiene barriers at poultry farm level should be documented. In general the efficiency of procedures to lower the prevalence of *Campylobacter* at farm level needs further scrutiny as it appears that significant reductions of *Campylobacter* prevalence of broilers is possible based on the Swedish experience.

The effect of a general change in the meat inspection procedures related to the avoidance of faecal- and cross-contamination should be investigated.

The effect of different food treatments and preservation techniques on *Campylobacter* sp. survival and death should be further investigated with a view to optimise such processes.

The implementation of procedures to avoid cross-contamination together with procedures that will ensure sufficient heat treatment to eliminate *Campylobacter* sp. should be promoted. The use of decontamination procedures for poultry carcasses have also been considered and the use of such procedures have been dealt with in the SCVPH report on benefits and limitations of antimicrobial treatments for poultry carcasses of October 30, 1998. Education and information should focus on correct handling and storage of foods at appropriate temperature and the risks associated with cross contamination. In addition the risks associated with ingestion of undercooked foods and contaminated drinking water should be stressed.

A primary prerequisite for measuring the effect of such management options is a monitoring and surveillance system allowing for an assessment of prevalence in the food and disease incidence. Moreover, a practical sub-typing system for the relevant strains would be needed to separate the effects according to animal species origin (pathogen accounting). Added value would come from the harmonisation and scientific validation of the case definitions, sampling, laboratory and reporting procedures, and the collation and analysis of these data at the Community level, with a Community dissemination of the results.

### *6.1.6. Research needs*

Knowing the reasons for the increased incidence of human campylobacteriosis could give important clues for controlling the disease as a zoonosis.

An improved elucidation of the causes of the infections is needed, including further research into the natural reservoirs of the microorganism. An efficient subspecies typing system is needed for isolates from the environment, production animals, food and patients. The development of reliable and

workable quantitative methods for enumeration of *Campylobacter* in food should be encouraged. The potential for reducing the *Campylobacter* prevalence in food by reducing the prevalences in production animals and by optimising production processes during slaughtering and food processing should be explored. The importance of fluoroquinolone resistant strains of *Campylobacter* should be assessed.

## 6.2. Listeria monocytogenes

### *6.2.1. The pathogen and the animal hosts*

This summary is based on the report from the SCVPH of September 22, 1999.

*Listeria monocytogenes* is widespread in nature and can be found in soil, foliage and the faeces of animals and humans.

*L. monocytogenes* is a Gram-positive, facultatively anaerobic, non-sporeforming rod. Subtyping data together with epidemiological evidence may indicate that some strains are more pathogenic than other for humans. Out of 24 outbreaks reported in literature since 1966 14 outbreaks (58%) and around 40% of the cases (1359/3338) were attributed to serovar 4b, while serovars 1/2 a, b were attributed to 8 outbreaks and 11% (385/3338) of the cases. None of the (sub-)typing methods can be used to discriminate pathogenic from non-pathogenic or less virulent strains. Therefore, all *Listeria monocytogenes*, including those present in food, should be regarded as potentially pathogenic.

### *6.2.2. Human disease and disease incidence*

*Listeria monocytogenes* infections most frequently result in meningitis, with or without septicaemia, or septicaemia alone. Immuno-compromised individuals are particularly vulnerable. In pregnant women listeriosis may produce a self-limiting flu-like illness. In many instances this infection spreads to the foetus producing a disseminated infection resulting in miscarriage, stillbirth, or prematurely birth of a gravely ill child. Although the disease can be treated with antimicrobial drugs the use of these agents is not always successful. Three recent documented foodborne outbreaks of listeriosis include many cases where the presence of high levels of *L. monocytogenes* has resulted in the rapid onset of symptoms of vomiting and diarrhoea with few apparent cases of the more classical infection.

Because of the long incubation periods (1 to 90 days) bacterial isolates are rarely available from the left over food suspected in cases of listeriosis. In those instances where bacterial isolates are available, the levels of *L. monocytogenes* detected both from unopened foods and from food remnants obtained from the patients have usually been high ($>10^3$/g). This feature together with the limited data on the recovery of the organism from foods implicated in illness support the likelihood of a high infectious dose for infection through food. However, considerable caution is required because of the small number of cases where information is available and the likelihood of wide differences in susceptibility to infection between individuals because of

their immune status. The possibility of infection from low numbers of *L. monocytogenes* especially among the immuno-compromised cannot be discounted.

The human incidence of listeriosis appears to be between 1 and 15 reported cases per million per year based on internationally published incidence data.

While the annual incidence of human listeriosis is low (1-15 cases reported per million inhabitants), the case fatality rate (the proportion of cases that die) is reported being between 20 and 40%. In immuno-compromised individuals the reported case fatality rates may approach 75%. Hence, listeriosis appears as an infrequent but serious public health threat in particular for high risk groups such as elderly, immuno-compromised persons (*i.e.* cancer, transplant, HIV, rheumatic, diabetic, or chronic alcoholic patients) and pregnant women.

Four factors might result in an increased incidence of listeriosis in the future: a) The increasing proportion of susceptible people be it due to old age or immuno-suppressive treatments and/or diseases (this proportion is estimated at 25% in the EU Member States). b) The increased use of cold stored ready-to-eat foods where there is prolonged time intervals (weeks, months) between processing and consumption. c) That listeriosis has appeared with diarrhoeal expression only. d) *L. monocytogenes* occurs in the environment as well as in production systems and related environments.

### *6.2.3. Prevalence and ecology in food*

The prevalence in food animals seems to be between 1-10%. Some investigations seem to show that *L. monocytogenes* can establish itself within a slaughterhouse, meat, dairy or fish processing factories. *L. monocytogenes* can create a biofilm on stainless steel surfaces and can be isolated from equipment, cold stores and floors. Hence food receiving a heat treatment during production can become contaminated post-heating in the production environment. Experience from production plants show that some others can function without *L. monocytogenes* problems while comparable plants have continuing problems.

Some general trends can be derived from a cross-section of published data from Europe as well as the rest of the World during the last decades:

- *L. monocytogenes* prevalence in ready-to-eat products is well documented in many countries. The food groups most often investigated are poultry meat, meat products, salads, raw milk and dairy products and fish products.

- Quantitative data are scarce and when presented, low numbers (<100 *L. monocytogenes*/g) are often reported.

*L. monocytogenes* is a psychrotroph pathogen and is capable of growth at refrigerator temperatures. The minimum pH for growth in foods is 4.6-5.0. *L. monocytogenes* can grow under aerobic, micro-aerophilic, and anaerobic conditions, and in vacuum. It appears to be capable of survival on meat regardless of treatments such as freezing, surface dehydration, and simulated

spray chilling. Growth is highly dependent on the temperature, pH and type of meat, as well as background micro-flora. Poultry meat supports growth better than other meat products. Growth of *L. monocytogenes* on cold-smoked cod, cold-smoked salmon, crab meat, cooked shrimp, and cooked crawfish tail meat stored at 4-10 °C has been observed.

*6.2.4.* Management options in place

The traditional cooling-chain concept does not prevent the growth of *L. monocytogenes.* The focus has therefore been on the prevention of contamination of ready-to-eat products. The finding that some production plants can function without *L. monocytogenes* problems seem to show that good production hygiene can prevent/minimize problems.

For products receiving heat treatment, focus is on preventing post-heat treatment contamination. For other products, raw materials with a low prevalence of *L. monocytogenes* can contribute to a better final product. In some production units, efforts are made to reduce/eliminate *L. monocytogenes* colonisation of production environment. Some Member States give specific advice to susceptible consumer groups, and experience indicates that information campaigns directed at pregnant women can have an effect through change in diet.

*6.2.5. Future management options*

- A general food safety objective (FSO) should be to keep the concentration of *L. monocytogenes* in food below 100cfu/g at time of consumption.

- The grouping of foods according to *L. monocytogenes* growth potential and the setting of relevant *L. monocytogenes* limits (FSO) according to food groups, *i.e.* lowering the limits to absence in 25g at the time of production for vulnerable foods as a preventive measure.

- The consideration of appropriate temperature and storage time combinations for vulnerable food groups.

- The finding that some production plants can function without *L. monocytogenes* problems while comparable plants have recurrent problems underline the necessity of improvements in production hygiene. HACCP should be geared to reduce/eliminate *L. monocytogenes* colonisation of production environment.

- It is relevant to give specific advice to susceptible consumer groups.

*6.2.6. Research needs*

- The effect of FSO initiatives mentioned in 6.6.5 should be evaluated through monitoring and surveillance investigations of food, especially including quantitative investigations, as well as efficient monitoring of human listeriosis.

- The potential for real time monitoring for *L. monocytogenes* at the production line should be considered.

- Technological changes in food production and food storage regimes should be evaluated with regard to *L. monocytogenes* prevalence and growth.

- Further research should be directed towards control of house strains in food production facilities.

Experimental data on *L. monocytogenes* growth are lacking for a number of specific commodities. This information is needed also to support predictive model estimations of growth potential.

## *6.3. Salmonella*

### *6.3.1. The pathogen and the animal hosts*

*Salmonella* sp. is member of the family *Enterobacteriaceae* and consists of Gram-negative, oxidase negative bacteria, with small rod-shaped cells, straight-sided and not exceeding 1.5µm in width. Most *Salmonella* sp. are motile with peritrichous flagellae. Members of the genus are responsible for diseases of humans and animals. The degree of host adaptation varies and affects the pathogenicity for humans in three ways: 1) Serotypes adapted to humans, such as *S. typhi* and *S. paratyphi*, usually cause severe diseases with septicaemic-typhoid syndrome (enteric fever) and these serotypes are not usually pathogenic to animals. 2) The common serotypes, such as *S. typhimurium* and *S. enteritidis* cause usually foodborne gastrointestinal infections of varying severity. 3) The serotypes which are highly adapted to an animal host such as *S. abortus-ovis* (sheep), *S. gallinarum* (poultry), *S. cholerae-suis* (pigs), and *S. dublin* (cattle) may produce no, mild or serious disease in humans.

The not host adapted serotypes are those of principal zoonotic significance.

The principal reservoir of the common *Salmonella* sp. is the gastrointestinal tract of mammals and birds. *S. enteritidis* and *S. typhimurium* are the serotypes most frequently associated with eggs or poultry and other farm animals, respectively. Animals infected with the non-host adapted *Salmonella* sp. are usually asymptomatic carriers. Some of them, however, may exhibit clinical signs of low or moderate severity. *Salmonella* sp. may also be isolated from clinically healthy cold-blooded animals such as little turtles or other reptiles kept as house pets, from dogs and cats, from wild birds and from invertebrates such as snails and cockroaches. *Salmonella* sp. are able to survive and under certain conditions, maybe even multiply in the external environment and water.

### *6.3.2. Human disease and disease incidence*

Infections with the ubiquitous *Salmonella* sp. are characterised by febrile gastro-enteritis, *i.e.* diarrhoea, stomachache, fever, headache, nausea, vomiting and malaise. The first symptoms appear 12-24 h after infection and

usually continue for about 3-5 days (range 2-7 days). In a few percent of the cases invasive disease develops outside the intestine *e.g.* septicaemia and infections of the internal organs, bones and joints. Some of these complicated cases are fatal. Complications like reactive arthritis and persistent abdominal symptoms (diarrhoea, constipation and abdominal pain) can occur after the acute phase of disease. Strains with reduced sensitivity to antibiotics are commonly detected in farm animals and the human population. Their relative proportion to other *Salmonella* sp. is increasing and their spectrum of antibiotic resistance is extending, lately including fluoroquinolone resistance.

Human salmonellosis is the zoonotic disease with highest reported incidence in most European countries. The reported incidence rates per 100.000 inhabitants in 1998 ranged from 1.9 in Portugal to 135.7 in Belgium. Although a large proportion of the observed variation is accounted by differences in monitoring systems, diagnostic methods and way of reporting, a considerable proportion may be due to different habits in food preparation and consumption and the prevalence in foods in the Member states.

### *6.3.3. Prevalence and ecology in food*

During the current slaughtering process a percentage of the carcasses is directly or indirectly contaminated with contents of the gastrointestinal tract of slaughtered carrier animals. In addition to faecal contamination of carcasses, *Salmonella* can be transmitted to humans via eggs. Eggs can become contaminated either by transovarian (*S. enteritidis*) or transshell transmission. However any food may become contaminated with *Salmonella* if cross-contamination is permitted at any stage of the food chain from the abattoir, dairy plant or egg processing plant to the point of consumption.

Fresh poultry meat (*Gallus gallus*) is frequently found contaminated (reported prevalences at retail ranging from 1 to 55% in 1998). At lower prevalences *Salmonella* sp. are also detected on pork, beef, in other meat products and in raw eggs and dairy products. Recently, alfalfa sprouts have also been found contaminated in several European markets indicating the importance of manure contaminated produce as a vehicle for human salmonellosis.

*Salmonella* optimum growth occurs at 37°C, with lowest reported temperatures at around 5°C. The upper temperature limit for growth is around 45°C. The heat resistance increases markedly at low $A_w$ levels particularly in foods which also have a high fat content. The *Salmonella* concentrations decline during frozen storage, the rate being greater at temperatures around the freezing point of meat (-2°C to -5°C). The pH for optimum growth is between 6.6 and 8.2, with values above 9.0 and below 4.0 being usually bactericidal. A minimum growth pH of 4.05 has been recorded but depending on the acid used to lower it the minimum may be as high as 5.5. Regarding available moisture, growth inhibition has been reported for $A_w$ values below 0.94 in media with neutral pH, with higher $A_w$ values required as the pH is decreased towards growth minima.

### *6.3.4. Management options in place*

Feed production control and feed heat treatment are essential for preventing *Salmonella* sp. entering the farm. If the farm can receive feed free from *Salmonella*, the probability of maintaining the farm free from *Salmonella* improves. In particular the implementation of HACCP in feed manufacturing including the end point verification has been instrumental in achieving the improved *Salmonella* status in primary production seen in some EU Member States.

For all animals, the establishment of on-farm good manufacturing practices (*e.g.* all-in all-out production, cleaning and disinfection between successive batches) and introduction of hygiene barriers seem to be effective in controlling the infection cycle in the majority of farms. In addition, in domestic fowl production the efficient control of *Salmonella* sp. in all parent-animal flocks reduces the prevalence of the organism at production stage *e.g.* turkeys, ducks, broilers, and layers.

In some EU Member States trade in livestock from flocks or herds positive for *Salmonella* is restricted.

The use of vaccination and competitive exclusion has been helpful in reducing the *Salmonella* prevalence in broiler and layer flocks, while the use in breeding herds is of more doubtful value.

At the slaughterhouse, prevention of carcass contamination with faeces is improved by covering of the bungs with a plastic bag the moment the anuses are cut loose. In addition, slaughtering of infected animal populations at the end of the day or at a different slaughterline favorably affects the prevalence of contaminated carcasses. The use of decontamination procedures for poultry carcasses have also be considered and the use of such procedures have been dealt with thoroughly in the Scientific Committee on Veterinary measures relating to Public Health report on benefits and limitations of antimicrobial treatments for poultry carcasses of 30 October 1998.

The slaughterhouse monitoring and surveillance is a critical point for *Salmonella* control in some Member States. Slaughterhouse samples (*e.g.* meat-juice samples, microbiological samples) are routinely collected, following statistically determined sample sizes, and tested by ELISA or isolation methods. The extent of this monitoring and surveillance varies between Member States. Results are used to identify infected animal populations or to classify the animal populations to prevalence categories and apply appropriate control measures on farm and at slaughter. Furthermore, the microbiological results are used to estimate the prevalence of infected meat and meat products, the sources of infection and the pathogenic strains involved.

The maintenance of the cold chain throughout the production and retail system, as well as hygienic measures to prevent cross-contamination can contribute to a situation where the problems are not increased throughout the chain.

### *6.3.5. Future management options*

The main effort should be directed towards the development of strategies to control the infection in farm animal populations by breaking the on-farm cycles. These strategies should include three components. (1) The farm animals should be examined with diagnostic tests that will accurately detect on-farm infections with the serotypes of highest human significance. (2) The implementation of sets of control measures in those farms that have an unacceptable prevalence of infection. (3) The introduction of feed controls ensuring that the feed used on the farm is free from *Salmonella*. The strategy should also include provisions for pinpointing farms infected with strains with reduced susceptibility to antibiotics. Samples collected at the slaughterhouses can be the basis for these strategic programs. At the same time attention should also be given to the uniform incorporation of steps in the slaughtering process intentionally designed to reduce the hazard of carcass contamination.

Education of food handlers and of the general public should focus on correct handling, cooking and storage of foods. For uncooked food one should avoid contamination and ensure hygienic handling, while cooked foods should be adequately cooked and protected from contamination.

The risks posed by the use of manure and recycled sewage and slurry for fertilizing vegetables and berries should be investigated. The possible control options ought to be investigated, since a substantial growth is foreseen in organic farming. The potential of irrigation water as a source of salmonellosis should also be investigated.

The use of probiotics and competitive exclusion in order to lower the *Salmonella* prevalence in primary production should be further investigated.

### *6.3.6. Research needs*

The development and evaluation of accurate diagnostic techniques for the detection of the infection in the live animals is the cornerstone of any preventive action combined with standardised definitive typing methods. These techniques should be validated and uniformly applied in all Member States throughout the food chain and allow for the establishment of large scale monitoring and surveillance schemes. A comprehensive set of control measures for the on-farm cycles of salmonellosis should be developed for all farm animals. This can only be achieved through more thorough understanding of the on-farm epidemiology of the infections with the serotypes of highest human significance.

## 6.4. Verotoxigenic *Escherichia coli* (VTEC)

### *6.4.1. The pathogen and the animal hosts*

VTEC is a group of *E. coli* that produce verotoxin. This group of bacteria has many synonyms the most common one being shigatoxin producing *E. coli* (STEC) while the term enterohaemorrhagic *E. coli* (EHEC) is used interchangeably, resulting in some confusion. In this report the term VTEC will be used. Disease produced by VTEC appears to be associated with a

subset of strains with the serotype O157:H7 as the predominant one. A lot of other verotoxin producing serotypes may also produce disease in humans, the most common serotypes being O26, O103, O111, and O145. However, not all VTEC are associated with human disease(see also Annex I). Most research on VTEC has been done on the serotype O157 that is easily recognisable among other *E. coli* strains by its inability to ferment sorbitol. All other VTEC serotypes are phenotypically similar to the harmless *E. coli* strains inhabiting the gastrointestinal tract of humans and all warm-blooded animals. This means that our knowledge about the disease caused by and the sources of non-O157 VTEC are rather scarce and inadequate. VTEC O157 appears to have ruminants as its reservoirs, but it has also been isolated from pigs, dogs, cats, horses, sea gulls and geese. The VTEC O157 bacteria appear to survive for months on straw, wood surfaces and in water.

### *6.4.2. Human disease and disease incidence*

The clinical manifestations of VTEC in humans range from symptom-free carriage, diarrhoea, haemorrhagic colitis (HC), haemolytic uraemic syndrome (HUS) thrombotic thrombocytopenic purpura (TTP) to death. Haemorrhagic colitis is often associated with abdominal cramps, bloody stools, but seldom fever. The average period between exposure and illness period is 3 days, while most patients recover within 7 days. The diarrhoeal illness may be biphasic typically starting with abdominal cramps and diarrhoea the first few days, which after a short phase of recovery might become bloody during the next 1-2 days. Especially in children the disease may progress into HUS typically 6 days after onset of diarrhoea. Among the patients with HUS a few percent die acutely and some of the survivors develop end-stage renal disease. However, in outbreaks among elderly, the mortality could be up to 50%. In humans VTEC O157 can be shed in the stool for several weeks after the resolution of symptoms. While the bacteria do not appear to cause disease in adult ruminants, neonatal calves show clinical symptoms (diarrhoea and enterocolitis) if ingesting VTEC O157:H7. HC and HUS appear to be more common after infections with VTEC O157 than with non-O157 VTEC. However the proportion of cases of HC and HUS caused by non-O157 VTEC may be of the same magnitude or even higher than with the VTEC O157 infections, since the non-O157 VTEC infections might be more frequent.

The Community incidence in 1997 was 7 VTEC cases and 1 HUS case per million inhabitants, *i.e.* a total of 1912 cases of VTEC infections and 316 HUS cases, with geographical variations, *e.g.* in Scotland the VTEC incidence was close to 100 per million. However, the diagnostic habits in the laboratories vary a lot between the Member States and even within each Member State. The monitoring systems have different diagnostic sensitivities. Thus, it is difficult to compare the incidence of disease caused by VTEC between the Member States. The dominance of the serotype VTEC O157 in some northern and central European countries is contrasted by a reporting of other serotypes associated with HC and HUS in the Mediterranean countries.

### *6.4.3. Prevalence and ecology in food*

The risk factors for human exposure are linked to either direct or indirect exposure to and ingestion of faecal contents from ruminants or humans; this exposure can be minuscule given the infectious dose is possibly as low as 10 bacteria. The exposure can be food-borne through undercooked meats such as hamburgers, unpasteurised milk and contaminated salads, berries, sprouts and fruits. Another source is cross-contamination from contaminated raw meat. Several Japanese outbreaks were associated with radish sprouts indicating sprouts as a risk food since the bacteria, possibly originating from biological fertiliser such as manure or slurry from sewage treatment, can multiply during the sprouting process. In principle four routes of infection could be identified: person to person, food-borne such as raw meat, unpasteurised milk, contaminated fresh produce or drinking water, environmental such as swimming in a contaminated lake or swimming pool, and direct contact with farm animals. The Community report on trends and sources of zoonotic agents in animals, feedstuffs, food and man in the European Union in 1998 (SANCO/409/2000 rev2 FINAL) seems to indicate prevalences of VTEC O157 in cattle herds of 10% or more, and in individual animals around 1% or more, while in beef or minced meat the prevalence is 0-1%. However, since only the serotype VTEC O157 is reported on, a serious information bias in the scientific body of knowledge is introduced against non-O157 serotypes.

### *6.4.4. Management options in place*

Few specific risk management interventions have been put in place regarding VTEC in EU Member States. Because of some outbreaks in the USA receiving a high level of press and public attention, the VTEC O157:H7 is also known as the 'hamburger-bacterium'. Hence, one of the important preventive measures receiving most attention has been the heating of minced meat to ensure a core temperature above 72°C for 2 minutes *e.g.* Irish recommendations.

Another preventive measure is to ensure that consumers only drink pasteurised milk, since outbreak investigations have implicated unpasteurised milk several times.

Some Member States have introduced standard procedures to regulate hygienic production conditions as well as storage conditions (cooling) of sprouts.

### *6.4.5. Future Management options*

- Manure handling

Manure should be disposed of in such a way that neither drinking water nor growing vegetables, fruits, berries nor products thereof, (foreseen consumption without heat treatment) could be contaminated directly or indirectly by effluents from the manure disposal.

- Direct animal contacts:

Farm visitors, in particular children handling calves and people visiting cattle pastures should be advised to wash their hands before eating. In Sweden, children under 5 years old are advised to not visit cattle herds during the summer season.

- Animal management and handling

The grouping of calves appears to be a critical phase for spreading VTEC O157 between calves in the primary production.

It is possible that feeding could alter the VTEC shedding of and tolerance of acidity of the VTEC from infected calves and cattle.

- With regard to transport, slaughter, secondary processing the Scientific Veterinary Committee's report of 1997 recommends the following:

– clean animals when sent for slaughter

– better transport conditions of slaughter animals

– a review of dressing and evisceration process (reference is made to the SCVPH report on meat inspection procedures of February 24, 2000).

– hygiene and cold chain maintained throughout the food chain to avoid cross-contamination

– decontamination of carcasses if needed

– education of food safety for persons working with food

- Milk

A labelling procedure should inform about the risks of drinking unpasteurised milk.

Children and elderly being the most susceptible groups should be adviced not to drink unpasteurised milk.

- Food at retail and catering

The proper heat treatment of meat preparations such as hamburgers or steaks or roast beefs would eliminate this route of transmission for verotoxigenic *E. coli*.

The avoidance of any possibility for cross-contamination of ready to eat foods from raw meats should be a priority.

Fruit juice produced from fallen fruit should be pasteurised

- Home - person to person transmission

Patients suffering from VTEC infections should be advised not to prepare food for others;

People visiting or working on farms should wear appropriate protective clothing.

Children with bloody diarrhoea should not be allowed into swimming pools.

### *6.4.6. Research needs*

The research needs include:

- identification of the clinical importance of non-O157 VTEC
- improvement of the diagnostic methods for all VTEC-serotypes
- identification of host specific factors in the VTEC pathogenesis,
- identifying the reservoirs of all VTEC of clinical importance
- quantifying the importance of different transmission routes *i.e.*, surface water and environment,
- harmonised diagnostic procedures in humans, food and live animals,
- the impact of calf management and feeding on shedding of the bacteria
- the impact of transport and slaughter practices, and
- predictive models for the survival of virulent VTEC bacteria.

## 6.5. Cryptosporidium

### *6.5.1. The pathogen and the animal hosts*

Among *Cryptosporidium* species, *C parvum* is of public health concern. It is an obligate intracellular coccidian parasite that carries out its parasitic lifecycle in one host. The reservoir hosts for human infections are ruminants, primarily cattle and sheep. Following the ingestion of thick walled oocysts, these encyst in the small intestine and free sporozoites penetrate the microvilli of the host enterocytes where mature zygotes develop. Oocysts are developed from these fertilised zygotes and are subsequently released in the faeces. The infection is spread to other hosts when the oocysts are ingested. The infectious dose is low and water contaminated with ruminant faeces presents the greatest public health risk.

### *6.5.2. Human disease and disease incidence*

Infection of humans usually occurs as a diarrhoeal illness three days to one week after ingestion of oocysts. Most cases have a prolonged but self-limiting course of disease. However, in immuno-compromised patients the illness can be life threatening. The disease is globally distributed with an annual incidence in developed countries of < 1 to 4.5 % and a much higher incidence in developing countries. Infections peak in children between one and five

years of age. There is a lesser peak in adults of 20-40 years. Maternal and acquired immunity and opportunity for exposure to the oocysts contribute to the varying rates of infection between age groups and geographical regions, respectively. There is no monitoring and surveillance at EU level.

### *6.5.3. Prevalence and ecology in food*

Human infections arise primarily from drinking contaminated water but swimming or other recreational activities in contaminated water can also lead to infections. Fresh produce irrigated or washed with contaminated water is a possible vehicle for the organism and this may be an important mode of transmission in traveller's diarrhoea. Person to person spread is an important mode of transmission particularly in situations where hygiene is poor.

Oocysts can survive in the environment for several months in cold moist conditions. The oocysts are resistant to most chemical disinfectants especially chlorine containing compounds and ozone used in the treatment of drinking water. Oocysts are sensitive to desiccation requiring moisture for survival. They are sensitive to heat and are readily destroyed by pasteurisation temperatures.

### *6.5.4. Management options in place*

There are a number of approaches for control and management to prevent or reduce the level of contamination. These include: protection of the catchment areas of water sources from animals, improvement of drinking water treatment, and the implementation of hygiene practices and potable water during harvesting, washing and packaging of fruits and vegetables,

Minimising the risk of water contamination is the cornerstone to the control of this zoonosis. The lower the quality of the source waters the greater the reliance on water treatment. Efficient filtration is the only effective way to remove oocysts. The performance of filtration plants should be monitored continuously and treated water of constant quality should be produced irrespective of the quality of the raw water. Effective management of water supplies to isolate particular reservoirs and the use of safer sources during periods of high risk *i.e.* high rainfall, storms are important.

Immuno-compromised persons need to be aware of the risks of contaminated water and that the public water supplies cannot always be guaranteed safe. For the control of human infections associated with drinking the public water supply or the management of identified contamination, protocols on when to issue, and subsequently lift, advise on boiling water for drinking and other control measures should be developed by the public health officials.

Strict regimens should be in place to prevent and control contamination of water in swimming pools. Those caring for vulnerable groups such as infants and the elderly need to be aware as this organism, like other enteric pathogens, can spread from person to person if hygiene practices are not optimum. Direct contact with animals presents a risk and adequate supervision of children and hand washing should be ensured if this transmission route is to be interrupted.

Effective monitoring and surveillance in humans and animals, and the monitoring and surveillance of water supplies are essential if problems are to be identified early, preventive initiatives introduced and their effect monitored.

A forum must be developed in each Member State where those working to control this pathogen can share experience and ideas and develop strategies in risk management. This forum should include water supply managers, water engineers, veterinarians, microbiologists, epidemiologists, public health administrators and other relevant professionals.

*6.5.5. Future management options*

Eradication in the ruminant populations both domestic and wild ruminants is not a feasible option. However proper management of young animals may prevent clinical disease and reduce the amount of oocysts being shed.

The introduction of more efficient methods of filtration can reduce the risks of contaminated water entering the public supply.

Effective monitoring and surveillance in animals and humans is necessary to establish the burden of disease in humans, the important animal reservoirs and the evaluation of control measures. A consistent and harmonised approach to diagnosis and monitoring and surveillance is required across Member States if meaningful comparisons are to be made.

Use of hygienic practices by food handlers at all stages from farm to fork can be introduced, such as pasteurisation of juices and irradiation of products.

*6.5.6. Research needs*

Current methods do not allow determination of whether oocysts present in drinking water are viable or infectious.

Improved methods of oocysts removal are needed that can cheaply treat large volumes of water.

Improved methods to identify *Cryptosporidium* sp. in foods are needed as is the assessment of the risk to public health of the presence of *Cryptosporidium* in food.

Molecular typing of strains of *C. parvum* should improve the understanding of the epidemiology of this pathogen.

## 6.6. Echinococcus sp.

*6.6.1. The pathogens and the animal hosts*

*Echinococcus* (E.) sp. are helminth cestode parasites. As many parasites, *Echinococcus* sp. are characterised by a cycle which involves final hosts and intermediate hosts, each harbouring different stages of the parasite life. Two species of parasitic organisms of the genus *Echinococcus* are known to occur in Europe, namely *E. multilocularis* and *E. granulosus*, causing two different

chronic diseases, alveolar echinococcosis (AE) and cystic echinococcosis (CE), respectively.

*Final hosts*, carnivores, host the "*adult" form* of the parasite: adult worms (3-6 mm) live in their small bowel, and hundreds of microscopic eggs are dispersed daily with the faeces of the carnivore and may contaminate any water or food which is not boiled/cooked before human consumption.

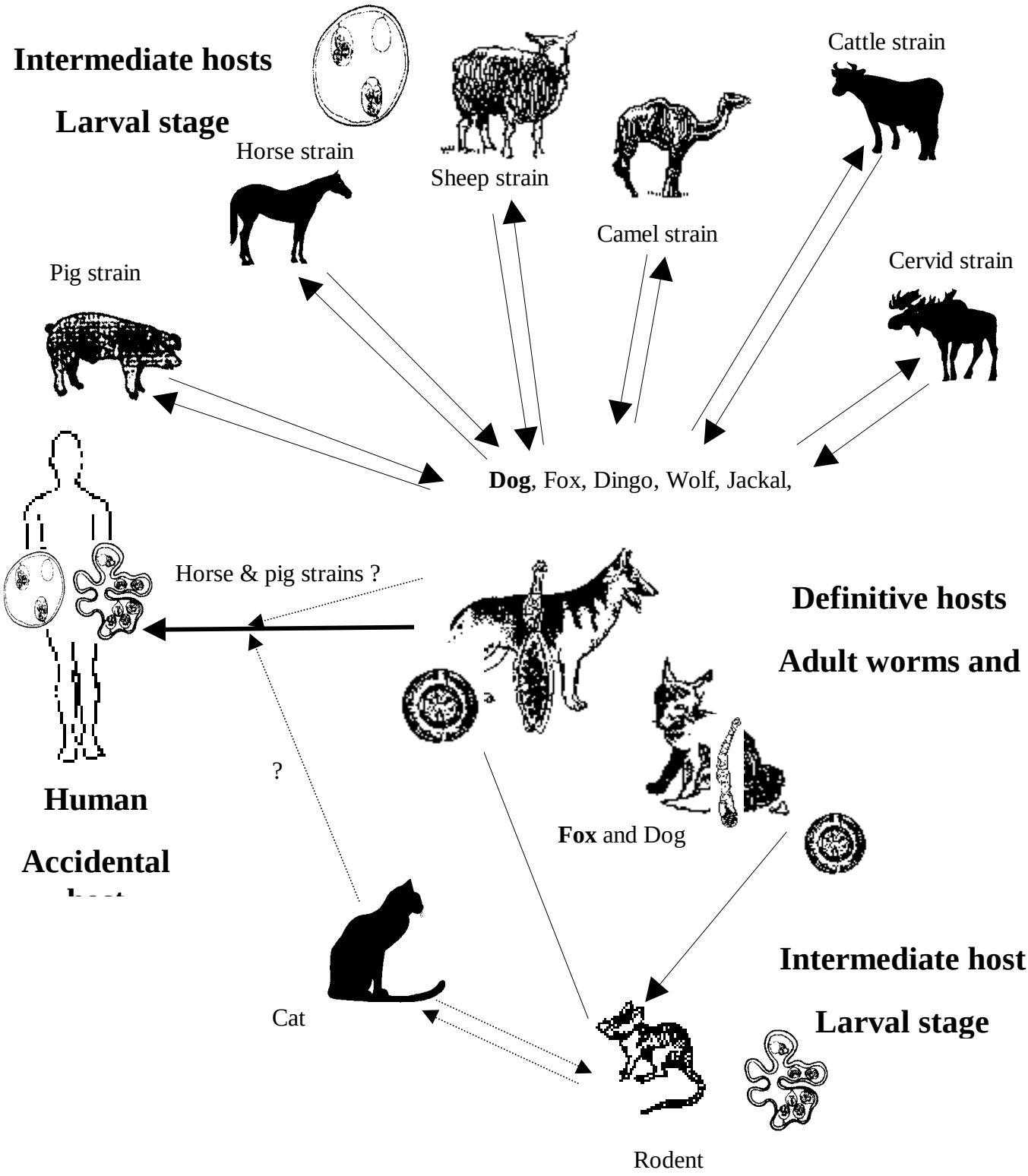
*Intermediate hosts* host the "*larval" form* of the parasite, also called "*metacestode*". It constitutes a cyst filled with fluid, well separated from the surrounding host tissues, in CE, and a tumour-like continuously growing polycystic and fibrous mass, in AE. Parasitic cysts become fertile by giving rise to the particular form which will be able to re-create the adult form in the final host, the "protoscolex" which will transform into an adult worm when eaten by a carnivore.

The cycle of *Echinococcus granulosus* in Europe is predominantly domestic involving dogs as final hosts and sheep, cattle, reindeer, pigs and horses as intermediate hosts. Wild animals can occasionally be involved in the cycle. In endemic areas, cysts are found in prevalences of 1-40% in cattle and 1-80% in sheep at the abattoir, while the prevalence in dogs might be up to 50% dogs that might contaminate humans. The lowest prevalence is observed in those EU countries with developed control campaigns. However, the highest prevalences are currently found in Central European countries bordering the EU where the considerable increase observed in the past 10 years is of major concern for the future. The risk factor for human exposure is represented by direct and indirect contact with faeces from the final hosts. Children are often found infected, because of their closer contacts with dogs or with environments/foods polluted by dog faeces.

The cycle of *E. multilocularis* in Europe is predominantly sylvatic involving red foxes as final hosts and rodents (voles) as intermediate hosts. In some countries dogs and cats have also been identified as final hosts. The prevalence of *E. multilocularis* infection in foxes ranges from 15% to 70% in these endemic areas. Recent trends are represented by an increase in the area of distribution and in percentage of infected foxes, presence of infected foxes in big cities and a newly recognised infection of dogs and cats. This could lead to major changes in the populations at risk in the near future.

Life cycle of the various strains of *E. granulosus*

*Ref. : J. Eckert, R.C.A. Thompson. Acta Tropica, 64 (1997) 19-34*




Life cycle of *E. multilocularis*

*Ref. : B. Gottstein. Clinical Microbiology Reviews, July 1992, 248-261*

### *6.6.2. Human disease and disease incidence*

Both *Echinococcus* sp. diseases are characterised by their very specific geographic distribution, due to the particularities of the parasitic cycles and of human behaviour that lead to contamination. Typically, *E. granulosus* infection/cystic echinococcosis occur in Southern/Mediterranean Member States of the EU and border countries, nearly only imported cases occurring in Northern countries. Conversely, *E. multilocularis* infection/alveolar echinococcosis occurs in northern Member States of the EU and border countries; with the exception of Turkey where both infections/diseases are observed. In addition marked regional differences may occur, which gives very limited value to incidence/prevalence data expressed at a country level.

Cystic echinococcosis in humans behaves as a benign tumour, unique or multiple, in the liver or lungs, in most of cases; however any tissue or organ may be involved, including brain, bone, spleen, and kidney. After a silent asymptomatic period various symptoms and signs are observed, depending on the primary location of the cyst(s). Rupture of the cyst may lead to life-threatening anaphylactic shock and to dissemination to many tissues and organs. Treatment is represented by surgery, interventional radiology (identification, puncture and sterilisation of the cysts), and benzimidazole drugs. Depending on the considered area, annual incidence of CE human surgical cases in endemic countries ranges from 1 to 20/100 000 inhabitants.

Alveolar echinococcosis in humans presents, in most cases, as a liver tumour, which mimics a cancer, progressively invading bile ducts and liver vessels and leading to numerous complications. Metastases may occur, especially in the lungs and in the brain (12% at the time of diagnosis); the latter location seems to be favoured by immuno-suppression. Until the beginning of the eighties, the disease was fatal within 5 years in most cases. Because of earlier diagnosis and better medical management (including surgery, continuous treatment with albendazole, and ultimately liver transplantation in some cases), the prognosis and quality of life has improved a lot in most of patients. This improvement of the patients' condition was associated with a considerable increase in economic cost because of the medical treatment for life, of the cost of major operations including liver transplantation and of a necessary regular follow-up. In regions endemic for AE, a 1 to 20/100 000/year incidence is observed when the rural population at risk only is considered, despite an overall very low prevalence at the country level.

### *6.6.3. Prevalence and ecology in food*

It must be stressed that humans are not infected through meat or other animal products from the intermediate hosts (*i.e.* livestock in CE). Slaughtered animals serve as reservoir for the infection of dogs that can contaminate any food (including water). The eggs are very resistant to environmental conditions between –70° and +70°, and especially are not destroyed by freezing; theoretically a single cyst containing protoscoleces may infect a carnivore, and a single egg may infect intermediate hosts including humans.

### *6.6.4. Management options in place*

Cystic echinococcosis is theoretically an eradicable disease, but numerous factors are involved in the maintenance of the cycle, including behavioural and cultural factors that are more resistant to regulations than mere facts.

Inspection of cysts at the abattoir is currently performed and control measures have been implemented in most of the EU countries.

Nevertheless, many control programmes have failed, and the disease remains a threat to human health in some countries of the EU, and in most of the border nations of central Europe.

Alveolar echinococcosis is not eradicable, because of the sylvatic cycle. Control approaches have been rather scarce until now. Basic recommendations concerning consumption of raw berries, fruits and vegetables collected in nature or in non-fenced kitchen gardens are given to the populations at risk.

### *6.6.5. Future management options*

An efficient management programme for cystic echinococcosis includes the following measures:

- Control of stray dogs, registration of owned dogs and education of dog owners for the proper feeding and preventive treatment of the dogs.
- Testing with arecoline or coproantigen test of dogs in the infected areas ;
- In control programmes the treatment with praziquantel or an equivalent drug of all dogs in infected villages with hydatid cysts at least 3-4 times every year but preferably every month (and appropriate destruction of the stools, since praziquantel does not kill the infectious eggs).
- The regular use of praziquantel or an equivalent drug in baits to treat stray dogs and foxes ;
- Control of movements of food animals and dogs from the infected areas to the “clean” ones;
- Marking and control of movements of animals from infected flocks or herds.
- Strict measures to prevent illegal slaughter
- Fencing of kitchen gardens (family and commercial) to prevent any access of dogs (or other canids) to vegetables for human consumption; control of stray dogs, especially around outside market facilities
- Education of the public; mass screening in the population of endemic areas: especially using ultrasound exams, it may be a part of education campaigns.

- Vaccination of flocks is now available and could be a part of control measures

For alveolar echinococcosis a pilot project has been designed and implemented in an endemic area of Southern Germany, using baiting of foxes with praziquantel. Only preliminary results are available, suggesting that the approach is feasible; however a full efficacy would need the treatment of foxes on a large area, as has been achieved for rabies vaccination, and frequently repeated campaigns of treatment.

Current lack of knowledge on the exact circumstances of contamination, and new epidemiological trends, including potential contamination by urban foxes and by pets, require more research studies at the EU level in order to adapt control and health education.

Harmonisation of case reporting at the EU level is an urgent need, since the official notification of echinococcosis, either for human or animal cases, is different in the various EU Member States, and available data are not comparable. In addition, CE and AE are not clearly differentiated, although the diseases and the management options are different. Finally, it must be stressed that the diseases are apparent in humans only months and even years after contamination. For CE, effects of management options can be measured by the prevalence of cysts observed in food animal viscera at slaughtering, by the level of infection in dog faeces using newly developed tools (copro-antigen detection), and by the incidence of human cases, in hospitals or through mass screening. For AE, the monitoring of control programmes can only be achieved by systematic registration of human cases. Because of the severity of this disease and the apparent emergence of cases in new areas of the EU and border countries, continuous and systematic monitoring and surveillance is mandatory, and should be co-ordinated at the European level.

### *6.6.6. Research needs*

Research needs include characterisation of *Echinococcus* sub-species differences in pathogenicity; studies on immuno-genetic characteristics of the susceptible populations; and multicentre evaluation of therapeutic options. The transmission routes and the ecological and behavioural factors involved in the *E. multilocularis* cycle in nature and in human contamination should be studied at the European level, using standardised laboratory and sociological tools and common sampling strategies in order to adapt risk management to the current situation.

## 6.7. Trichinella sp.

### *6.7.1. The pathogen and the animal hosts*

*Trichinella* sp. are helminth nematode parasites, still endemic in most countries of the EU and in Central Europe border countries. In the EU, four species of *Trichinellae* are found:

- *Trichinella spiralis*, the etiologic agent of domestic trichinellosis,
- *Trichinella britovi*, the etiologic agent of sylvatic trichinellosis in the temperate parts of the EU,
- *Trichinella nativa* in the coldest and arctic parts of the EU, and
- *Trichinella pseudospiralis*, sporadically reported in Spain and recently in France.

The adult (intestinal *Trichinella*) and the infective larva (muscle *Trichinella*) occur within a single host (auto-heteroxeny); there is no free-living stage. Muscle larvae are considered to be infective from day 15 post-infection in pigs, and occur as lemon-shaped 0.3-0.8/0.2-0.4 mm in size cysts, in striated muscles and especially in so-called predilection sites (diaphragmatic, intercostal, masseter muscles and the tongue). Infection occurs when flesh containing muscle *Trichinellae* is eaten by animal or human hosts.

*In the sylvatic habitat*, trichinellosis affects carnivores with cannibalistic and scavenger behaviour; the main reservoir of the disease is the red fox, although in Finland the racoon dog is also a reservoir ; mustelids and other carnivores may also serve as hosts but have only a secondary role. In the sylvatic habitat wild boars represent the main source of infection for man.

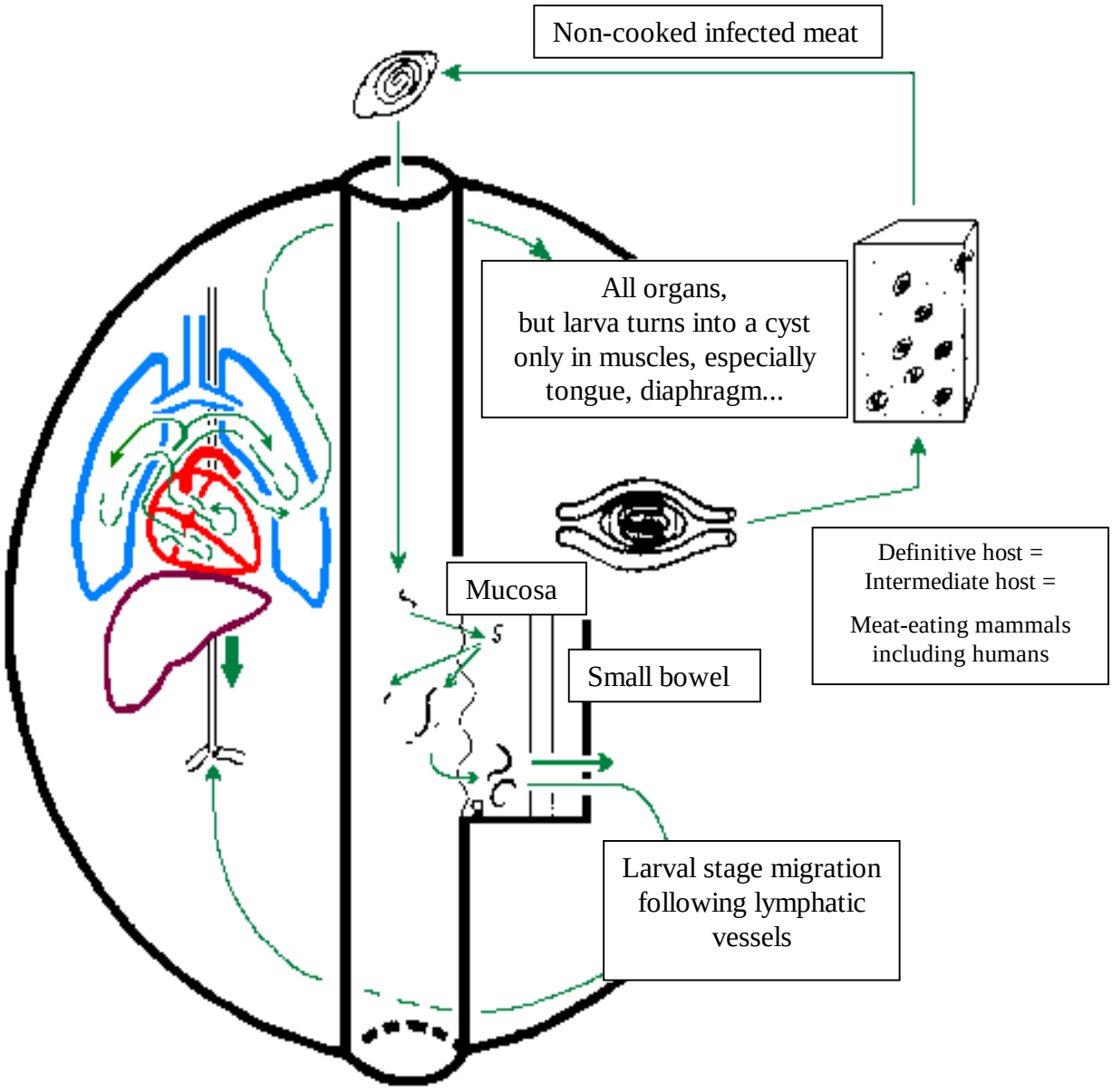
*In the domestic habitat*, the main sources of infection are domestic pigs for man in areas where traditional pig-rearing practices still prevail, and synanthropic rats for animals. Wild boars are more infected in regions where domestic trichinellosis is present, especially if traditional pig-rearing practices are associated with poor sanitary conditions resulting in the creation of small rubbish dumps containing *Trichinella*-infected pork waste near farms and village where wild boars can easily gain access.

Most of the human outbreaks observed in the EU in the past 20 years were due to consumption of *Trichinella*-infected horsemeat. This unusual host may have been infected by adding pig meat to horse diet or feeding horses with hay pellets containing rodent remnants, in countries where sylvatic (USA and Canada) and domestic (Central Europe border countries) trichinellosis are highly prevalent.

Sylvatic *T. britovi* and *T. nativa* may invade, and *T. spiralis* may return to domestic reservoirs when humans fail in the management of wildlife and domestic animals. For example by pasturing domestic animals (pigs and horses) in remote wild areas or by feeding domestic animals with remains of sylvatic animals.

## Evolution cycle of Trichinellosis

*Adapted from ANN'OFEL*



### *6.7.2. Human disease and disease incidence*

Trichinellosis in humans is a parasitic febrile myositis acquired by the consumption of raw or undercooked meat containing infectious larvae. Fever, myalgia, face oedema, and blood hypereosinophilia are the main symptoms and signs. Although most cases recover rapidly, a small percentage can be lethal in the absence of appropriate treatment, and chronic sequellae (mostly neurologic) may occur in some cases. Specific anti-helminthic drugs, such as mebendazole and albendazole, are efficient for treating trichinellosis, if the treatment is initiated early enough; chronic lesions may become resistant to any treatment. In the past 25 years, human trichinellosis due to the consumption of local domestic animals has not been reported in Austria, Belgium, Denmark, Finland, Great Britain, Ireland, Luxembourg, Portugal, Sweden or the Netherlands. However, 36 outbreaks of trichinellosis in the EU Member States have been published from 1966 to 1999, 20 were reported in the last decade and mainly in Spain. This is an underestimation of the number of real cases, since mild cases are usually misdiagnosed as flu, and most outbreaks are not reported in international journals; for instance, only 6 among the 19 outbreaks that occurred in France between 1975 and 1998 were reported in indexed journals. In 1998, a survey carried out amongst the EU representatives of the International Commission on Trichinellosis identified 10 outbreaks involving at least 785 patients in France, Germany, Spain and Italy. More than 2500 human cases of trichinellosis have been caused by the consumption of horsemeat in France and Italy in the past 20 years. Most Spanish outbreaks were due to pork; pig infection by *Trichinella* is still occasionally disclosed at meat inspection in this country. The incidence is relatively high in Central Europe countries sharing borders with the EU: 1806 cases were reported in 1995-1996 in the ex-Yugoslavia, and 3092 cases in Romania for the 1995-June 1997 period; more than 300 cases were reported in 1998 in Slovakia after eating raw sausages made of dog meat. Outbreaks from wild boar are becoming more frequent in France (south-eastern part), Italy and Spain. The increasing number of outbreaks related to wild boar might be explained by the modification of the ecology of the rural parts of Europe, such as laying land fallow and the decreasing number of farmers that favour the proliferation of boars the number of which has increased by 9 fold in the past 20 years in France. Cultural behaviours such as hunter meals with undercooked roasted ribs also favour human infection.

### *6.7.3. Management options in place*

Only few cases of human trichinellosis due to consumption of meat from industrialised pig-farms has been reported since World War Two. Major outbreaks were linked to imported horsemeat (horses slaughtered and controlled abroad or horses slaughtered and controlled in the EU). In the EU Member States, about 190 000 000 pigs are examined every year for detecting larvae of *Trichinella* in muscle tissues, according to the EU legislation. This routine monitoring, surveillance and control by removing infected carcasses from the food chain has not prevented occurrence of major outbreaks. According to the recent trends in the epidemiology of trichinellosis, these measures seem currently poorly adapted to the actual risks. The main reasons

for this failure may be: 1) a relatively low diagnostic sensitivity of the current methods, especially because of the size of the examined samples that prevents diagnosis of mild larval infection; 2) routine controls not conducted systematically as required by the EU legislation; 3) mistaken entry of positive carcasses into the food chain; 4) absence of control for game locally consumed by the hunters themselves or their families/friends, or illegally sold; 5) and poor information to the consumers who are totally confident in the meat inspection system and eat raw pork or horse meat. The absence of trichinellosis in many Member States should lead to reconsideration of the cost effectiveness of the current control strategy in these countries.

### *6.7.4. Future management options*

In order to ensure a better protection of the consumer, alternative measures could be proposed:

*At the industrialised pig-farm level:*

- Barriers for preventing the entrance of rodents and other animals into the pigsty and the food store
- Admission of new animals to the farm only after serological examination
- Sanitary disposal of dead animals
- No raw or improperly swill feeding to pigs reared at the farm
- No rubbish dump present in the immediate area of the farm

Such measures could prevent the transmission of not only trichinellosis but also of other pathogens such as *Toxoplasma gondii* or *Taenia solium*

*At the slaughterhouse*

- Control of infection restricted to pigs raised by traditional pig-rearing practices and wild boars
- Careful control of imported meat and commercially distributed boars and other wild animals (*e.g. Trichinella* in crocodile meat)
- Control of imported horses and horse meat
- Improvement of the predictive values of meat examination procedures
- Quality insurance and testing of technicians skills

*At the consumer level*

Information on the risks of eating raw or undercooked meat, especially pork, horse and wild boar

*At the Veterinary/Health services level*

- Harmonisation of the reporting procedures at the EU level, both for human cases and meat inspection reporting;

- Co-ordination of Health and Veterinary services at the regional and national level in case of limited outbreaks;

- Co-ordinated monitoring and surveillance/alert system at the EU level to allow proper and rapid response in case of huge outbreaks

Reduction in the number of outbreaks in the various countries is a good indicator of the efficiency of a management programme. However, reliability must be ensured by a common case definition, accurate diagnostic tests (including serological evidence and species identification), and a central registry for human cases and epidemiological investigations performed at every outbreak.

*6.7.5. Research needs*

Centralisation of data by a European Network is needed to make possible sound epidemiological studies and judge more accurately the evolution of the disease and the specific measures that should be implemented in case of real re-emergence.

Development and standardisation of reliable laboratory techniques (for species identification, serology, and antigen detection) are necessary to implement alternative strategies of control and establish proper alert systems.

## 7. THE WHOLE FOOD PRODUCTION CHAIN : FARM TO FORK OR STABLE TO TABLE - RISK FACTORS AND POSSIBLE CONTROL OPTIONS

### 7.1. Farm

The control of the feed given to animal on the farm is the first barrier to introduction of zoonotic pathogens in primary production. The experience gained from Salmonella control suggests this as a control point. Moreover, the control of live animals before being introduced to the herd or flock is another option where the risk of introducing zoonotic agents could be reduced.

Farms having visitors or external workers (day visits and farm holidays) would benefit from introducing hygiene programmes to protect visitors from zoonotic infections and animals from exposure to pathogens, such as appropriate clothing.

Many of the most common zoonotic, food-borne pathogens causing human illness are found in the intestine of animal species used for food production. This includes *Salmonella*, *Campylobacter* and VTEC. Typically the bacteria do not cause disease in the animals, and typically the shedding of bacteria is intermittent resulting in difficulties in detecting infected individual animals. Another complicating factor is the occurrence of carrier animals harbouring the pathogens for prolonged periods.

Because these pathogens often do not cause any symptoms in the animal and apparently do not affect the production system, there has been little economic incentive for the producers to control them. The factors responsible for the introduction of these zoonotic agents into and the maintenance of these in the herd is generally not well understood. Contamination can enter through an infected animal introduced into the herd, as parental infection (typically in poultry farming) or as a more unspecific infection from the environment. Environmental contamination could stem from the wild fauna or from other animal herds through faecal contamination of water, pastures or even the air (aerosols).

The two main routes for the zoonotic faecal agents to reach the human consumer is through faecally contaminated animal products (meat and raw milk), infected eggs and through faecally contaminated produce (vegetables and fruits) or water (intended for drinking, processing, irrigation and recreational purposes). The faecal contamination of meat occurs primarily at the slaughterhouse, whereas the contamination of vegetables or fruits is often a result of the practice of using contaminated irrigation water, animal manure as fertiliser, or the effluents from storage of manure.

It has been suggested that the increase in the human incidence of these zoonotic diseases reflect a higher level of contaminated animals at the farm level. The data to compare the herd prevalences from 10-20 years ago with the present situation are not sufficiently reliable. However, the experience from some Member States, where a herd prevalence reduction strategy has been initiated for some or all *Salmonella* serotypes, seem to indicate that the lowering of herd prevalences result in a reduction in the human incidence of these disease types (Anonymous, 1994). In several Member States, interventions to control *Salmonella enteritidis* infections in poultry flocks have resulted in a dramatic decrease in the incidence of human infections.

The contribution made by the intensification of livestock production to the incidence of zoonotic infections in humans needs to be examined. Interventions to reduce the prevalence and transmission of pathogens in the livestock reservoir need to be identified and implemented.

A better understanding of the ecology and infection patterns of these microorganisms will lead to a better understanding of the relevant risk factors at the farm level. But even without a full comprehension of all factors, experience from some Member States show that intervention at the farm level can significantly lower both herd and animal prevalence. This experience relates primarily to *Salmonella* whereas experience for *Campylobacter* is limited and for VTEC O157 virtually non-existent (Report on Trends and Sources of Zoonotic Agents in Animals, Feedstuffs, Food and Man in the European Union in 1998).

For *Trichinella*, and some other parasitic pathogens, potential measures to prevent the introduction of the pathogen at farm level include: the introduction of barriers for preventing rodents in the pens or the food store; serological examination of new animals, sanitary disposal of dead animals; no rubbish dump present in the immediate area of the farm; and avoidance of introduction of raw or improperly heated meat as feed.

For the prevention of *Echinococcus granulosus* infection, management options at the farm level include different measures aiming at the control of dog populations (stray and owned), wild reservoirs and flocks (see 6.6.4 and 6.6.5)

Whatever risk management strategy is applied at farm level, an important prerequisite for assessing the situation and deciding upon intervention strategies is an updated knowledge about the true herd and animal prevalences of these agents, *i.e.* good epidemiological intelligence. This information can be used passively to oversee the situation or actively to apply specific management regimes to positive or highly infected herds. This effort should ultimately be aimed at a significant reduction in the number of infected animals that enter from this to the next stage of the food production stage. Additionally such information can be used to guide a sensible manure strategy, with the aim of ensuring that ready to eat produce is not contaminated with zoonotic agents from animal faecal sources.

## 7.2. Transport and lairage

Mixing and stressing of animals during transportation and in the lairage have both been shown to increase the occurrence of *Salmonella* and *Campylobacter* among animals. This increase is subsequently reflected in the prevalence of contaminated carcasses (Berends *et al.*, 1996; Hogue *et al.*, 1998; Line *et al.*, 1997; Puyalto *et al.*, 1997; Rigby *et al.*, 1982; Stern *et al.*, 1995; Wray *et al.*, 1991). A differentiation in the mode of transmission within infected and non-infected lots of animals on the farm of origin has to be made. In the infected lots the amplification of infection is due (1) to an increase in the number of shedding animals, through re-activation of latent infections, and/or (2) to exposure to environmental pathogens on uncleaned lorries and lairage pens (Berends *et al.*, 1996; Puyalto *et al.*, 1997; Stern *et al.*, 1995; Wray *et al.*, 1991). In the non infected lots the intensity of infection is due (1) to exposure to pathogen-bearing trucks and lairage pens, or (2) to co-mingling with infected lots of animals on the trucks or in the lairage pens (Berends *et al.*, 1998; Kampelmacher *et al.*, 1963; Morgan *et al.*, 1987; Williams and Newell, 1970).

At least in the case of *Salmonella* sp., faecal shedding from animals acquiring the infection, either orally or through aerosols, during transportation and lairage occurs within few hours of infection (Becker *et al.*, 1989; Fedorka-Cray *et al.*, 1995;). Berends *et al.* (1996) estimated that within only 2-6 hours after loading, the number of salmonella-shedding pigs within an infected lot might double. Currently applied Good Manufacturing Practices (GMP) of cleaning and disinfection of trucks and lairage pens will not prevent circulation of the *Salmonella* sp. or the *Campylobacter* sp. within an infected lot but can reduce cross-contamination of subsequent lots from other farms. The proportion of these infections that is prevented by the currently applied GMP protocols has not been accurately estimated. Berends *et al.* (1996) suggested that proper cleaning and disinfection of lorries and lairage pens between batches of pigs can prevent 75% of the contamination of non-infected batches from previous infected ones. However, their model was deterministic and did not allow for the variations in ‘proper’ cleaning that exist in practice. Strictly adhering to GMP of cleaning and disinfection might

only reduce the current rise in the incidence of pig salmonellosis during transportation and lairage by 10% (Berends *et al.*, 1998).

Animal *Salmonella*, and likely *Campylobacter* infections are strongly dependent upon on farm cycles (Berends *et al.*, 1998; Hogue *et al.*, 1998; Oosterom and Notermans, 1983; Stern *et al.*, 1995;), and the intensity of infection is amplified during transport of animals to slaughter. Alternative systems that inhibit contact between lots of animals of different origins during transportation and lairage need to be adapted in addition to intensification of current cleaning and disinfection protocols. These should incorporate transportation of different lots in separate containers and lairage into pens separated by concrete.

Farms are not sterile environments and it has to be assumed that some animals may be carriers of zoonotic pathogens. Therefore it is important that animals and poultry are transported in clean vehicles with the minimum of stress. Where possible the slaughtering of animals should be as close to the farm of origin as possible. Reducing journey time reduces the opportunity for transmission of pathogens. Cleaning vehicles between consignments and control measures must be taken on farms to ensure vehicles coming to collect animals and poultry are not introducing infection to the farm.

Lairages should be clean and stress reduced to the minimum. *Ante mortem* inspection should be vigilant and ill animals identified rapidly isolated to reduce opportunities for transmission of pathogens. The identification of diseased, injured, stressed or grossly faecally contaminated animals should precipitate an investigation of the transport system and the farm of origin.

In addition to the transport of livestock, other farm produce must be transported with the attention to hygienic handling and storage practices.

## 7.3. Slaughter

Slaughterhouses and abattoirs are food businesses and should pay the same attention to food safety as any other food business. The animals entering the plant should be as clean as possible. Grossly contaminated feathers and hides increase the amount of faecal material entering the plant and increase the likelihood of cross-contamination. Husbandry initiatives to produce stock as clean as practically possible should be encouraged.

Contamination and cross-contamination of carcasses and cuts occur while infected animals are being slaughtered. The risk of contamination of the meat cannot be eliminated under current slaughtering procedures. Implementation of Good Manufacturing Practices which are based on proper Critical Control Point analyses will, however, at best maintain the prevalence of contaminated carcasses and cuts (Mousing et al, 1997).

Prevention of carcass contamination with faeces should be the priority. However, the hygienic condition of walls, floors, ceilings or human carriers present in the slaughterline should not be disregarded. A strong correlation between the proportion of animals with *Salmonella* sp. in their faeces and the proportion of contaminated carcasses at the end of the slaughterline was

detected (Oosterom and Notermans, 1983; Oosterom *et al.*, 1985). The *Salmonella* sp. found on the carcasses were of the same type as those carried by the animals slaughtered the same day (Berends *et al.*, 1997; Limpitakis *et al.*, 1999). Berends *et al.* (1997) calculated that pigs with *Salmonella* in their faeces are 3-4 times more likely to end up as a positive carcass than pigs that are not carriers. Roughly the same estimate applies also to calves with *Salmonella* in their faeces (Berends *et al.*, 1997). About 70% of all carcass contamination results from pigs themselves being carriers and about 30% because of cross contamination (Berends *et al.*, 1997; Oosterom and Notermans, 1983).

Although the current slaughtering process of all animals allows for contamination of carcasses and for cross-contamination between infected and uninfected carcasses, and thus acts as an amplifier for the prevalence of pathogens, there are actually no steps in the process intentionally designed to reduce the hazards of carcass contamination. Investigators have tested some possibilities (Berends *et al.*, 1997; Borch *et al.*, 1996). For *Salmonella*, covering of the bungs with a plastic bag the moment the anuses are cut loose has favorably affected the prevalence of contaminated pig and beef carcasses in Danish slaughterhouses (Mousing *et al.*, 1997; Annual Report on Zoonoses in Denmark, 1998). Also, slaughtering of heavily infected flocks or herds in the end of the day and taking special precautions to reduce the hazard of meat contamination seem to reduce the risk of cross-contamination and the overall prevalence of infected meat (Hald *et al.*, 1999). Other measures are still under investigation as is the replacing of the spin-chiller by forced air-cooling for reduction of *Campylobacter*-contamination of poultry carcasses. Those measures that are proven both effective in the reduction of the prevalence of contaminated carcasses and practical in the incorporation into the slaughter process should be uniformly implemented.

Pathogen reduction treatments ('decontamination') in poultry slaughtering have been recently reviewed (SCVPH Report, 1998). These treatments have an effect in reducing pathogen contamination of carcasses but the extent is directly related to the initial level of contamination. For example a treatment that effectively reduces pathogen population on carcasses by 3 logs will reduce an initial population of $10^8$ to $10^5$, a population of $10^3$ to 1 and will decontaminate a carcass with initial population of $10^2$. Evidently, if the initial pathogen load is high, these treatments will not affect the prevalence of contaminated carcasses. Hence, in the overall reduction of the risk for foodborne disease, adoption and implementation of these treatments are beneficial as long as they are used in addition to other control measures.

For most food borne bacterial pathogens such as *Salmonella, Campylobacter, Listeria monocytogenes* or VTEC the traditional meat inspection procedures has a low diagnostic sensitivity (see Chapter 4). This is the case because these problems primarily relate to faecal contamination of the carcass, *i.e.* no visually evident changes can be seen in the carcasses. Therefore the traditional meat inspection procedure, as applied in EU Member States, cannot control these now important zoonotic pathogens.

However, the slaughterhouse is the 'key point' for the currently applied large scale surveillance and monitoring schemes (Mousing *et al.*, 1997; Wierup,

1997; Annual Report on Zoonoses in Denmark, 1998). Slaughterhouse samples (*e.g.* meat-juice samples, microbiological samples) are routinely collected, following statistically determined sample sizes, and tested by ELISA or isolation methods. Results are used to identify infected animal populations or to classify the animal populations to prevalence categories and apply appropriate control measures on farm and at slaughter. Further, microbiological results are used to estimate the prevalence of infected meat and meat products, the sources of infection and the pathogenic strains involved.

Animals entering the slaughter plants will often have pathogens in their intestinal tract. Therefore the practices within the plant should be focused on reducing the likelihood that contamination of the meat occurs. In addition to on farm initiatives to reduce disease and carriage of infective agents, sock should be as clean as possible entering the processing plants. This reduces the faecal load entering the plant and makes cross contamination during hide and fleece removal less likely. Historically some meat inspection efforts have been directed at controlling *tuberculosis* and *cysticercosis*. Visual inspection with incision of organs and glands has been the norm. Some of the major zoonotic pathogens causing human illness are not being addressed by the current procedures. The available modern laboratory techniques are not being applied routinely as part of the inspection process. The threats of *Salmonella*, *Campylobacter* and VTEC are not being optimally addressed. Farms and abattoirs are not operating theatres and pathogens will be present, however the objective should be to reduce the bacterial load on the final product. Trained operatives with an awareness of food safety are essential. Removal of the hides and evisceration are critical control points to avoid faecal contamination. Intervention to reduce the bacterial load such as chemical washes and steam pasteurisation are risk reduction initiatives in addition to the HACCP approach that are worthy of discussion within the EU. Meat juice ELISA monitoring of carcasses for *Salmonella* as undertaken in the Danish pig industry is an example of how infected farms are identified and remedial action taken. An integrated approach to disease control should be adopted throughout the EU. Microbial monitoring of carcasses coming into the abattoirs is necessary if we are to quantify the extent pathogens entering the food chain and mount an effective response. Currently the results of animal monitoring often come from veterinary diagnostic laboratories and reflect the disease status of sick animals rather than those entering the food chain. Microbial monitoring of carcasses and meat leaving the abattoirs will enable an evolution of the effectiveness of risk reduction strategies at the prior stages of the food chain and an assessment of the safety of product entering the remaining segments of the food chain.

The current meat inspection efforts regarding *Trichinella* should be focused on the relevant high risk practices such as control of traditionally raised pigs, imported meat and commercially distributed wild boars and other wild animals (*e.g.* crocodile), imported horses and horse meat. There is also a need for improvement of the sensitivity of meat examination procedures (see Chapter 6.4.4). Additionally, the absence of human trichinellosis in many Member States could lead to reconsideration of the cost–effectiveness of the control strategy in these countries.

To control *Echinococcus granulosus* infection, management at the slaughterhouse level is essential; it includes enforcement of control measures against illegal slaughter; inspection for hydatid cysts of all animals slaughtered; burial or safe destruction of cadavers and offal of food animals; training of personnel involved in slaughter.

## 7.4. Secondary processing

Numbers of bacteria in food can change at all stages of food production and processing, depending on the nature of the food and the way it is handled, stored and processed (Walls and Scott, 1997). Risk assessment of microbiological hazards must consider the fate of the hazards in foods (and the disease process following infection). The dynamics of microbial growth, survival and death should be explicitly considered together with distribution of the agent in appropriate foodstuffs.

The International Commission on Microbiological Specifications for Foods has recommended six steps for the management of microbiological hazards in foods in international trade. The steps include to establish a food safety objective (FSO), to confirm that the FSO is achievable through the application of GMP and HACCP and to establish microbiological criteria, when appropriate (Tompkin, 1998). A food safety objective is a concept in which one states the frequency or maximum concentration of a microbiological hazard in a food considered acceptable for consumption. Industry and regulatory authorities should make appropriate adjustments in their food safety management (*i.e.* GMP, HACCP) and inspection systems to meet the FSO. Control measures can be based upon performance criteria or process criteria. The FSO approach is an effective way of managing the microbiological hazards for foods in international trade and should facilitate the harmonisation of trade where the practices of one country differ from those of another, yet both provide safe products. This approach can also be applied to the management of domestically produced foods. (Tompkin, 1998).

There are different approaches to control microorganisms in food, *i.e.* assuring death of the pathogen (by technology), excluding multiplication/growth (without death) during the process and storage (by technology) and avoiding initial and subsequent contamination. Important clues for selecting the optimal control include

- the characteristics of the pathogenic agents of interest, the microbial ecology of the food,
- the initial contamination of the raw materials,
- the effect of the production, processing, handling, distribution steps and preparation by the final consumer on the microbial agent,
- the level of sanitation, the potential for (re) contamination, and
- the characteristics of the food that may influence the potential for growth of the pathogen in the food under various conditions.

Characteristics of the agent and the food commodity cover the capacity of the procedure or the potential of the agent to both survive and grow in the commodity. Impact of food technologies on survival and growth in various food commodities is different. Risk management options currently used are canning pasteurisation, lowering $a_w$, pH, competing flora, nitrate / nitrite, organic acids, preservatives, drying, smoking, heating, chilling, freezing, irradiation, exclusion of oxygen, and packaging. Management options at the processing plant cover the design of facilities, the production flow (separation of processing steps), processing (*i.e.* control and maintenance of temperature), and personnel involved in handling procedures, slicing, packing, mincing. Examples of bacterial count reduction by using such methods or combinations thereof in several food commodities are given in literature (for example Calicioglu *et al.* 1997; Connor and Kotrola, 1996; Goodfellow and Brown, 1978; Hinkens *et al.* 1996; Müller et al 1998; Robins et al, 1994).

During secondary processing cross contamination must be prevented, conditions that permit multiplication of any pathogens present must be avoided and where possible interventions to eliminate pathogens should be incorporated into the process.

Predictive microbiology (mathematical modelling) can forecast the growth, death or survival of microorganisms in response to environmental conditions and the likely number of microorganisms present in food at the time of consumption. Predictive microbiology can be used to select the most appropriate option for the food commodity of interest (Walls and Scott, 1997). The aim of this approach to microbiological food safety is to understand the responses of the concern to the most important controlling factors in the food environment, to build a cumulative store of information, and to develop the means of interpolating calculated microbial responses (Roberts, 1998). Examples of the usage of such models are given by Jones *et al.*, (1994), Roberts, (1998), Sutherland *et al.*, (1995), and Walls and Scott, (1997),

## 7.5. Retail, catering and at home

Risk management of food borne zoonoses in the retail, catering and home stages of the food chain, must deal with the residual risks from the earlier part of the food chain (feed, farm, primary and secondary processing). The following risk management objectives could be formulated as optimal suggestions to prevent contaminated food from entering this stage and to deal with residual risks from the earlier parts of the food chain through following strategies:

- to prevent the zoonotic bacteria multiplying,
- to prevent contamination of the food by water, the premises or the food handler
- to prevent cross-contamination between raw and ready to eat foods, and
- to kill the zoonotic agents by cooking or other treatments of the food

- to educate the consumer how to handle the food hygienically.

The Codex Alimentarius Commission has issued recommendations on food hygiene both at retail and consumption stages (1999) dealing with the risks in a structured way. While for specific pathogens such as VTEC O157:H7 the Pennington report (1996) and Irish Food Safety Authority (1999) have given detailed risk management suggestions.

Training of staff at all stages of the food chain is important as without their awareness of the risks, food safety cannot become an integral part of the food business. Consumers need to be aware that some foodstuffs such as raw meat carry a risk and must be appropriately handled in domestic kitchens if illness is to be prevented. Furthermore certain foods *e.g*. unpasteurised cheeses represent increased risk for vulnerable subsets of the population such as people with immuno-suppression

### *7.5.1. Retail*

The retail stage includes both large supermarkets and small convenience stores, thus the number of participants is larger than earlier in the food chain. One retailer might infect many consumers, indicating the need for efficient risk management at this stage also.

Segerson (1999) has suggested that in the cases where the consumers can detect food risks, the firms can be persuaded by market forces to invest in food safety, thus pointing to a voluntary approach. This suggests those companies with valuable brand names and supermarket chains have incentives to attain a food safety beyond the statutory and due diligence requirements. Following these assumptions one approach could be to inform the consumer about the food safety of different products and retail outlets, to enable the consumer to make informed choices. Additionally, the advice given from the food safety authorities to any part of the retail sector both individual operators and groups could be publicly available.

In the cases where the consumers cannot detect food safety risk and thus discriminate against high risk products or retailers, regulation is more of a necessity. Van Schothorst (1998) proposed that the regulation should be in the form of food safety objectives (FSO), while leaving it to the enterprises themselves how to achieve these objectives through GMP and HACCP procedures. Hence, promoting informed consumer choices might be an efficient way of promoting food safety in the retail side, while the setting of FSO would be an efficient regulatory approach.

Three additional risk management measures should be noted: the provision of safe drinking and processing water, the control of pest and vermin, and the prevention of food contamination by pet animals (cats and dogs). Pest and vermin control is necessary whenever perishable foods are handled through the whole chain from farm to fork. The background for the prevention of food contamination by pet animals is outlined in Chapters 6.6. and 6.7.

Continuous training and education in food safety for all working with food in the retail sector is necessary to ensure that knowledge is disseminated and

implemented. If people working with food have diarrhoea, are diagnosed with zoonotic agents or have severe skin lesions, they should discontinue working with food (IFST, 1999) at least until symptom free. Then the need for good hygiene practices should be outlined before they are allowed to return to food handling.

The retail sector could contribute to lowering the residual risks in the food chain by giving the consumers advice on how to safely prepare the food as suggested by the Codex Alimentarius (1999).

Food that carries a risk of containing zoonotic pathogens should be appropriately labelled. Instructions could include information on cooking to kill the pathogens, refrigeration to prevent multiplication and handling instructions to avoid cross-contamination. There must also be labelling of packaged products that are either raw or partially cooked and intended for consumption without further cooking in both retail and catering sector. This would enable us to set shelf life and appropriate storage conditions. Furthermore those in particularly vulnerably subsets of the population should be alerted to the specific risk for them so that they can avoid this product if they so choose.

For certain parasitic food-borne zoonoses such as *Echinococcus* sp. infections, risk management does not concern meat, since infected meat (containing cysts) is not infectious to humans. However other types of food may be contaminated, especially vegetables, if dogs have access to gardens where vegetables are grown for human consumption, or if they have access to such food during their transport or retail. The following measures should thus be implemented in those areas where echinococcosis is endemic: fencing of kitchen gardens (family and commercial) to prevent any access of dogs to vegetables aimed at human consumption; and control stray dogs, especially around outside market facilities.

### *7.5.2. Caterers*

The catering industry differs from the retail industry in that the consumer is offered ready-to-eat food with little possibility for further risk reduction before eating. The consumers have less information about the food to be consumed as no EU health marks, origin or producer identification is easily available. Hence the consumer is left to trust the implicit guarantees of the caterers such as brand names, the due diligence concept and the guarantees afforded by the food safety authorities.

The considerations for the retail stage do generally also apply to the catering stage. The important factors in safe catering are

(1) The provision of safe ingredients

(2) Appropriate storage and cooling to prevent any pathogen multiplying

(3) Prevention of cross-contamination

(4) Sufficient cooking to kill pathogen

(5) Staff training to raise hygienic practices and the need for vigilance

The provision of raw ingredients of high quality from recognised suppliers who operate codes of good practice and HACCP where appropriate is essential. The safe storage of food must comply with criteria for temperatures and storage periods based on public health considerations. Where such temperature and storage criteria is lacking they should be established as a priority. Moreover, the provision of raw food with as little residual risks as possible and precautions to avoid cross contamination of food ready to eat should be priorities in the catering sector. The heat treatment of food is another important safety hurdle. Proper heat treatment would kill most zoonotic bacteria such as *Salmonella*, VTEC O157, *Campylobacter* and *Listeria monocytogenes*, and all zoonotic parasites. For example the Irish Food Safety Authority (1999) recommends heat treatment of minced meat products (hamburgers) for at least 2 minutes to kill VTEC O157 (70°C). Cooked food should be served at once or chilled.

*7.5.3. Home*

At home the consumer, as the last link in the food chain, has to deal with any residual risk. This last risk reduction step does not preclude the feed producer, farmer and primary and secondary food processor, and the retailers from their obligations to provide safe food.

As in commercial catering, appropriate stage/refrigeration, prevention of cross-contamination and adequate cooking are important control steps in domestic kitchens. Knowledge of food hygiene is important and initiatives to educate consumers in food safety are essential. These should be targeted at different population subsets - school children, young adults, pregnant women, elderly, or vulnerable people with tailored information to have maximum effect. The IFST (1999) has published on avoiding cross-contamination in the home indicating sources of pathogens such as domestic pets, vermin (insects and rodents) and raw food (such as fresh meat, poultry, eggs). Cross-contamination could happen by the use of the same knives, work surfaces and ustensils for raw foods and ready to eat foods. Furthermore, all consumers should be educated through school and other channels about the handling of foods, including basic hygiene such as washing hands. Moreover, to maintain an appropriate cold chain refrigerators should include a section where the 0-3ºC can be achieved and controlled.

Foods that are safe if prepared under traditional settings may not be as safe when handled by unaccustomed consumers, *i.e.* fresh cheese stored in the fridge for weeks might increase the risk for listeriosis. (Linnan *et al.*, 1988). Beard (1991) suggested that the HACCP approach should be extended into the home through the education of the consumer about the critical control points. For example, in the case of introducing partially preserved, minimally processed non-sterile foods with extended shelf life, Rhodes (1991) suggested educating consumers and food handlers in the differences between these foods and traditional refrigerated foods, based on a HACCP approach. Daniels (1991) suggested that for these foods one should consider temperature audits as far as possible towards the point of consumption. Whether these suggestions are practical risk management options remain

unclear. For vulnerable groups (immuno-compromised persons) and those groups eating novel foodstuffs either due to novel use or preparation in the home, a targeted effort should be made to inform the particular group about putative risks.

Echinococcosis is tightly linked to human behaviour towards dogs and food, cultural habits, and misunderstanding of the real risks of such behaviours and habits for health. Important messages for health education include avoiding contacts between dogs and food; proper dog feeding (excluding raw sheep and cattle offal) and regular treatment with praziquantel or an equivalent drug; proper cooking of human foods that were possibly in contact with parasite eggs.

## 8. MEDICAL ASPECTS OF ZOONOSES CONTROL

### 8.1. Vulnerable groups

What may be a mild disease for a healthy adult can be life threatening for a frail elderly person, infant or a person suffering from some concurrent disease. The ideal should be to have food that is safe for the weakest members of society. In the absence of this ideal food safety, vulnerable groups and those caring for them should be made aware of the risks. Zoonotic pathogens, initially acquired by the foodborne route can spread to other individuals by the person to person route. Therefore it is important that caregivers in institutions such as old folks homes, nursing homes, day care centres, hospitals and in crèches are well trained, aware of the risks and able to ensure that food hygiene practices are optimal and the personal hygiene and infection control are adequate.

Among zoonotic agents, some may cause disease in most exposed subjects. Conversely, some give significant signs and symptoms only to those individuals whose natural defences against these agents are deficient because of inherited or acquired immune depression. Such diseases are called “opportunistic diseases”. Most of the pathogens, however, may cause disease in subjects with normal immune defences as well as in immuno-suppressed patients, but the course of the disease is accelerated and/or the severity markedly increased in the latter.

The particularly at risk groups are subjects with AIDS, patients treated by cytotoxic chemotherapeutic agents or irradiation, and patients treated with glucocorticosteroids or immuno-suppressants for chronic systemic auto-immune diseases or to prevent organ rejection after transplantation. Exclusive opportunistic agents might cause significant disease to these patients only. However, it must be stressed that these conditions are more and more frequent among the European populations, and that persons with the above-mentioned health problems represent vulnerable groups for all significant zoonoses studied in this report.

Other patients with a variety of associated diseases such as diabetes mellitus, liver cirrhosis, chronic renal failure, chronic anaemia, may also be considered vulnerable groups for most of the zoonoses. Specific associations of

resistance/susceptibility to zoonoses with immunogenetic particularities of the individuals have also been demonstrated (for instance in *Echinococcus* infection).

Because of immaturity of their immune systems against particular bacteria, and due to their behaviour, infants and very young children represent a vulnerable group for bacterial and parasitic infections. Malnutrition and various degrees of immune impairment make elderly another vulnerable group, especially for enteric bacteria (*Salmonella, Campylobacter*, VTEC) and intracellular bacteria (*Listeria monocytogenes*). Finally also pregnancy is associated with a certain, albeit limited, degree of immune depression, and also represents a vulnerable condition, regarding *Listeria monocytogenes* infections.

## 8.2. Investigation of outbreaks

Human infections with zoonotic pathogens occur as sporadic cases or as part of outbreaks. Sporadic cases are those with no known epidemiological link to another case. In these it is most often impossible to establish whether the route of transmission was foodborne. Conversely, outbreaks if thoroughly investigated, present the opportunity to identify the pathogen, the food vehicle involved and the factors in the food preparation and handling that contributed to the outbreak. Foods can be implicated on the basis of the identification of the pathogen in the food, on statistical evidence from epidemiological studies demonstrating an association between consumption of the food and illness. The collaborative effort of medical, veterinary and food authorities is necessary in the control of outbreaks if the infections shall be traced back to the source and corrective actions taken where faults are identified throughout the food chain.

## 8.3. Risk communication

The European Commission "White Paper on Food Safety (2000)" emphasises risk communication as a key element in ensuring that consumers are kept informed and in reducing the risk of undue food safety concerns. The ease of access for all stakeholders to relevant information, and the ability to formulate questions and to express concerns will be a cornerstone of the new food safety policy within the EU.

It is important to convey to the public, all sectors of the food industry and the public health professionals that some products cannot be produced without a residual risk of zoonotic infection for the consumer. An absolute guarantee against infection can never be given, therefore it should be explained that zero risk is not achievable. To avoid precipitating food scares the current risk should be put in perspective and the strategies to reduce risk and manage it to prevent human disease outlined.

Risk communication is defined as the interactive exchange of information concerning risk between risk assessors, risk managers, consumers and other stakeholders (Anonymous, 1998). The purpose of risk communication could be elaborated as the exchange of information enabling all stakeholders to get

information about the risk and to accurately assess and if possible to address the concerns of each other.

Leiss (1997) reviewed the risk communication in the BSE case within EU during 1990's, the dioxin case in USA and Canada from 1974-1996, the outbreaks of VTEC O157:H7 traced back to hamburgers in USA, and the risks associated with PCBs (polychlorinated biphenyls) for the Inuits in Canada. Leiss concluded that both industry and regulators are responsible for effective risk communication. Effective risk communication must address the concerns of the public, put scientific findings in context, and take into account that risk information vacuums might amplify a crisis. The perception of health risks by the public can be quite different from those risks that appear when collating morbidity and mortality statistics. Ulleberg and Rundmo (1997) found that in Norway, people were much more concerned with the risks posed by chemical food additives than food contaminated with microbiological pathogens, while it is clear that a greater number of people develop disease from the latter. People do also tend to underestimate and accept well-known everyday voluntary risks such as smoking and car driving, while to them unknown and imposed risks such as foodborne diseases are overestimated and rejected. Risk communication must address both people's risk perception as well as the objective estimations of risks.

Moreover, the use of risk communication in the form of education and training can be a tool for public health improvement (Schwabe, 1984) and the failure to gain the acceptance of the public health information of the target audience has delayed or obstructed many public health campaigns. In other words unless the target audience accepts the information offered and changes its behaviour accordingly, it is useless from a risk management perspective, however scientifically sound.

Hence, it appears that risk communication has several disparate purposes in risk analysis:

- For the scientific community to draw the attention of the public and risk managers to issues of concern, *i.e*. to facilitate the risk assessment information.

- For the risk managers, public and private, to accurately evaluate and manage the risks to public health.

- For the general public to draw the attention of the scientific community and risk managers to its concerns.

- To inform the general public about appropriate measures to reduce public health risks associated with zoonotic agents.

- To promote interaction between risk assessors, managers and stakeholders when doing risk analysis.

Risk communicators should have adequate tools for each purpose. The general public might perceive risks quite differently to the risk managers and the scientific community. Risk communication should be a tool for accurately

communicating these perceptions as well as exchanging the relevant risk information in an intelligible way between all stakeholders in a risk analysis process.

## 9. OBJECTIVES FOR ZOONOTIC PATHOGEN CONTROL

The aim of zoonotic pathogen control is to reduce the incidence of human disease. This can be achieved by elimination of the pathogen at the most appropriate stage in the food chain. Where this is not feasible incremental risk reduction at all stages of the food chain is the approach to adopt together with communication to the final consumer of the residual risk and how to manage it.

In risk analysis terminology the risk estimate, which is the basic outcome of a microbiological risk assessment, represents the actual risk, and could be presented as the fraction of the population contracting a food-borne disease (or dying from it) annually. The risk estimate can be higher or lower than an acceptable risk level. If the actual level is higher than the acceptable risk level, risk management decisions are necessary to define initiatives to reach a lower risk level, *i.e.* the *target risk level.* The use of the word 'target risk level' reflects the dynamic nature of food-borne microbial disease risk.

Target risk levels should be set primarily in relation to the incidence of human disease, since the risk concept inherently relates to human disease. However, in a number of cases the risk management initiatives will only indirectly relate to human disease. Instead, the primary initiatives will centre on the attainment of a tolerable level of the pathogen in the food. Such levels are likely to be referred to in the future as *Food Safety Objectives (FSO´s).*

FSOs, which is yet a concept without an international definition, could in the future represent the practical application of risk management decisions. For most pathogens, FSO's are basically intended, on the basis of relevant risk assessments, to outline the tolerable level, *i.e.* the maximum concentration or prevalence**,** of a pathogen in relevant products. A tolerable level in relation to a number of the traditional food-borne pathogens, such as *Staphylococcus aureus* and *Bacillus cereus*, are typically concentrations, mainly because the pathogenesis includes a toxin effect which is directly related to quantity or dose. However, for most of the relevant new zoonotic pathogens, such as *Salmonella* or *Campylobacter*, the main ethiological factor is a transient colonisation of the gut, which is not directly related to quantity or dose. At the same time, while the former bacteria are tolerated in some ready to eat products below certain concentrations, the latter are not tolerated in ready to eat products. However, as mentioned above, the risk may be different for different consumers with varying degrees of immune defences and this parameter has to be taken into account. In the future it is likely that realistic targets (FSO's) for *Salmonella* and *Campylobacter* will be set in relation to raw products and in the form of maximum prevalences to be achieved by the producers.

The determination of safe, realistic and achievable risk levels depends not only upon the hazard and risk situation, but also upon a number of socio-economic and technological factors. Accordingly the best management option could be: control at the source, action plans in the production level, introduction of general hygiene measures, introduction of specific production control measures, criteria in relevant

parts of the production chain as well as in the final product at the point of consumption. Significant differences exist between different regions in the socio-economic factors and the production systems as well as in the prevalence of certain food-borne zoonotic pathogens, notably *Salmonella* sp. Therefore FSO's should not be considered universal, neither in time nor in space. FSO's could in some situations reflect relevant and significant regional differences, and likewise FSO's should be reviewed at regular intervals.

Since prevalence targets can differ between regions, an important task of the future will be to define methods to assess the prevalence of certain zoonotic pathogens in a reliable and comparable way between regions. Likewise a further harmonisation of monitoring and data presentation requirements is necessary. An appropriate system for the presentation and comparison of relevant prevalences between regions will be one of the ways to enable the efficient risk management option of informed consumer choice.

Moreover, it should be noted that travel abroad represents a frequent but often overlooked risk for the exposure for zoonotic pathogens.

## 10. CONCLUSIONS

- The aim of food-borne zoonotic pathogen control is to reduce the incidence of human disease. This can be achieved by elimination of the pathogen at the most appropriate stage in the food chain. Where this is not feasible, incremental risk reduction at all stages of the food chain is the approach to adopt together with communication to the final consumer of the residual risk and how to manage it. The possibilities of risk reduction at home do not substitute for the risk management measures possible earlier in the food chain.

- The methods for detection and reporting are neither standardised nor harmonised for most zoonotic agents of concern. When appropriate, subtyping is not used in most Member States in a uniform way, apart from *Salmonella* serotyping. Therefore, prevalence data of the infection in animals, food contamination and incidence data of the disease in humans are generally not aligned to be comparable within the EU.

- Sentinel laboratories are rarely used as sources of epidemiological information on zoonoses. Networks exist for several zoonotic agents, but the range of activities is limited and there is no public access to the information produced in these networks established at the moment

- Because of these different protocols and methods for the sampling, analysis, and reporting of the same zoonotic agents and diseases between and within Member States, the existing incidence data on human food-borne zoonoses from different Member States are limited or not available. The available data indicate however an increase in many reported food-borne zoonotic infections over the last 20 years.

- At present in the EU, zoonoses risk management is not generally based on formal risk assessment as described in the "Principles for the development of risk assessment of microbial hazards under Directive 93/43/EEC concerning the hygiene of foodstuffs - Principles for the development of microbiological criteria for animal products and products of animal origin intended for human consumption". The content of the Annexes of this report should not be considered formal risk assessment.

- The ubiquitous nature of the pathogens and the expansion of travel and trade among Member States as well as with third countries could challenge the efficacy of national programmes to control zoonoses.

- The Committee identified the following zoonotic agents as public health priorities in Europe: *Salmonella* sp., *Campylobacter* sp., *verotoxigenic Escherichia coli* (VTEC), *Listeria monocytogenes, Cryptosporidium* sp., *Echinococcus granulosus / multilocularis* and *Trichinella spiralis*. If referring to the number of reported human cases, the most important food-borne zoonoses currently are *Salmonella* and *Campylobacter*, however a full description of relative importance would also involve considerations on loss of (quality of) life as well as economical considerations.

- The monitoring and surveillance data of a number of other (mainly non food-borne) zoonotic pathogens as well as of viral food borne zoonoses are not collated and analysed on the Community level.

## 11. RECOMMENDATIONS

There is a potential for significant improvement in the present food control and inspection procedures, which to our present knowledge could reverse the increasing trend in zoonotic food-borne disease. More could be done to enhance food safety and what is done could be done better:

- Monitoring and surveillance of food-borne zoonotic diseases and food-borne zoonotic agents in the EU should be revised with the objective of
    - following epidemiological trends in live animals and food.
    - estimating the true incidence of human diseases in each member state
    - allowing the comparison of data between EU-Member States, and
    - early detection of outbreaks of human diseases
- The establishment of comparable surveillance programs throughout the EU-Member States should be targeted towards important food-borne zoonotic agents.
- Common definitions of cases, terminology, sampling schemes, laboratory protocols and methodology are needed
- Existing sentinel surveillance systems could be used to estimate the true human incidence of zoonotic diseases in all Member States. They should be linked between Member States or implemented where not available. Moreover, population-based studies determining the sensitivities of these sentinel systems should regularly be performed to produce data comparable between Member States.
- Ad hoc epidemiological studies should be performed to identify and assess risk factors
- A formal collaboration between the medical, veterinary, food and feed authorities in each member state is needed to strengthen the zoonosis prevention, outbreak recognition and control. In particular, there should be a seamless supervision of the food chain from feed mills to the point of sale to the consumers.
- Networks should be encouraged for the important zoonotic agents and results should be made accessible to all relevant groups in the food chain. Zoonoses centers/task forces and sentinel labs could be helpful to achieve this objective. These networks should be closely linked to the epidemiological network established by Decision 98/2118/EC.
- A Community network should be set up for the detection of emerging zoonoses.
- The risk management initiatives for control of food-borne zoonoses should be based on formal risk assessments and data on human incidence should be used to measure the effect of the control options established.
- Food safety objectives (FSO) should be set in relation to a tolerable incidence of human disease, but in a number of cases the primary initiatives will relate to a tolerable level, *i.e.* concentration or prevalence of the pathogen in food and/or in animals. A principle of sequential incremental risk reduction should be applied. Risk reduction

should be sought through integrated initiatives from feed mills to the points of consumption.

- The prevalence reduction strategies at farm level for *Salmonella* sp., *Campylobacter* sp. and VTEC O157 should be further investigated. Community control programs for *Salmonella* in feed and for control in breeding animals could be helpful in reducing the prevalence of salmonella on farms.

- The food-borne zoonotic risks related to organic farming need to be assessed and guidelines produced.

- Specific actions at farm level should also be planned for the control of some parasitic zoonoses, together with consistent modifications of the current meat inspection procedures at the abattoir.

- There is a need for a thorough change of current meat inspection procedures with respect to public health priorities, emphasising the hazards that are currently most significant.

- Slaughterhouse monitoring should provide the epidemiological intelligence about the zoonotic agents entering the food chain and on the effectiveness of control measures in primary production.

- The present food control system should be re-focused to address the most significant risks to public health.

- Appropriate training programmes in personal and production hygiene for participants in all stages of the food chain are needed. A formal training in personal and home hygiene might be implemented in all primary schools.

- Proactive EU programmes should be encouraged to communicate risks as well as ways to manage them to all sectors in the food chain, with simple and consistent messages targeted at different population groups. These programmes should be continuously evaluated.

- Specific messages should address vulnerable groups and those giving care to these groups.

- The Committee draws the attention to the possible risks for public health posed by other (environment borne, and/or viral) zoonoses and recommends that these be also assessed.

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# 13. ANNEXES

## 13.1. ANNEX I

### *13.1.1. Annex I.a : Thermophilic Campylobacter*

## I. RISK ASSESSMENT

A. Hazard identification

In the 1970s, with the development of suitable selective media, it was established that *Campylobacter jejuni* and to a lesser extent *Campylobacter coli* were a major cause of diarrhoeal illness (Skirrow, 1977). *Campylobacter* is now rivalling and even surpassing *Salmonella* in importance in many countries. In 1997 the incidence rate of *Campylobacter* had exceeded that of *Salmonella* in Spain, Sweden, The Netherlands, Scotland, Northern Ireland, and England and Wales (Anon., 1999).

The number of human *Campylobacter* cases is registered in twelve EU Member States. The incidence rates per 100.000 inhabitants from 1995 to 1997 are shown in Fig. 1. In general, it can be noted that the number of reported human cases is increasing in many countries indicating that *Campylobacter* is the cause of an increasing human health problem. The incidence rates vary widely (from 9.5 in Spain up to 108 per 100.000 inhabitants in Scotland in 1997) probably due to differences in surveillance systems, diagnostic methods and way of reporting. Therefore, the data from the Member States should not be compared directly.

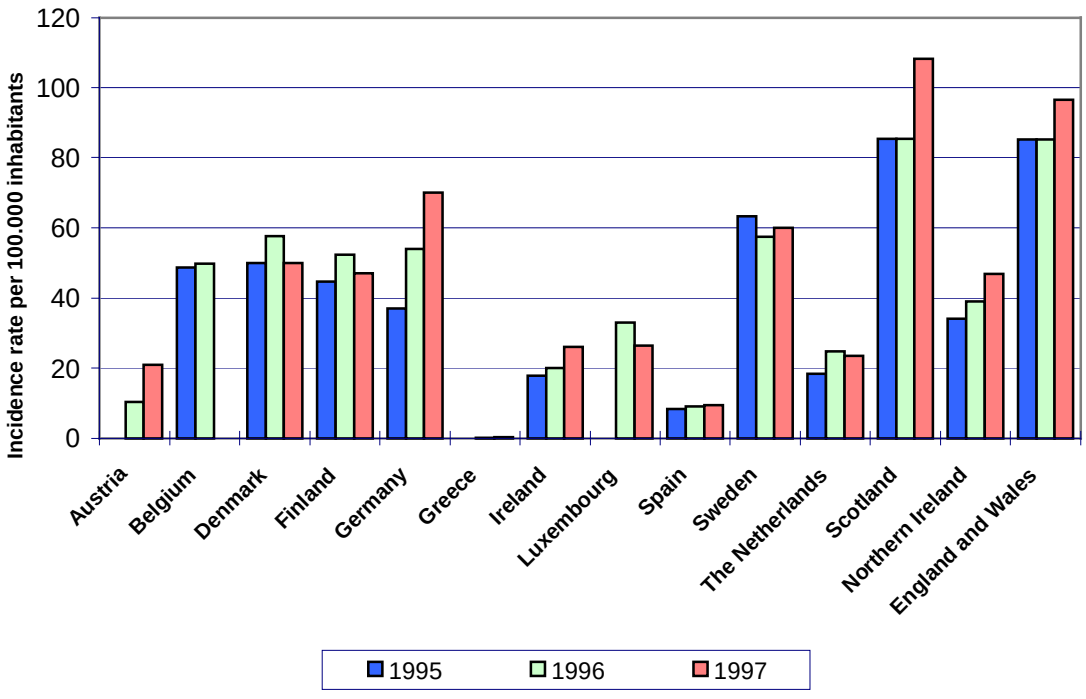


**Figure 1.** Campylobacteriosis in humans. Incidence rate per 100.000 inhabitants (Anon., 1999)

Fig. 1 reflects the laboratory confirmed cases of *Campylobacter*, cases where the patients have consulted a doctor/hospital, who following has found *Campylobacter* in a stool sample, *i.e.* only a fraction of the true number of infections. The true rate of infection is considered to be 10-100 times as high as the reported cases (Kapperud, 1994; Skirrow, 1991).

(1) Characteristics of the organism

*Campylobacter* are non-sporeforming, oxidase-positive, Gram-negative rods. Cells are pleomorphic. Log-phase cells have a characteristic slender, curved or spiral shape and have flagella, usually single, at one or both poles (monotrichate or amphitricate) and are highly motile, spinning around their long axes and frequently reversing direction. As cultures age, spiral or curved forms may be replaced by coccoid forms (Barrow and Feltham, 1993).

In general, *Campylobacter* do not grow in conventional aerobic or anaerobic culture systems. *Campylobacter* do not ferment or oxidize sugars and are oxygen-sensitive microaerophiles, growing best in an atmosphere containing 5-10% oxygen. Most strains grow in sloppy media (0.16% agar) incubated aerobically and suitably supplemented with oxygen scavenging compounds (*e.g.* blood, haemin, inorganic iron salts, pyruvate and charcoal) (Barrow and Feltham, 1993).

*C. jejuni* and to a lesser extent *C. coli* are the species most often encountered in medical laboratories as causes of acute enterocolitis in man (Anon., 1999; Nielsen *et al.*, 1997; Wooldridge & Ketley, 1997). They are distinguished from most other *Campylobacter* by their high optimum growth temperature (42°C). *C. jejuni* has two subspecies; subsp. *jejuni* – the familiar cause of enterocolitis in man and subsp. *doylei* – a more fastidious and slower growing organism which does not grow at 43°C. *C. upsaliensis* also appears to be enteropathogenic for man. This species is related to the "thermophilic" *Campylobacter* though not all strains grow at 43°C. As primary isolation of this species usually requires the use of selective filtration and non-selective media incubated at 37°C, this species is seldom detected by conventional methods used for *C. jejuni* and *C. coli*. *C. lari* is "thermophilic like *C. jejuni* and *C. coli* but is of low virulence and encountered only occasionally in man (Barrow and Feltham, 1993).

(2) Reservoir

The principal reservoir of pathogenic *Campylobacter* sp. is the alimentary tract of wild and domesticated animals and birds. The prevalence of *Campylobacter* in these animals and birds as reported for 1997 by the Member States (Anon., 1999) is listed in Table 1. From these data it is not easily evident that *Campylobacter* is commonly found in broilers, fowls, cattle, pigs, wild animals and birds, and in dogs. Other investigations have shown that healthy puppies and kittens (Hald & Madsen, 1997), rhodents (Berndtson, 1996; Cabrita *et al.*, 1992), beetles (Jacobs-Reitsma *et al.*, 1995), and houseflies (Berndtson, 1996; Rosef & Kapperud, 1983) may also carry *Campylobacter*.

Water is also an important part of the ecology of *Campylobacter*. *Campylobacter* has been isolated from surface water, rivers, and lakes at prevalences up to about 50% (Arvanitidou *et al.*, 1995; Bolton *et al.*, 1987; Brennhovd *et al.*, 1992; Carter *et al.*, 1987). Additionally, *Campylobacter* has been found in sand from bathing beaches at a prevalence of 45% (Bolton *et al.*, 1999). This means that *Campylobacter* may be present in untreated drinking water and bathing water. *Campylobacter* is introduced into the

water by sewage and faeces from wild animals and birds. The isolation frequency of *Campylobacter* from water is highest in cold winter months (Brennhovd *et al.*, 1992; Carter *et al.*, 1987). This is explained by a higher survival rate at low temperatures. It has been shown that in water *C. jejuni* survived for one to over four weeks at 4°C, whereas at 25°C the bacterium persisted for only 4 days (Blaser *et al.*, 1980). Another study has shown that *C. jejuni* remained recoverable for up to four months when suspended in aged, filter-sterilized stream water held at 4°C (Rollins and Colwell, 1986). At 25°C and 37°C the bacteria became nonculturable within 28 and 10 days, respectively.

In water and other environments with sub-optimal growth conditions, *Campylobacter* may convert into a "viable but nonculturable state". The importance of this "state" in transmission of *Campylobacter* to animals and man is not agreed upon. The question is if the viable nonculturable organisms are still virulent or if they can reverse into a culturable, virulent state after passage through a host. In some studies "viable but nonculturable" organisms have shown to regain culturability after passage through for example chicks (Stern *et al.*, 1994), mice (Jones *et al.*, 1991), and rats (Saha *et al.*, 1991). In other studies it has not been possible to demonstrate that "viable but nonculturable" *Campylobacter* can change into a culturable state (Beumer *et al.*, 1992; Boucher *et al.*, 1994; Fernley *et al.*, 1996; Korsak & Popowski, 1997; Medema *et al.*, 1992).

*C. jejuni* and *C. coli* seem to have a favoured reservoir. *C. jejuni* is predominantly associated with poultry (Tauxe, 1992), but have also been isolated from cattle, sheep, goats, dogs and cats (Anon., 1999; Nielsen *et al.*, 1997) *C. coli* is predominantly found in pigs (Nielsen *et al.*, 1997; Rosef *et al.*, 1983), but has also been isolated from poultry, cattle, and sheep (Anon., 1999). In a Norwegian survey, 100 percent of the pigs examined were infected with *C. coli* (Rosef *et al.*, 1983).

B. Hazard characterisation

(1) Disease

Enteropathogenic *Campylobacter* can cause an acute enterocolitis, which is distinguished from illness caused by other pathogens. The incubation period may vary from 1 to 11 days, typically 1-3 days. The main symptoms are malaise, fever, severe abdominal pain and diarrhoea. Vomiting is not common. The diarrhoea may produce stools that can vary from profuse and watery to bloody and dysenteric. In most cases the diarrhoea is self-limiting and may persist for up to a week, although mild relapses often occur. In 20% of the cases symptoms may last from one to three weeks (Allos & Blaser, 1995). Excretion of the organism may continue for up to 2-3 weeks.

*Late complications*

In rare cases, *Campylobacter* has shown to cause the serious disease, Guillain-Barré syndrome (GBS), a demyelating disorder resulting in acute neuromuscular paralysis. Early symptoms of GBS include burning sensations and numbness that can progress to flaccid paralysis. It has been estimated to occur about once in every 1000 cases of campylobacteriosis, *i.e.* up to 40% of all GBS cases in the US occur after *Campylobacter* infections (Allos, 1997; Mishu *et al.*, 1993; Mishu & Blaser, 1993). GBS seems to be more common in males than females (Mishu *et al.*, 1993). Although most GBS patients recover (about 70%), chronic complications and death may occur (Blaser *et al.*, 1997). There is no relation between the severity of the gastrointestinal symptoms and the likelihood of developing GBS after infection with *C. jejuni*; in fact, even asymptomatic

infections can trigger GBS (Allos & Blaser, 1995). The pathogenesis of GBS is only partly known. GBS is presumably caused by an immunological cross-reaction between *Campylobacter* anti-genes (lipopolysaccharides) and glycolipids or myelin proteins in the peripheral nervous system. The serotype O:19 seems to be more often involved in this condition than other *Campylobacter* serotypes (Allos, 1997; Blaser & Allos, 1995).

Campylobacteriosis is also associated with reactive arthritis (incomplete Reiters Syndrome). Multiple joints can be affected, particularly the knee joint. Pain and incapacitation can last for months or become chronic. It has been estimated that reactive arthritis occurs in approx. 1% of patients with campylobacteriosis. The sterile postinfection process occurs seven to ten days after onset of diarrhoea (Peterson, 1994). Reactive arthritis is often associated with tissuetype HLA-B27 and cannot be separated from the affectation of the joints that may follow from a *Yersinia*, *Salmonella* or *Shigella* infection (Allos & Blaser, 1995; Peterson, 1994). The condition is immunological and cannot be treated with antibiotics. The medical treatment may consist of a non steroid anti inflammatory drug (NSAID). The pathogenesis of this entity is unknown (Allos & Blaser, 1995).

In sporadic cases, campylobacteriosis have also been associated with a rare variant of poly-neuritis called the Miller Fisher Syndrome (Roberts *et al.*, 1987).

In general, very few deaths are related to *Campylobacter* infections and these deaths do usually occur among infants, elderly and immuno-suppressed individuals (Tauxe, 1992). In England and Wales fewer than 10 deaths of approx. 280.000 cases has been reported from 1981 to 1991 (0,0036%) .

*Antimicrobial resistance*

Antimicrobial resistance may prolong illness and compromise treatment of patients with bacteremia. In the beginning of the 1990[ies], fluoroquinolone-resistant *C. jejuni* emerged in human populations in Europe as reported in the UK, Austria, Finland and the Netherlands (Piddock, 1995). This resistance has been linked to the approval of enrofloxacin for treatment of diseases of broiler chickens as investigations have shown that fluoroquinolone-sensitive *C. jejuni* strains were able to convert to resistant forms when fluoroquinolone was added to broiler chicken feed (Jacobs-Reitsma *et al.*, 1994). In general, most human *Campylobacter* infections are self-limiting and do not need antimicrobial therapy. However, in severe cases medication may be necessary. In such cases the drug choice is usually erythromycin, though fluoroquinolones such as ciprofloxacin and norfloxacin are also used (Blaser *et al.*, 1983). Hence, fluoroquinolone resistance may cause severe problems in cases where drug treatment is required.

(2) Virulence / pathogenicity

The pathogenesis of *Campylobacter* has been reviewed by several authors (Ketley, 1995;1997; Smith, 1996; Wooldridge & Ketley, 1997). In general, the mechanisms involved in the pathogenesis of *Campylobacter* are rather poorly understood. Motility, chemotaxis and the flagella are known to be important factors in the virulence as they are required for attachment and colonisation of the gut epithelium (Ketley, 1997). Once colonisation has occurred, *Campylobacter* may perturb the normal absorptive capacity of the intestine by damaging epithelial cell function either directly, by cell invasion and/or production of toxin(s), or indirectly, following the initiation of an inflammatory response (Wooldridge & Ketley, 1997). Several virulence determinants have been described to be

involved in the induction of diarrhoea; adhesion and invasion molecules, outer membrane proteins, lipopolysaccharides, stress proteins, flagella and motility, M cells, iron acquiring mechanisms, and cytotonic and cytotoxic factors (Smith, 1996). However, their relative role and importance for development of diarrhoea it not quite clear. The ability of *Campylobacter* to invade host cells in vitro is well established and cytotoxin production is consistently reported (Ketley, 1997). Early reports of enterotoxin production have not been confirmed and thus the opinion that *Campylobacter* produce an enterotoxin is no longer widely held (Allos & Blaser, 1995; Wooldridge & Ketley, 1997). Not all strains involved in human enteritis produce toxins, and no correlation has been found between serotype and toxin production (Fricker & Park, 1989).

(3) Dose-response

The infective dose depends upon a number of factors including the virulence of the strain, the vehicle with which it is ingested and the susceptibility of the individual.

*Susceptibility*

At risk populations often include the elderly, children and individuals suffering from illnesses that compromise their immune systems (*e.g.* aids and cancer patients). As regards campylobacteriosis young adults (around 15-25 years old) appear to be more susceptible or exposed than other age groups (Blaser *et al.*, 1983; Engberg & Nielsen, 1998; Kapperud & Aasen, 1992; Stafford *et al.*, 1996).

*Vehicle*

The vehicle with which the *Campylobacter* are ingested is important for development of illness. In a volunteer feeding experiment, the illness rate was higher in volunteers given the organisms in bicarbonate as compared to milk (Black *et al.*, 1988). This can be explained by the barrier effect of the gastric acid, which is reduced when *Campylobacter* are ingested with a buffering vehicle.

*Dose-response investigations*

The infective dose of *C. jejuni* has been investigated in a few experiments involving volunteers. In one experiment a dose of 500 organisms ingested with milk caused illness in one volunteer (Robinson, 1981). In another experiment involving 111 healthy young adults from Baltimore, doses ranging from 800 to 20 mill. organisms caused diarrhoeal illness (Black *et al.*, 1988). Rates of infection increased with dose, but development of illness did not show a clear dose relation.

In another outbreak at a restaurant, the number of *C. jejuni* in the causative chicken meal ranged from 53 to 750 *Campylobacter* per g (Rosenfield *et al.*, 1985).

The mathematical relationship between the ingested dose and the probability of infection (or illness) can be applied to quantify the risk of acquiring an infection by exposure to known numbers of *Campylobacter* via a certain vehicle

*Immunity*

Patients suffering from campylobacteriosis may develop immunity for the causative *Campylobacter* strain (for a period of time). This was demonstrated in the investigation by Black *et al.* (1988), where the ill volunteers developed a serum antigen response to the

*Campylobacter* strain they had ingested and hence were protected from subsequent illness but not infection with the same strain. Required immunity may explain why employees in broiler slaughterhouse get campylobacteriosis in the beginning of an employment, but not after a while (Christenson *et al.*, 1983). In addition, a higher rate of poultry and meat process workers than the normal population have been found to have complement fixing antibody against *Campylobacter* (Jones & Robinson, 1981).

C. Exposure assessment

(1) Microbial ecology

As *Campylobacter* is a common inhabitant of the gastrointestinal tract of warm-blooded animals, faeces content will inevitably contaminate the meat during slaughter and evisceration.

In general, the number of *Campylobacter* has shown to decline during the slaughter processes, primarily as a result of the dehydration that takes place during forced chilling procedures.

Investigations of poultry processing plants have shown that *C. jejuni* is present at all stages of production due to faecal contamination and that scalding, plugging, cooling, freezing and subsequent storage cannot eliminate the organism (Oosterom *et al.*, 1983).

All *Campylobacter* species grow at 37°C. *C. jejuni* and *C. coli* have optimum at 42-45°C but do not survive cooking or pasteurisation temperatures (D-values are 0.21-2.25 minutes at 55-60°C) (ICMSF, 1996). They do not grow below 28°C and survive poorly at room temperature, *i.e.* they do not multiply in food stored at temperatures of minus 18°C to plus 28°C. Although their viability declines during chill and frozen storage, they may persist under these conditions for prolonged periods. Survival has been recorded in milk and water at 4°C after several weeks of storage and in frozen poultry after several months. They are also particularly sensitive to other adverse conditions such as drying and reduced pH. *Campylobacter* is for example inhibited at pH values below 5.1 and sensitive to salt concentrations above 1.5% (ICMSF, 1996).

Exposed to chemical or physical stress conditions *Campylobacter* have shown to revert to a "viable but non-culturably" state where the organism cannot be isolated by cultural methods but remains active (infective). Evidence for this is conflicting. Some studies have shown that viable not-culturable strains can revert to a culturable state by passage through an animal host (Jones *et al.*, 1991; Saha *et al.*, 1991; Stern *et al.*, 1994). Other studies have not been able to confirm this finding (Beumer *et al.*, 1992; Boucher *et al.*, 1994; Fernley *et al.*, 1996; Korsak & Popowski, 1997; Medema *et al.*, 1992).

(2) Prevalence in food

The incidence of *Campylobacter* in food in 1997 is seen in Table 2. This table shows that especially poultry meat is infected with *Campylobacter* (prevalences up to 85.7%). At low frequencies, *Campylobacter* has also been found in beef, pork, other meat products, raw milk and milk products, and in fish and fish products. In 1996, also oysters and mussels were found to contain *Campylobacter* at a prevalence of 11% and 58%, respectively (Anon., 1998a). Other food items, from which *C. jejuni* has been detected, are

mushrooms (Doyle & Schoeni, 1986), fresh vegetables such as spinach, lettuce, radish, green unions, parsley and potatoes (Park & Sanders, 1992).

A seasonal variation has been observed in poultry meat at retail level with the highest prevalences in summer and the lowest in winter (Rosenquist & Nielsen, 1999).

(3) Consumption data

Consumption data are needed when estimates for the exposure of *Campylobacter* in a given food item are to be calculated.

D. Risk characterisation

(1) Incidence in human medicine

Most human *Campylobacter* infections occur as sporadic single cases or as part of small family related outbreaks, but larger outbreaks have been described. Outbreaks and sporadic cases seem to have different epidemiological characteristics. For example, the sporadic cases seem to peak in summer, whereas the outbreaks (based on 57 outbreaks in the United States) seem to culminate in May and October (Tauxe, 1992).

*Age and sex distribution*

All age groups may become infected with *Campylobacter*. However, the reporting rate of campylobacteriosis is higher for young adults (around 15 - 25 years) and young children (Blaser *et al.*, 1983; Brieseman, 1990; Kapperud & Aasen, 1992; Stafford *et al.*, 1996). The high incidence rate in children may be a result of a higher notification rate in this age group as compared to adults, reflecting that parents more likely seek medical care for their children. The high incidence rate in young adults has been suggested to be due to a higher travel activity in this age group compared to other age groups (Kapperud & Aasen, 1992), a higher recreational activity including participation in water sports (Skirrow, 1987), and an increased exposure to high risk food items (Engberg & Nielsen, 1998). The higher incidence may also be a result of poor food handling practices in a population that has left the parents and still has to learn how to prepare food.

The incidence rate is higher in males than females (1.2-1.5 times), the difference being more pronounced in the younger age groups (Kapperud & Aasen, 1992; Skirrow, 1987; Stafford *et al.*, 1996). The reason for this sex difference has not been explained.

*Area distribution*

The campylobacteriosis incidence seems to be area-dependent, *i.e.* some areas in for example Denmark, Norway and New Zealand have a much higher incidence than the rest of the country (Brieseman, 1990; Engberg & Nielsen, 1998; Kapperud, 1994). In UK and New Zealand *Campylobacter* infections have occurred at a higher incidence in rural than urban areas (Brieseman, 1990; Skirrow, 1987). In Norway and Australia the opposite has been observed (Stafford *et al.*, 1996; Kapperud & Aasen, 1992). In Norway, the higher incidence in urban areas was explained by a higher proportion of imported cases in these areas as compared to rural areas (Kapperud & Aasen, 1992).

*Seasonal variation in the number of human cases*

Seasonal variations in the number of human cases has been noticed in several countries including Sweden, Denmark, Norway, UK and New Zealand with a more than doubling of the incidences in late summer (Brieseman, 1990; Kapperud & Aasen, 1992; Newell *et al.*, 1999; Skirrow, 1991). The significance of seasonality seems to increase with increasing latitude (Kapperud & Aasen, 1992). The late summer peak coincides with seasonal habits of travelling abroad, but domestically acquired infections also increase in number during this period (Engberg & Nielsen, 1998; Kapperud, 1994). The prevalence of *Campylobacter* in broilers shows a similar seasonality. However, the broiler flocks tend to peak after the human cases (Berndtson, 1996; Kapperud *et al.*, 1993; Newell *et al.*, 1999). If poultry were the primary source of human infection, it should be expected that the broilers peaked before the humans and not the other way around.

Using Penner serotyping and pulsed-field gel electrophoresis of restriction enzyme-produced DNA fragments on isolates obtained from human and veterinary cases, raw milk, chicken and untreated water (from a restricted geographical area), Hudson *et al.* (1999) found that some *Campylobacter* types dominated in summer while others dominated in winter. This finding may reflect different survival patterns among *Campylobacter* strains. The pathogenicity of the isolates were not examined, but one could speculate if at least some of the seasonality in the number of human cases could be explained by the "summer"-types being more pathogenic than the "winter"-types.

(2) Risk factors

The risk factors that have usually been associated with outbreaks of campylobacteriosis are consumption of unpasteurised milk, untreated surface water, or food, particularly poultry (Finch & Blake, 1985; Peabody *et al.*, 1997).

The risk factors of sporadic *Campylobacter* infections have been studied in several case-control studies (Adak *et al.*, 1995; Brieseman, 1990; Deming *et al.*, 1987; Harris *et al.*, 1986; Hopkins *et al.*, 1984; Kapperud *et al.*, 1992; Lighton *et al.*, 1991; Neal & Slack, 1997; Neimann *et al.*, 1998; Norkrans & Svedheim, 1982; Oosterom *et al.*, 1984; Saeed *et al.*, 1993; Schorr *et al.*, 1994; Southern *et al.*, 1990).

The most frequently identified risk factors in these studies have been

- eating undercooked poultry,
- handling raw poultry,
- (daily) contact with (diarrheic) dogs or cats, particularly young pets such as kittens and puppies,
- drinking unprocessed (raw) water,
- drinking unpasteurised milk or dairy products,
- drinking doorstep delivered milk with caps damaged by birds,
- eating barbequed poultry, pork or sausages,
- eating poultry liver, and
- journeys abroad.

Other risk factors that have been related to campylobacteriosis are consumption of contaminated shellfish (Griffin *et al.*, 1983), consumption of contaminated cucumbers (Kirk *et al.*, 1997), diabetes melitus, and medication with omeprazole and $H_2$ and $H_2$ antagonists (=anti-secretory drugs) (Neal & Slack, 1997).

Travel abroad seems to be a common cause of campylobacteriosis. In Denmark and UK travelling abroad has been estimated to account for 10-15% of the reported cases (Cowden, 1992; Mølbak *et al.*, 1999). In Sweden and Norway the estimated percentage is 40-60% (Berndtson, 1996; Kapperud & Aasen, 1992). Campylobacteriosis has mainly been associated with travel to the Mediterranean countries and Asia (Kapperud, 1994; Mølbak *et al.*, 1999; Neimann *et al.*, 1998).

Overlap is reported between serotypes of *C. jejuni* found in humans, poultry and cattle (Nielsen *et al*, 1997), humans, water and chicken (Hudson *et al.*, 1999), and humans, offal, beef, sewage and poultry (Fricker & Park, 1989), indicating that foods of animal origin may play a major role in transmitting *C. jejuni* to humans.

Although a number of risk factors have been described, these do not explain all the *Campylobacter* infections. Therefore, more work has to be directed into elucidating the epidemiology of *Campylobacter* in order to get an overview over the actual causes of *Campylobacter* infections and thereby provide a basis for a more specific control strategy.

(3) Risk quantification

So far it has not been possible to quantify the number of *Campylobacter* cases which the different risk factors give rise to. This is because only a minor part of the human cases is registered, the causative agent is seldom found, and isolates are not routinely sub-typed. Sub-typing of isolates from patients, food, production animals, and environment may contribute to elucidate causal relations.

Quantitative risk assessment is a tool to estimate the risk of illness caused by a given risk factor.

Selected parts of a quantitative risk assessment model for *C. jejuni* in chicken is available (Fazil *et al.*, 1999a), and another risk assessment on broilers is being carried out (Hartnett *et al.*, 1999). The risk assessment model carried out by Fazil *et al.* (1999b) identified the concentration of *C. jejuni* on chickens entering the process as an important determinant of risk, which implies that given current production and processing performance, the steps taken to reduce the load and prevalence prior to slaughter would significantly reduce the risk to the consumer (Fazil *et al.*, 1999b).

(4) Risk in the future

The number of human *Campylobacter* cases seems to increase in most European countries (Fig. 1). This in combination with the increasing fluoroquinolone resistance among *Campylobacter* isolates could give rise to more human cases with prolonged illness, because medical treatment is compromised by the resistance.

To reduce the risk of *Campylobacter* infections in the future, more work has to be done to elucidate the causes of the infections, including case-control studies and subspecies typing of isolates from environment, production animals, food and patients. In addition, more efforts have to be directed into reducing the *Campylobacter* prevalence in food for

example by reducing the prevalences in production animals and by optimizing production processes during slaughtering and food processing.

## II. RECOMMENDATIONS FOR RISK MANAGEMENT OPTIONS

Due to the ubiquitous distribution of *Campylobacter* in the environment, the possibilities of prevention and control in the food chain from ”stable to table” will to a great extend depend on the management in the primary production, *i.e.* the possibilities of preventing the introduction of *Campylobacter* in flocks or herds of production animals and prevention of faecal contamination of ready to eat foods like some fruits, vegetables and shellfish. Further on preventive hygienic measures along the production line from slaughter to retail level based on the HACCP-concept are now recognised as the most efficient way of controlling foodborne pathogens including *Campylobacter* (ICMSF, 1988). As *Campylobacter* are present in the environment and a wide range of foods, education and information regarding safe handling of water and foods - *i.e.* risk communication - may be considered as the most efficient preventive tool at consumer level.

Several sources of *Campylobacter* infections in humans have been revealed by case-control investigations, but since the most significant sources have not yet been pointed out and may differ from country to country the most cost-effective preventive options still have to be investigated by further research.

### A. Farm level

#### (1) Poultry

At farm level several options have been discussed for the prevention of contamination or reduction of contamination levels of live birds by *Campylobacter*. In order to validate proposed tools like vaccination and competitive exclusion further research are needed since no conclusive results have been published so far (Stern, 1994; Widders *et al.*, 1996). Until now establishing of ”strict hygienic barriers” at each poultry house seems to be the only preventive option shown to work in practice (Kapperud *et al.*, 1983; Humphrey *et al.*, 1993; Berndtson *et al.*, 1996). Hygienic barriers should as a minimum include strict hygienic routines when the farm workers enter the rearing room, avoiding partly slaughter of flocks, active pest control, avoiding contact with other animals and non authorised personnel and disinfection of drinking water if necessary. Regarding the introduction of *Campylobacter* in broiler flocks the possible benefits of restricted contact with the environment seen in the intensive broiler production could pose a paradox to the raising demands by the consumers on increased animal welfare as admittance to free areas increases the risk for *Campylobacter* exposure and colonisation of broilers due to the wide distribution of *Campylobacter* in the environment.

#### (2) Other production animals (cattle, pigs and lamb).

Due to the more extensive management routines traditionally related to this kind of production animals, the positive effect of special preventive measures regarding *Campylobacter* colonisation at farm level – beside common Good Agricultural Practice - may be considered less cost effective than attempts to reduce the contamination level at slaughter and secondary production. Investigations shows that even though a high prevalence is seen in the living animals, the frequency of *Campylobacter* positive samples of beef and pork at retail is low (Anon., 1998b).

(3) Milk

Options for preventing *Campylobacter* contamination in the primary production of milk should be based on Good Agricultural Practice *e.g.* avoiding faecal contamination, ensure effective hygienic procedures in udder care and sufficient cooling capacity at the farm.

B. Slaughter

(1) Poultry

A large proportion of the broiler flocks delivered to the slaughterhouse may harbour *Campylobacter* which means that the preventive measures at this site of production should mainly focus on a reduction of the contamination level of the broiler carcass and prevention of cross contamination. Due to the very industrialised processes and the excessive use of water related to modern broiler slaughtering these preventive measures can be hard to implement. This is indicated by a relatively high prevalence of *Campylobacter* positive samples of broiler products at retail level. Several options – *e.g.* the use of different disinfectants - have been tried in order reduce the contamination level in scalding and chilling water and on the broiler carcasses (Okrend *et al.*, 1986; Hudson *et al*,. 1987). Apparently, none of these techniques have shown a satisfactory result mainly due to the heavy organic load in the process water and due to the residence of *Campylobacter* in the deeper layer of the of skin *e.g.* the feather follicles and in the peritoneal cavity (Berndtson *et al.*, 1992). Ongoing research indicates that replacing the spinchiller by forced air cooling could reduce the level of cross contamination (Thornø, personal communication, 1999). In general, preventive measures regarding *Campylobacter* contamination in poultry at slaughter should be based on hygienic design of the production equipment and implementation of the HACCP concept.

(2) Other production animals (cattle, pigs and lamb).

The relatively low prevalence of *Campylobacter* positive samples of beef and pork at retail level compared to the high frequency of *Campylobacter* seen in live animals indicates that processes involved in slaughter and secondary production to a certain degree will reduce the level of contamination and the risk of cross contamination. The processes mainly responsible for this reduction may be the individual handling of each carcass (prevention of cross contamination) and the use of forced air cooling of the carcasses (reduction of the contamination level). Especially the forced cooling procedure where the humidity of the air is reduced has shown beneficial regarding the level of *Campylobacter* contamination since these organisms are very sensitive to a dry environment (Doyle *et al.*, 1982; Oosterom *et al.*, 1985). In general, preventive measures regarding *Campylobacter* contamination at slaughter on carcasses of cattle, pigs and lamb should be based on the HACCP concept.

(3) Milk

At the dairy level preventive measures based on HACCP should focus on a safe pasteurisation procedure and avoidance of cross contamination.

C. Secondary production, commercial caterers, transport and retail

For all kinds of foods, the main preventive measures at this level of production and distribution should be based on implementation of procedures to avoid cross contamination and temperature abuse together with procedures that will secure sufficient heat treatment in relevant food items in order to eliminate *Campylobacter* present (ICMSF, 1988; Bryan, 1990). The safety and quality of foods at this stage of production and distribution should be ensured and documented by implementation of a HACCP based quality assurance system (Schlundt, 1999).

D. Home - consumers including vulnerable groups

At the consumer level preventive measures should mainly be based on risk communication such as education and information (Foegeding *et al.*, 1996, Lammerding, 1997; Schlundt, 1999). Education and information should focus on correct handling and storage of foods and the risks associated with cross contamination and temperature abuse. Further on risks associated with ingestion of undercooked foods and contaminated drinking water should be stressed out ( Worsfold, 1997).

## III. MONITORING

The effectiveness of implemented risk management tools should be validated through monitoring and surveillance (WHO, 1997; Schlundt, 1999). Both the frequency and the level of the pathogen and the impact on the number of human cases of disease caused by the pathogen should be included. Programmes for monitoring the effect should be established at all relevant stages in the production of foods where a certain factor for the control of *Campylobacter* contamination has been implemented. Relevant sites for monitoring could be the flock prevalence at farm level, the frequency and the level of contamination in products at slaughter houses and the frequency and level of contamination in foods at retail. Changes in the number of human cases of *Campylobacter* infections should be monitored by establishing surveillance-programmes based on data generated by medical staff in both practice and hospitals.

Comparable data and methods for analysis

Comparable data regarding the presence and the numbers of *Campylobacter* in foods within and between countries greatly depends on validated and harmonised methods for analysis. Further on the ”options of choice” in risk management should be based on quantitative risk assessment (WHO, 1997) which rely on quantitative methods of analysis. Therefore the authorities should take action to ensure that such well-documented quantitative methods for analysis are developed and are available for the Member States.

In order to point out the most important sources of human *Campylobacter* infections – and thereby be able to make the right choice within the risk management options - it is important that a sufficiently discriminatory and validated method for sub-typing *Campylobacter* species is developed and implemented throughout the Member States.

## IV. TABLES

Table 1 Prevalence of *Campylobacter* in domesticated and wild animals and birds in 1997 (mod. after Anon., 1999)

| Source | Country | Prevalence ** (%) | Number of units investigated | Unit | Dominating serotypes (percentages are based on the number of positive units) |
|---|---|---|---|---|---|
| POULTRY, FOWL | | | | | |
| Fowl, all | D | 47.1 | 17 | farms | |
| Fowl, all | D | 5.0 | 334 | animals | jejuni (41%), coli (18%) |
| Poultry, all | I | 9.9 | 71 | animals | |
| BROILER | | | | | |
| Broiler | D | < 0.3 | 343 | animals | |
| broiler, at slaughter | DK | 37.0 | 1037 | samples | jejuni (76%), coli (14%) |
| Broiler | NL | 44.7* | 47 | animals | |
| broiler, at slaughter | S | 9.8 | 3641 | farms | |
| CATTLE | | | | | |
| Cattle | D | 10.2 | 10051 | animals | |
| cattle, dairy | D | < 1.4 | 74 | farms | |
| cattle, dairy | D | < 0.5 | 217 | animals | |
| Cattle | D | 0.3 | 287 | farms | |
| cattle, at slaughter | DK | 51.0 | 96 | 1 animal/herd | jejuni (96%), coli (2%) |
| cattle, bulls | FIN | < 0.3 | 367 | animals | |
| Cattle | I | 52.7* | 55 | animals | |
| Cattle | I | < 6 | 17 | animals | |
| Dairy | I | < 0.4* | 269 | animals | |
| Cattle | L | 50.0 | 40 | animals | |
| Cattle | NL | 1.4 | 141 | animals | |
| Cattle | P | 1.1* | 91 | animals | |
| PIGS | | | | | |
| Pigs | D | 0.5* | 196 | farms | |
| Pigs | D | 8.0 | 1629 | animals | coli (40%), jejuni (1%) |
| pigs, at slaughter | DK | 59.0 | 319 | 1 animal/herd | coli (95%), jejuni (3%) |
| Pigs | I | 13.1* | 61 | farms | |
| SHEEP AND GOATS | | | | | |
| Goats | D | < 4 | 28 | animals | |
| Sheep | D | 6.0 | 117 | animals | jejuni (14%), coli (14%) |
| Sheep | FIN | < 0.8 | 125 | animals | |
| Sheep | I | 0.9* | 891 | animals | jejuni (38%) |
| Goats | I | < 7 | 16 | animals | |
| Sheep | NL | < 2 | 41 | animals | |
| sheep | P | < 7 | 15 | samples | |
| SOLIPEDS | | | | | |
| Solipeds | D | 1.0 | 1488 | animals | |
| Solipeds | NL | < 0.1 | 823 | animals | |
| WILDLIFE | | | | | |
| wildlife | DK | 8 | 232 | animals | |
| deer | DK | < 4 | 24 | animals | |
| european hare | DK | 3 | 38 | animals | |
| red fox | DK | 14 | 29 | animals | |
| birds, other | DK | 12 | 25 | animals | |
| water birds | DK | 14 | 16 | animals | |
| marine mammals | DK | 55 | 11 | animals | |
| mammals | DK | 6 | 180 | animals | |
| OTHER ANIMALS | | | | | |
| dogs | D | 2.2 | 1472 | animals | jejuni (73%) |
| dogs | FIN | 12.0 | 100 | animals | |
| dogs | I | 4.4 | 46 | animals | |
| dogs | NL | 17.1 | 82 | animals | |
| cats | D | 0.4 | 751 | animals | jejuni (100%) |
| cats | NL | 0.4 | 533 | animals | |
| reptiles | NL | < 3 | 30 | animals | |
| birds | NL | < 0.2 | 468 | animals | |

** < p, no positive samples were found, p = prevalence if one positive sample; * thermophilic *Campylobacter*

Table 2. Prevalence of *Campylobacter* in food in EU in 1997 (mod. after Anon., 1999)

| Food item | **Country** | Prevalence ** (%) | Number of samples | Dominating serotypes |
|---|---|---|---|---|
| MEAT | | | | |
| meat except poultry meat | D | < 0.3 | 286 | |
| meat | I | < 5 | 22 | |
| BEEF | | | | |
| beef | I | < 7 | 15 | |
| at retail, not heat treated | DK | 0.7 | 516 | |
| beef | S | < 1 | 100 | |
| beef | UK (N.IR.) | 15.0* | 320 | jejuni (60%), coli (19%) |
| PORK | | | | |
| pork | D | < 0.6 | 165 | |
| at retail, not heat treated | DK | 1.0 | 433 | |
| pork | I | < 8 | 13 | |
| pork | S | < 1 | 97 | |
| OTHER MEAT | | | | |
| wild game | D | < 10 | 10 | |
| different types of food; beef, pork and broiler | S | 1.51 | 529 | |
| MINCED MEAT AND PREPARATIONS | | | | |
| minced meat and meat preparations | A | < 3 | 37 | |
| meat preparation, raw material | D | < 0.4 | 254 | |
| meat preparation | I | < 1 | 99 | |
| MEAT PRODUCTS | | | | |
| meat products, heat treated | D | < 1 | 103 | |
| meat products, treated other than heat | D | < 2 | 61 | |
| meat products | P | 6.0* | 67 | coli |
| meat products, dried and fermented | UK (E&W) | < 0.2 | 455 | |

Table 3. Prevalence of *Campylobacter* in food in EU in 1997 (mod. after Anon., 1999) ctd.

| | | | | |
|---|---|---|---|---|
| POULTRY MEAT | | | | |
| poultry meat | A | 10.5* | 19 | |
| poultry meat ready for consumption | A | 14.3 | 14 | jejuni |
| poultry meat | D | 20.1 | 812 | jejuni (75%), coli (21%) |
| poultry meat products | D | 2.5 | 40 | jejuni |
| poultry meat, at retail, not heat treated | DK | 33.0 | 676 | |
| broiler cuts, at retail | F | 10.5 | 114 | |
| poultry meat | I | 1.9* | 52 | |
| poultry meat, at retail | I | < 8 | 12 | |
| poultry meat, chilled, fresh, at retail | NL | 31.7 | 1314 | |
| poultry meat ready for consumption | P | 85.7* | 28 | jejuni (50%), coli (50%) |
| swabs of poultry carcasses | P | 73.3* | 60 | jejuni (52%), coli (48%) |
| poultry meat at retail | P | 84.2* | 19 | jejuni (38%), coli (62%) |
| EGGS | | | | |
| eggs | A | < 8 | 12 | |
| MILK | | | | |
| raw | A | < 1.4 | 73 | |
| raw, at farm | D | 1 | 257 | jejuni |
| raw, certified | D | < 0.2 | 542 | |
| raw | I | < 5 | 19 | |
| pasteurised | D | < 4 | 23 | |
| UHT/sterilised | D | < 8 | 12 | |
| MILK PRODUCTS | | | | |
| milk products | A | < 2 | 49 | |
| milk products | D | 1 | 89 | jejuni |
| raw milk products | D | < 1.4 | 74 | |
| FISH AND PRODUCTS | | | | |
| fish and products | D | 1.1 | 90 | jejuni |

** < p, no positive samples were found, p = prevalence if one positive sample

* thermophilic *Campylobacter* N.IR. = Northern Ireland, E&W = England and Wales

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### *13.1.2. Annex I.b : Salmonella*

## I. RISK ASSESSMENT

A. Hazard Identification

Salmonellosis is the main cause of food borne human gastroenteritis in most European countries. It causes symptoms with a wide range of severity, from mild stomach upsets through varying degrees of enteritis to septicaemia and, in extreme cases, death. In some people the infection remains sub-clinical with no observed effects, and thereby effectively making them simply carriers of the causal organism.

Infection is most commonly associated with the consumption of meat (especially poultry and pork) and eggs and their products. It can enter the food chain at any point, from livestock feed, the on-farm production site, at the slaughterhouse or packing plant, in manufacturing, processing and retailing of food, through catering and food preparation at home. Although the presence of *Salmonella* anywhere in this food chain represents a potential hazard it is not necessarily passed on from one point to another so an infected food chain does not necessarily imply either infected food or cases of ill-health.

From the large number of *Salmonella* sp. that have been incriminated as or are potentially zoonotic, current research, and thus this report, is biased towards the relatively few serotypes and strains that are considered as frequent hazards (both in the past and in the foreseeable future).

(1) Characteristics of the organism

The genus *Salmonella* is a typical member of the family *Enterobacteriaceae* and consists of Gram-negative, oxidase negative bacteria, with small rod-shaped cells, straight-sided and not exceeding 1.5μm in width (Stanier *et al.*, 1986). Most *Salmonella* sp. are motile with peritrichous flagellae.

Members of the genus are responsible for diseases of humans and animals. The degree of host adaptation varies and affects the pathogenicity for humans in three ways: 1) Serotypes adapted to humans, such as *S. typhi* and *S. paratyphi*, usually cause grave diseases with septicaemic-typhoid syndrome (enteric fever); these serovars are not usually pathogenic to animals, 2) Ubiquitous serotypes, such as *S. typhimurium* and *S. enteritidis*, which affect both humans and a wide range of animals, cause usually foodborne gastrointestinal infections of varying severity, 3) Serotypes which are highly adapted to an animal host such as *S. abortus-ovis* (sheep), *S. gallinarum* (poultry), *S. cholerae-suis* (pigs), and *S. dublin* (cattle) may produce no, mild or serious disease in humans (Acha and Szyfres, 1987). The non-host adapted serovars are those with principal zoonotic significance.

(2) Reservoir

The principal reservoir of *Salmonella* sp. is the gastrointestinal tract of mammals and birds. *S. enteritidis* and *S. typhimurium* are the serotypes most frequently isolated from poultry and other farm animals, respectively (Annex II.b). Animals infected with the non-host adapted *Salmonella* sp. are usually asymptomatic carriers. Some of them, however, may exhibit clinical signs of low or moderate severity. *Salmonella* sp. were also isolated from clinically healthy cold-blooded animals such as little turtles kept as house pets, from

dogs and cats, from wild birds (Acha and Szyfres, 1986) and from invertebrates such as snails and cockroaches (D'Aoust, 1989; Devi and Murray, 1991). *Salmonella* sp. are able to survive and even multiply in the external environment and water (D'Aoust, 1989).

B. Hazard characterisation

(1) Disease

Infections with the non-human adapted *Salmonella* sp. are characterised by febrile gastroenteritis, *i.e.* diarrhea, stomachache, fever (up to 40°C), headache, nausea, vomiting and malaise. The first symptoms will appear after 12-24 h (range 5-72 h) and continue for about 3-4 days (range 2-7 days) (Baird-Parker, 1990; Flowers, 1988).

*Complications*

In approximately 5% of cases, sequellae arise (*e.g.*, septicaemia, endocarditis, multiple abscesses, polyarthritis, osteomyelitis). In about 2% of these complicated cases, the patient dies. Death usually occurs as a result of dehydration, severe kidney failure and/or sepsis and shock (Baird-Parker, 1990; Baird-Parker, 1994; Kvenberg and Archer, 1987; Lester *et al.*, 1991; Murray, 1987).

*Reduced sensitivity to antibiotics*

Reduced sensitivity of certain strains to antibiotics may not only prolong the duration of clinical disease but also affect the incidence of sequellae or death (Baggesen *et al.*, 1999). Currently, *S. typhimurium* DT104 is the most frequently reported strain with reduced sensitivity to a wide spectrum of antibiotics, lately encompassing fluoroquinolones (Davies and Funk, 1999). The strains with reduced sensitivity exhibit invasiveness which appears to be correlated with attributes such as heat and acid resistance, ability to survive in aerosols or on surfaces, and enhanced resistance to disinfectants (Humphrey, 1998). The frequency of their detection in farm animals and in humans is increasing (UK Veterinary Laboratories Agency, 1998; Baggesen *et al.*, 1999).

(2) Virulence / pathogenicity

The genetic control of many aspects of *Salmonella* virulence has not been elucidated. However, both plasmid and chromosomal genes appear to be involved. *Salmonella* sp., with the exception of *S. typhi*, carry a 50-60 Mda plasmid which has been associated with virulence in a number of serovars. The primary virulence plasmid phenotype is in the ability to spread beyond the initial site of infection (Gulig, 1990). Chromosomal genes are also important in determining virulence and appear to play a major role in determining the ability to survive and multiply in cells of the reticulo-endothelial system (Barrow, *et al.*, 1989). The *phoP* gene has been identified as being necessary for virulence and survival and may be a regulatory sequence necessary for the expression of a number of virulence factors (Fields, *et al.*, 1989).

*Salmonella* is usually orally ingested. It adheres to the epithelial cells in the ileum by means of mannose-resistant fimbriae and invades the host. Penetration of the epithelial cells is by receptor-mediated endocytosis, although *Salmonella* may also penetrate the epithelium at the boundary between adjacent cells (Williams, *et al.*, 1989; Ernst, *et al.*, 1990). It produces at least three enterotoxins and a cytotoxin (Mims, 1987). Bacterial multiplication occurs in the lymphoid tissue, at a rate which depends on the virulence of

the strain and the genetic background of the host (Zhang-Barber *et al.*, 1999). Its ability to resist both oxidative and non-oxidative phagocytic killing is probably due to cell wall components and especially to the lipopolyssacharide (LPS). The resistance of *S. typhimurium* to non-oxidative killing is directly proportional to LPS's length and complexity (Stinavage *et al.*, 1989). Loss of LPS during conversion of *S. enteritidis* phage type 4 to phage type 7 was correlated with loss of virulence (Chart *et al.*, 1989). Outer membrane proteins (OMPs) are involved in the oxidative killing (Stinavage *et al.*, 1989). Porins, a major type of OMPs, have been shown to interact with the macrophage membrane, decreasing the oxidative burst and hydrophobicity (Tufano *et al.*, 1989). The underlying mechanism appears to be activation of the adenylate cyclase system.

(3) Dose-response

The infective dose in healthy people varies according to serotype, strain as well as the type of food involved. Human adapted serotypes have been considered for many years to have lower infective doses than non-adapted serotypes. Less than $10^3$ *S. typhi* organisms caused a high proportion of outbreaks (Blaser and Newman, 1982). For non-adapted serotypes, there are grounds to believe that the volunteer feeding experiments used to determine infective doses had overestimated the number needed to initiate infection. Experimental studies have consistently indicated that a dose of $10^5$ to $10^7$ organisms is required to initiate infection. However, outbreak data showed that between $10^1$ and $10^{11}$ (median $10^2$) cells caused illness (see Table 1).

Further, it has been suggested that the infective dose is lower in foods of high fat or protein content, due to protection of *Salmonella* sp. from gastric acidity (Fontaine, *et al.*, 1980; Blaser and Newman, 1982).

**Table 1:** Examples of *Salmonella* infections where the number of cells ingested were less than a thousand (Blaser and Newman, 1982).

| Serotype | Estimated dose in cells | Vehicle |
|---|---|---|
| *S. typhimurium* | 17 | Water |
| *S. newport* | 60 | Hamburger |
| *S. eastbourne* | 10 | Chocolate |
| *S. heidelberg* | 10 | Cheese |

(4) Immunity

The mechanisms of development of immunity to *Salmonella* are still unclear. Both humoral and cell-mediated immunity are involved. From the empirical work on vaccine development it is shown that humoral immunity offers protection from clinical disease after challenge with the vaccine strain but usually not with heterologous strains or serotypes. Cell mediated immunity, on the other hand, plays a major role in gut tissue clearance (Zhang-Barber *et al.*, 1999).

B. Exposure assessment

(1) Microbial ecology

Contamination of the meat usually occurs during the slaughtering procedure through direct or indirect contact with the content of the gastrointestinal tract of carrier animals.

Eggs can acquire *Salmonella* by two routes, transovarian or trans-shell transmission. *Salmonella* sp. acquired from infected ovaries or oviduct tissue are introduced before shell formation and as such are present in the egg's interior (mainly *S. enteritidis*). Trans-shell transmission involves deposition of faecally-derived *Salmonella* on the shell and subsequent penetration into the egg's interior. An infection restricted to the ovaries results in an infected yolk; whereas an infection of the oviduct leads to the deposition of *S. enteritidis* in the albumen. The temperature and duration of the subsequent storage of contaminated eggs affects their *S. enteritidis* numbers. Storage at temperatures $\leq 12^{o}C$ prevents both the replication in the albumen and the migration from the albumen to the yolk (Brandshaw *et al.*, 1990; Braun and Fehlhaber, 1995).

Foodborne salmonellosis (due to *S. stanley, S. newport, S. infantis, S. anatum, S. seftenberg, S. havana, S. mbandaka*) may result from consumption of contaminated sprouts (mainly alfalfa sprouts). Sprout-seeds contaminated because of (1) the use of contaminated manure as fertilizer, (2) the use of fecally contaminated agricultural water, (3) poor agricultural and manufacturing practices are considered the sources of the human infections. Conditions during sprouting of these seeds (temperature, pH, $A_w$ etc.) are ideal for growth of *Salmonella* sp. (National Advisory Committee on Microbiological Criteria for Foods, 1999).

*Salmonella* sp. optimum growth occurs at 37°C (Jay, 1996). The lowest temperatures at which growth has been reported are 5.3°C for *S. heidelberg* and 6.2°C for *S. typhimurim* (Matches and Liston, 1968). Temperatures of around 45°C have been reported to be the upper limit for growth. The most heat resistant is *S. senftenberg 775W* (Dvalue=1.2 sec in 71.7°C). The heat resistance increases markedly at low $A_w$ levels particularly in foods which also have a high fat content (ICMSF,1980). *Salmonella* sp. numbers decline during frozen storage, the rate being greater at temperatures around the freezing point of meat (-2 to -5°C) (Varnam and Evans, 1996). The pH for optimum growth is between 6.6 and 8.2, with values above 9.0 and below 4.0 being bactericidal (Jay, 1996). A minimum growth pH of 4.05 has been recorded but depending on the acid used the minimum may be as high as 5.5 (Chung and Goepfert, 1970). Aeration favors growth at low pH values (Troller, 1976). Regarding available moisture, growth inhibition has been reported for $A_w$ values below 0.94 in media with neutral pH, with higher $A_w$ values required as the pH is decreased towards growth minima (Varnam and Evans, 1996).

(2) Prevalence in food

The reported by the Member States prevalence of *Salmonella* sp. in food is shown in Annex II. Fresh poultry meat (*Gallus gallus*) is frequently found contaminated (reported prevalences at retail range from 1 to 55% in 1998). At much lower prevalences *Salmonella* sp. are also found on pork, beef, other meat products and in raw eggs and dairy products. Recently, alfalafa sprouts have also been found contaminated in several European markets (National Advisory Committee on Microbiological Criteria for Foods, 1999).

(3) Consumption data

Consumption data as reported by EU-Member States can be found in Annex II. Accurate consumption data are needed to ascertain the risk of salmonellosis associated with exposure to a given food item. Thus, these data are of major importance in the development of valid risk assessments models.

C. Risk characterization

(1) Incidence in human medicine

The reported incidence rates of human salmonellosis in the EU-countries are shown in Annex II.b. However, official statistics underestimate the real incidence of human salmonellosis (Beckers, 1987; Genigeorgis, 1981). Berends *et al.* (1998) combined data from several studies and estimated the true average incidence of salmonellosis in The Netherlands at about 450 cases per 100,000 person-years at risk (95% confidence limits (CI): 300-700 cases per $10^5$ person-years at risk). Incidence estimates of about the same magnitude can be made for other countries (Baird-Parker, 1990; Baird-Parker, 1994; Bean and Griffin, 1990; Kvenberg and Archer, 1987; Lester *et al.*, 1991). For comparison, Hald and Wegener (1999) reported that the annual incidence of registered human cases in 1997 was in Denmark 95, in Germany 128.4, and in The Netherlands 17 cases per 100,000 person-years at risk. With regard to mortality, Berends *et al.* (1998) estimated that, in the Netherlands, on average, the death rate attributable directly or indirectly to the infection is 0.4 per $10^5$ person-years at risk.

*Age distribution*

The very young and the very old are considered as being more at risk of an infection with *Salmonella* than the average adult population. However, for the latter age-group this accepted belief may partially result from information bias (eg., people in this age-group are more likely to seek medical attention and diagnostic testing than healthy young adults). Berends *et al.* (1998) reviewed several studies and concluded that in The Netherlands, healthy elderly people do not seem necessarily to be more at risk than younger adults.

*Incidence in population subgroups*

People already suffering from a disease or condition that may directly or indirectly affect their immunocompetence are more prone to an infection than people in good health, and their infection more often becomes complicated (Blaser and Newman, 1982; Baird-Parker, 1994; Ryan *et al.*, 1997). From the data of Lester *et al.* (1991), Berends *et al.* (1998) calculated that the odds ratios of ‘underlying disease’ as a risk factor in `arising sequellae' and as a risk factor in ‘dying from this infection’ are 3.8 (95% CI: 1.8-8.2) and 3.6 (95% CI: 1.3-10.7), respectively. The same authors reported that the annual number of cases of salmonellosis per $10^5$ person-years at risk in the group of people with `underlying diseases' may roughly be estimated at 1200, that of arising sequellae at 60 and that of death at 1.2.

Use of antacids or insufficient gastric acid production (achlorhydria) can also be a risk factor. Berends *et al.* (1998) used the data of Riley *et al.* (1984) and calculated that the odds ratio of `excessive use of antacids' as a risk factor in becoming infected with *Salmonella* is 3.6 (95% CI: 1.1-10.4). The annual incidence of salmonellosis amongst

those who use antacids almost daily, or who suffer from achlorhydria, was estimated at about 1100 cases per $10^5$ person-years at risk (Berends *et al.*, 1998).

The administration of antibiotics with a disturbing effect on the normal gut flora, such as tetracyclines or broad spectrum penicillins, can lead to significantly more infections with *Salmonella*, both in animals and man (Pavia *et al.*, 1990). This effect is especially important in the first week after the last administration of these antibiotics. Berends *et al.* (1996) who did a stratified analysis of the data of several studies calculated a Mantel-Haenszel-corrected odds ratio of the previous use of broad spectrum antibiotics as a risk factor in becoming infected with *Salmonella* of 5.6 (95% CI: 4.4-7.5). The annual incidence of salmonellosis amongst persons who have recently used antibiotics with an adverse effect on the composition of the gut flora may be about 1700 cases per $10^5$ person-years at risk.

People who come into close contact with live animals, animal excrement, animal products or patients suffering from salmonellosis are potentially more at risk of an infection than others (Flowers, 1988; D'Aoust *et al.*, 1990; Barrow, 1992; Davies and Renton, 1992). Unfortunately there are no investigations where the higher risk incurred by farmers, slaughterline personnel, caterers or nurses is adequately quantified.

(2) Risk factors

The risk factors that have been associated with outbreaks of foodborne salmonellosis are consumption of raw eggs, inadequate cooking, improper cooling of cooked foods, inadequate reheating, delayed serving, cross-contamination between raw and cooked foods, inadequate cleaning of kitchen equipment, inadequate curing, improper hot holding and to a limited degree infected persons (Genigeorgis, 1986).

(3) Risk quantification

Risk quantification is the first part of risk analysis, the other being risk management and risk communication, targeting at the technical description and estimation of the probability of an undesired event. The adequacy of the technical description largely depends on the availability and quality of data and the validity of the distributional assumptions that are built-in the models employed.

The literature on risk assessment of foodborne pathogens is sparse. Buchanan and Whiting (1997) reason that this is because of lack of knowledge in accurate dose-response relationships and difficulty in estimating the actual levels of pathogenic microorganisms ingested by consumers because levels of microorganisms in foods can change drastically within a short time. They developed a model for assessing the risk of salmonellosis due to *S. enteritidis* in pasteurized liquid eggs. After testing different scenaria they identified that firstly the pasteurization temperature and secondly the pasteurization time is the most critical point for substantial risk reduction. Wachsmuth (1999) presented a model with deterministic elements for the risk of human salmonellosis due to *S. enteritidis* from eggs in the USA, which estimated that immediate cooling and storage of eggs at 7.2°C may reduce the incidence of human disease by 12%. Recently, the structure of a model for the risk assessment of *Salmonella* transmission within primary pork production in Denmark (endpoint: carcass in chilling room) has been presented by Staerk *et al.* (1999). Since the hazard characterization has not been finalized yet no recommendations have been published.

(4) Risk in the future

The human incidence within the EU remains high (see Annex II.b). A declining trend appears in Denmark where a national monitoring and control program has been applied in poultry flocks and in pig herds (Annual Report on Zoonoses in Denmark, 1998). The ubiquitous nature of *Salmonella* and the expansion of the trade among Member States as well as between the EU and third countries will challenge the efficacy of national programs.

An alarming event is the emergence and the possible dominance of human cases by multi-resistant strains, as *S. typhimurium* DT104. These strains are detected with increased frequency among animal and human populations and are expanding their spectrum of resistance.

To control the risk of human cases more attention has to be paid to the detection of subclinical salmonellosis and the control of its transmision on the farm and onto the food chain. To this respect, further research on the epidemiology of this transmission coupled with improvement of diagnostic methods targeting the serotypes with high human significance is of utmost importance.

## II. RECOMMENDATIONS FOR RISK MANAGEMENT OPTIONS

Introducing the recommendations for risk management options we have to acknowledge that the 'stable-to-table' concept of risk management for an organism with ubiquitus distribution in nature, as is *Salmonella*, should realize that all participants in the chain of food production and consumption bear some responsibility for reducing the risk of foodborne disease. The partitioning of the overall responsibility and the implementation of appropriate control measures across the continuum is rather obscure. For practical rather than scientifically sound reasons, formal recognition of specific food safety responsibilities continues to be concentrated on the slaughter/processing and, to a lesser extent, retail/distribution segments of the continuum. The logical appeal of controlling foodborne salmonellosis by reducing the prevalence of *Salmonella* sp. - carrying animals is hampered by the fact that the current epidemiological knowledge on risk factors for spread of *Salmonella* throughout farm animal populations is poor.

A. Farm level

Two models for control can be distinguished: 1) Exclusion – preventing the introduction of *Salmonella* sp. into the population, and 2) Non-exclusion – accepting the introduction of *Salmonella* sp. into the population, and introducing measures to reduce infection transmission during production.

(1) Exclusion

The large host-range of *Salmonella* and its ability to survive and even multiply in the external environment (D' Aoust, 1989) presents a challenge to rearing animal populations free of *Salmonella*. In the USA, McCapes and Riemann (1998) pointed-out that prevention of *Salmonella* introduction into poultry flocks would require complete re-design and re-construction of production systems based on principles of sanitary engineering, and incorporating sanitation, security and surveillance measures. However, the technical feasibility of preharvest control of *Salmonella* has been amply demonstrated

by the Swedish and the Danish poultry and swine industries (Wierup, 1997; Annual Report on Zoonoses in Denmark, 1998).

Available biosecurity measures are not attempting to completely exclude *Salmonella* introduction into animal populations but rather to minimize the risk of introduction. These measures are of high importance to those poultry flocks and swine herds that have been shown in large-scale monitoring and surveillance programs to be practically free of *Salmonella* (Annual Report on Zoonoses in Denmark, 1998). They are, however, less crucial in populations that are highly contaminated.

*Salmonella* control in animal feed is part of risk minimization programs currently in effect (Wierup, 1997; Annual Report on Zoonoses in Denmark, 1998). Without disputing the role of feed as a source of infection there are, however, some grounds on which to question the current monitoring and control protocols. In poultry, the serotypes of overriding human health significance (*S. enteritidis* and *S. typhimurium*) are relatively uncommon in feed compared with other serotypes (McIlroy, 1998). Similarly in Denmark, where *S. typhimurium* is of greatest significance in pigs and in people, this serotype is rarely found in swine feed (Stege *et al.*, 1997a). These observations may point to the fact that although *Salmonella* surveillance in animal feed should continue to encompass all *Salmonella* sp. (Wierup, 1997), *Salmonella* control measures should be focused upon serotypes responsible for most human disease.

Monitoring and control of *Salmonella* in parent-animal populations is a second important part of effective risk minimization programs (Wierup, 1997; Annual Report on Zoonoses in Denmark, 1998). Although indisputable for poultry production, the role of *Salmonella* infected pig breeders for the infection of finishing pigs has been recently questioned. In Denmark, removal of 10 week-old pigs from breeding farms infected with *S. typhimurium* to clean finishing facilities appeared to be effective in preventing infection at market age (Dahl *et al.*, 1997). In North Carolina herds, different serotypes were predominating in the breeding and in the finishing sections of farrow-to-finish herds (Davies *et al.*, 1998) and the serotype profile of fecal samples of batches of pigs followed from the nursery to the end of finishing were found to change (Davies and Funk, 1999). The evidence raise questions about the likely benefits to be gained, at the market pig level, through intensive monitoring and control of all *Salmonella* sp. in breeding stock. Rather, there may be merit in concentration of monitoring and control efforts against those serotypes (and even strains) with epidemiologic significance for human salmonellosis.

(2) Non-exclusion

Traditionally, measures to control the spread of *Salmonella* sp. after they were introduced into an animal population have been based on principles of improved hygiene and management (*e.g.* all-in all-out management, cleaning and disinfection between successive batches, rodent control, hygiene of personnel) that theoretically should reduce the transmission among animals of organisms shed in faeces of carriers (Berends *et al.*, 1998; Davies and Funk, 1999). While these rigorous measures have proven effective in many instances they failed in others (Wierup, 1997; Davies and Funk, 1999). Thus, we can argue that we do not have a set of control procedures that can be applied to contaminated commercial farms to invariably control *Salmonella*. Davies and Funk (1999) argued that traditional approaches target control of *Salmonella* outside the animal while the epidemiology of transmission is dominated by *Salmonella* infection inside the animal and animal-to-animal transmission. Two pieces of recent scientific information substantiate their argument; one adding to the current control options the other doubting them.

Field studies in Denmark, The Netherlands, Germany and Greece showed that feed related factors affected the seroprevalence of *Salmonella* in finishing pigs. Specifically, home-mixing was preventive factor compared to purchase of final feed (Stege *et al.*, 1997b). Also, preventive were wet-feed compared to dry-feed (Stege *et al.* 1997b; Dahl, 1998), non-pelleted dry-feed compared to pelleted dry-feed (Lo Fo Wong *et al.*, 1999), coarsely grounded compared to finely grounded feed (Wingstrand *et al.*, 1996), and addition of organic acids to water (Hansen *et al.*, 1999; Wingstrand *et al.*, 1996). Most likely coarse, non-pelleted or wet-feed have a beneficial effect on *Salmonella* transmission because of improved gastric health of pigs (Joergensen *et al.*, 1999).

The phenomenon of aerosol infection with *Salmonella* has been unequivocally demonstrated in several species (Fedorka-Cray *et al.*, 1995; Humphrey, 1998; Humphrey *et al.*, 1992; Wathes *et al.*, 1988; Wray and Davies, 1998). The most compelling evidence for the importance of aerosol transmission stem from two experimental studies in poultry and in pigs; one demonstrating that about 3% of eggs layed by hens orally challenged with $10^7$ *S. typhimurium* were infected, compared with 14% following aerosol challenge with 200 organisms (Humphrey, 1998) and the other demonstrating *S. typhimurium* in the intestinal tracts and lymph nodes of oesophagotomized pigs within 2-4 hours of aerosol exposure (Fedorka-Cray *et al.*, 1995). In addition to low-dose aerosol infection low-dose dust infection via the conjuctival route has also been demonstrated in poultry (Wray and Davies, 1998). Since these evidences are produced in experimental studies it is still unknown what is the relative importance of airborne transmission of *Salmonella*, compared with ingestion, on farms and during transport and lairage.

(3) Competitive exclusion

An adult-type microflora of the intestinal content can be established in young chicks by oral administration of suspensions or anaerobic cultures of gut contents from mature, *Salmonella*-free birds. In this way chicks become more resistant to an orall challenge with *Salmonella* sp. (Schleifer, 1985). Currently, several commercial preparations are available and can be administered in the hatchery to protect chicks at the earliest possible opportunity. This treatment is non-specific and offers protection against several serotypes.

(4) Vaccination

The development of efficacious vaccines and the use of vaccination for control of on-farm salmonellosis are hurdled by lack of adequate information regarding colonization and immunity of animals to the *Salmonella* serotypes that are usually associated with human disease. On empirical grounds the development of killed or attenuated vaccines against non-host specific serotypes has been attempted and the vaccines have been used in poultry and in pig farms (Zhang-Barber *et al.*, 1999; Davies and Funk, 1999). Reported results of field experiments exhibit variation in the vaccines' efficacy to prevent *Salmonella* infection, colonization and shedding (McCapes *et al.*, 1967; Truscott and Friars, 1972; Truscott, 1981; Thain *et al.*, 1984; Ghosh, 1989; Timms *et al.*, 1990; Gast *et al.*, 1992; Gast *et al.*, 1993; Gibson *et al.*, 1999). Vaccinal immunity appears to be serotype-specific. When coupled with improved attention to husbandry hygiene vaccination of broiler parent stock against *S. enteritidis* appears to offer in the reduction of its vertical and horizontal transmission.

B. Slaughterhouse

Contamination of carcasses and cross-contamination of cutts occurs because *Salmonella*-infected animals are being slaughtered. Therefore, the risk of contamination of the meat cannot be eliminated under current slaughtering procedures. Implementation of Good Manufacturing Practices which are based on proper Critical Control Point analyses will, however, at best contain the increase of prevalence of contaminated carcasses and cuts (Mousing *et al*, 1997).

(1) The animal as a risk factor

The faeces are particularly important in relation to carcass contamination. There is a strong correlation between the proportion of animals with *Salmonella* sp. in their faeces and the proportion of contaminated carcasses at the end of the slaughterline (Oosterom and Notermans, 1983; Oosterom *et al.*, 1985). Berends *et al.* (1997) calculated that pigs with *Salmonella* in their faeces are 3-4 times more likely to end up as a positive carcass than pigs that are not carriers. Roughly the same estimate applies also to calves with *Salmonella* in their faeces (Berends *et al.*, 1997). About 70% of all carcass contamination results from pigs themselves being carriers and about 30% because other pigs in the line are carriers (Oosterom and Notermans, 1983; Berends *et al.*, 1997).

(2) The process as a risk factor

The current slaughtering process of all animals is a large *Salmonella* prevalence amplifier. Although Good Manufacturing Practices based on the HACCP principles are highly recommended one have to acknowledge that there are actually no steps in the process intentionally designed to reduce the hazard of carcass contamination. Investigators have proposed some risk management measures (Borch *et al.*, 1996; Berends *et al.*, 1997). One of them, covering of the bungs with a plastic bag the moment the anuses are cut loose, has been incorporated into the pig and calf slaughter-lines in Danish slaughterhouses favorably affecting the prevalence of contaminated carcasses (Mousing *et al.*, 1997; Annual Report on Zoonoses in Denmark, 1998). Another one, slaughtering of heavily infected flocks or herds in the end of the day and taking special precautions to reduce the hazard of meat contamination seems to reduce the risk of cross-contamination and the overall prevalence of infected meat (Hald *et al.*, 1999).

(3) The slaughterhouse environment as a risk factor

The hygiene condition of walls, floors, ceilings or human carriers present, are usually unimportant factors with respect to carcass contamination with *Salmonella* in the slaughterline.This is substantiated by the fact that in most of the cases the *Salmonella* sp. found on the carcasses were only associated with animals slaughtered that day (Berends *et al.*, 1997; Limpitakis *et al.*, 1999)

C. Secondary production, commercial caterers, transport and retail

At this level of production and distribution the preventive measures should target known risk factors (*e.g.* cross-contamination, improper cooking and cooling, improper curing, improper preservation temperature) (Genigeorgis, 1986). The safety of foods should be ensured and documented by implementation of a HACCP-based quality assurance system.

D. Home-consumers

Although the food industry has the responsibility for the production of non-contaminated food, failings in the supply chain can occur and hence, there will always be some risk for *Salmonella* sp. -contaminated food to reach the consumer. Thus people should be educated to avoid cross-contamination and to be more meticulous in food preparation and preservation in the home. This will ensure safer food regardless of failings earlier in the supply chain.

## III. SURVEILLANCE AND MONITORING

The establishment of comparable surveillance and monitoring programs for animal salmonellosis throughout the EU-Member States should be considered. These programs will provide the means to: 1) partition the overall responsibility and accountability of preventive measures across the farm-to-fork continuum, 2) alert the scientific community and the relevant authorities about emerging risks, 3) evaluate the efficacy or the failures of currently applied risk reduction options, and 4) allocate control expenditures to those options with the highest benefit to food safety. They should be established at 3 sites, the farms, the slaughterhouses and at retail. Their aim should be the reduction of the prevalence of *Salmonella*-infected animals and products.

An important prerequisite for these programs is the establishment of common serological (for farm and slaughterhouse surveillance and monitoring) and microbiological (for slaughterhouse and retail surveillance and monitoring) methods and protocols. The characteristics of these methods should be continuously evaluated and improved with special attention to their accuracy in detecting infections with the serotypes with the highest human significance (*e.g. S. typhimurium* DT104).

Another important aspect of these programs is their dependence on scientifically sound sampling protocols that aim at producing valid prevalence estimates and not detecting 'process pitfalls' (*e.g.* oversampling of animals or products assumed at higher risk). Inevitably, prevalence data produced with the latter aim is not weighed for the prevalence of the 'process pitfalls' into the food chain. This may falsely direct preventive actions against these 'process pitfalls' and not against the prevalence of contaminated foods.

Monitoring should be sensitive to accurate and timely detection of the sources of human outbreaks, the responsible food chains, and the strains involved. This may best be accomplished by the establishment of zoonosis centers that will be able to confront foodborne salmonellosis by a multi-disciplinary approach.

### Comparable data and methods for analysis

Data regarding the prevalence of *Salmonella*-infected animals and products as well as the incidence of human cases of salmonellosis should be recorded across the EU-Member States in a harmonized manner and evaluated against standardized populations. To continuously assess the risk of salmonellosis to human health there should be a provision for genotyping a standard fraction of the most prevalent serotypes (*e.g. S. typhimurium* and *S. enteritidis*) by comparable methods.

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### *13.1.3. Annex I.c : Verotoxigenic Escherichia coli (VTEC)*

## I. Risk assessment

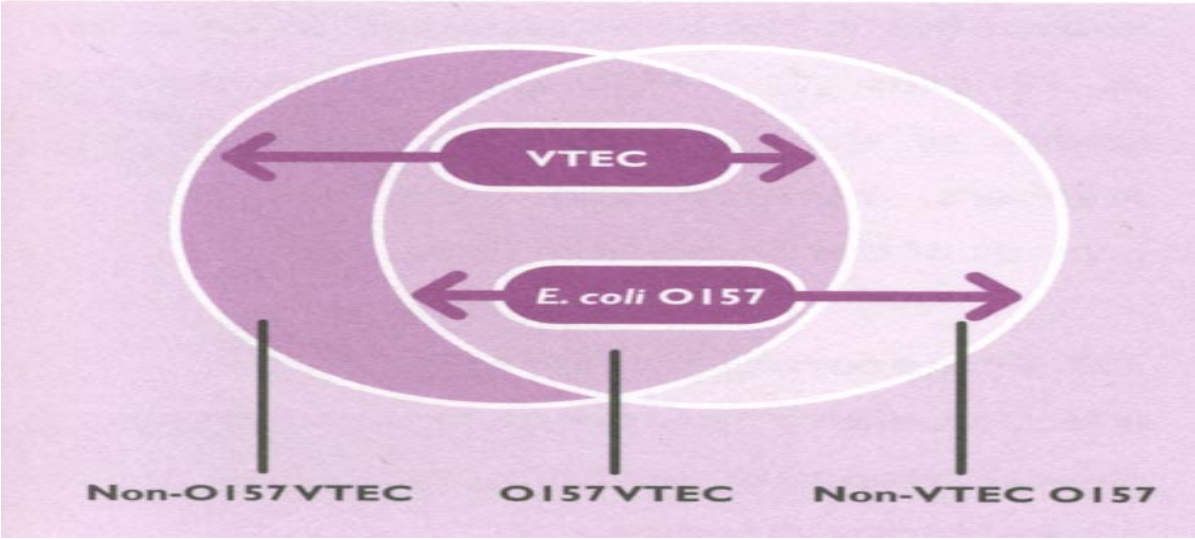
Several international bodies have published reviews on the issue of Verotoxigenic *Escherichia coli* (VTEC) such as the Advisory committee on the microbial safety (1995); the Pennington Report on the VTEC O157 outbreak in Scotland (1996); the Institute of Food Science and technology (1996) and the report of the Scientific Veterinary Committee on VTEC (1997), The WHO consultation on the prevention and control of VTEC (1997) and the Food Safety Authority of Ireland report on prevention of E *coli* O157:H7 infections (1999). It might be noted that these reviews emphasise the food borne route of transmission while other routes such as direct contact, environmental route or man to man should not be ignored.

A. Hazard identification

(1) Characteristics of the organism

**VTEC definition** – VTEC is a group of *E. coli* that produce verotoxin. This group of bacteria has many synonyms the most common one being shigatoxin producing *E. coli* (STEC) while the terms enterohaemorrhagic *E. coli* (EHEC), *E. coli* O157, *E. coli* O157:H7 are used interchangeably, resulting in confusion. In this report the term VTEC will be used, unless particular terms are used in the primary references. Disease produced by VTEC appears to be associated with a subset of strains with the serotype O157:H7 as the predominant one.(Mainil, 1999). A lot of other verotoxin producing serotypes may produce disease in humans, the most common serotypes being O26, O103, O111, and O145 (Boudailliez *et al.*, 1997, Blanco *et al.*, 1996, Tossi *et al.*, 1994, Goldwater and Bettelheim, 1998, Meng and Doyle, 1997). However, it is possible that not all VTEC are associated with human disease. Most research on VTEC has been done on the serotype O157 that is easily recognisable among other *E. coli* strains by its inability to ferment sorbitol.

Fig. 1 Schematic representation of the relationship between *E. coli* O157 and VTEC adapted from ACMSF report (1995) on VTEC. London: HMSO. (from "The prevention of *E. coli* 0157:H7 infection - a shared responsibility". Food Safety Authority of Ireland (1999).



All other VTEC serotypes are phenotypically similar to the harmless *E. coli* strains inhabiting the gastrointestinal tract of humans and all warm-blooded animals. This means that our knowledge about the disease caused and the sources of non-O157 VTEC are rather scarce and inadequate. Compared with other *E. coli*, the VTEC O157 appears to have ruminants as its reservoirs, but it has also been isolated from pigs, dogs, cats, horses, sea gulls and geese. The VTEC O157 bacteria has been reported to survive for months on straw, wood surfaces and in water.

(2) Reservoir

**Animal species** - *Escherichia coli* occurs in all species causing diarrhoea, generalised infections and mastitis (Anonymous, Scientific Veterinary Committee, 1997). The VTEC O157 appears to have ruminants as their reservoirs (Chapman *et al.*, 1997 and Wray *et al.*, 1993), while it has also been isolated from pigs, dogs, cats (Mainil, 1998) horses, sea-gulls and geese (Anonymous, Scientific Veterinary Committee, 1997).

**Route of transmission**. In principle 4 routes of infection could be identified person to person (Mead and Griffin, 1998); food-borne (Armstrong *et al.*, 1996) such as meat (Pennington report, 1996) and not pasteurised milk (Upton and Coia, 1994, Chapman *et al.*, 1993); environment such as swimming in a lake or pool (Brewster *et al.*, 1994, Paunio *et al.*, 1999); direct contact with farm animals (Morgan, 1998, Milne *et al.*, 1999, Schukla *et al.*, 1995, De Jong, 1998).

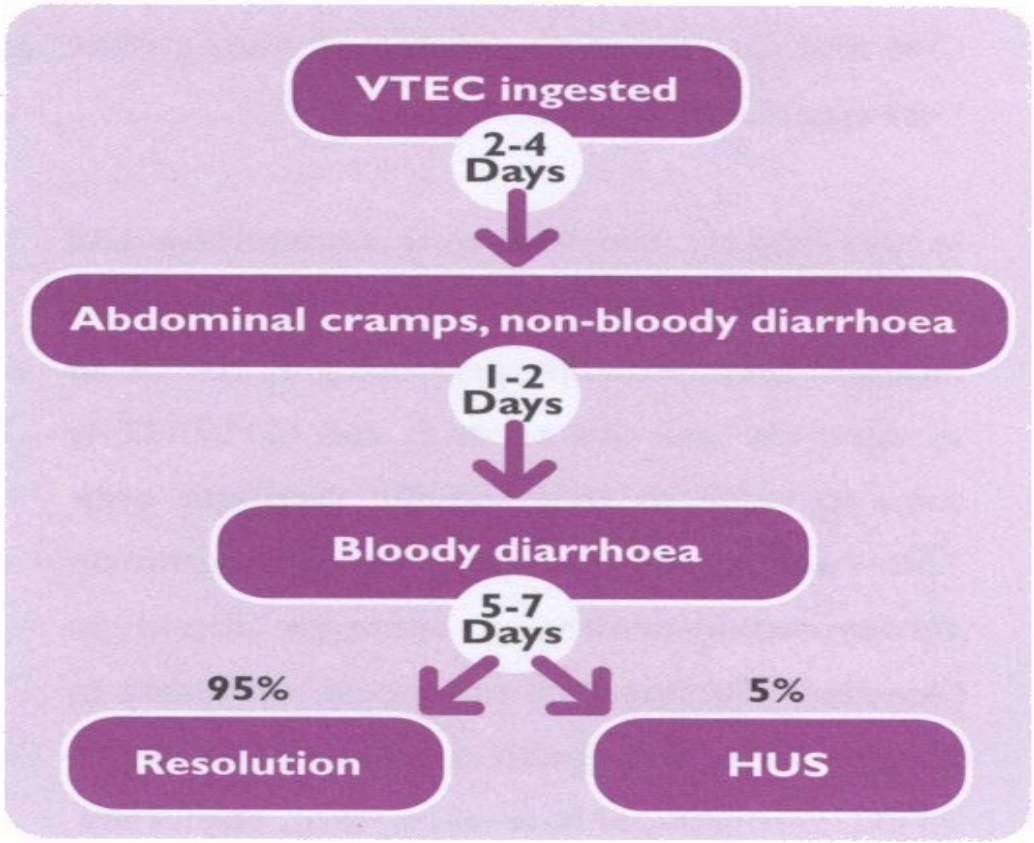
B. Hazard Characterisation

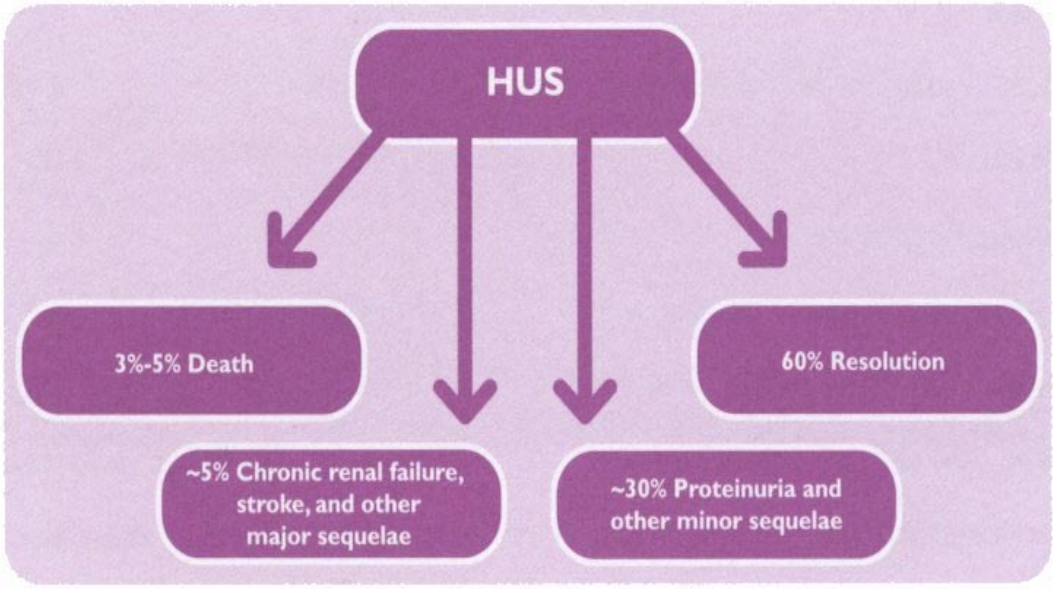
(1) Disease

According to Mead and Griffin, 1998, the clinical manifestations range from symptom free carriage, diarrhoea, haemorrhagic colitis, haemolytic uraemic syndrome (HUS) and death. Haemorrhagic colitis was often associated with abdominal cramps, bloody stools, but little or no fever. The average period between exposure and illness period was 3 days, but incubation periods between 1 and 8 days have been described. Most patients recover within 7 days. The illness typically starts with abdominal cramps, and non bloody diarrhoea the first 2 days, which might become bloody during the next 1-2 days in around 2/3 of the clinical cases (Slutsker *et al.*, 1997), while vomiting might occur in 1/3 to 2/3 of the cases.

The absence of fever, might lead clinician to suspect non-infectious diseases such as intussuception, ischemic colitis or appendicitis that might prompt exploratory surgery. Between 3-20% of the cases progress into HUS after typically 6 days after onset of diarrhoea (Slutsker *et al*, 1997, Wall *et al.*, 1996) see Fig 2. Another complication is thrombotic thrombocytopenic purpura (TTP) . Among the patients with HUS 3-5% die acutely and a similar percentage develop end-stage renal disease (Siegler, 1995). However, Reilly (1998) noted that in certain outbreaks among elderly the mortality could approach 50%. In humans VTEC O157 might shed in the stool for several weeks after the resolution of symptoms, and it seems that children carry the bacteria longer than adults. The median time in one study of shedding among children under 5 years of age are 17 days, while 38% were shedding for more than 20 days (Belongia *et al.*, 1993). While the bacteria does not appear to cause disease in adult ruminants, neonatal calves might be showing clinical symptoms (diarrhoea and enterocolitis) if ingesting E *coli* O157:H7 (Dean-Nystrom *et al.*, 1997).

**Fig. 2 Course of infection with VTEC and range of HUS Symptoms** (from Mead and Griffin in "The prevention of *E. coli* 0157:H7 infection - a shared responsibility". Food Safety Authority of Ireland (1999)).



(2) Virulence/pathogenicity

One of the most important characteristics is the ability of the *E. coli* bacteria to produce verotoxins (Shiga-like toxins) VT1 and VT2. The verotoxin 1 is indistinguishable from Shiga toxin produced by *Shigella dysenteriae* type 1 (Nataro and Karper, 1998). It appears in Sweden that the VTEC O157 associated with human cases produce VT2 and in some instances also VT1 (Anna Aspan Pers comm). Other virulence factors include intimin, an adhesion molecule (Nataro and Karper, 1998) and haemolysin (Law and Kelly, 1995) . However, the pathogenesis is not entirely clear and recent studies (Schmidt *et al.*, 1999) indicate that HUS and diarrhoea can be associated with E *coli* O157:H7 and H- that do not produce verotoxins. Riemann and Cliver (1997) and Sloncewski (1992) suggested that the bacteria's ability to survive in acidic environment (pH < 2) explained the bacteria's ability to survive in the stomach environment and to infect people if a low dose is ingested.

(3) Dose-response

The infectious dose has been reported to be low for example in one outbreak traced to salami, the average infectious dose was estimated at fewer than 50 organisms (Tilden *et al.*, 1996) and Doyle *et al.*, 1997 suggested that it might be less than 10 . Other studies (USDA, 1993 and Willshaw *et al.*, 1994) have indicated that less than 2 bacteria per 25 gram foodstuff were sufficient to cause infection. Armstrong *et al.*, (1996) suggested that in the large multistate VTEC outbreak associated with hamburger patties the total number of bacteria in each patty prior to heat treatment were less than 700. One consequence is infection can occur without bacterial growth in contaminated food (Anonymous, 1999). These findings refer to VTEC O157:H7 outbreaks, while the infectious doses for other VTEC serotypes are not well described.

C. Exposure assessment

(1) Microbial ecology

*Escherichia coli* occurs in all species causing diarrhoea, generalised infections and mastitis (Anonymous, Scientific Veterinary Committee, 1997). The VTEC bacteria and in particular VTEC O157 appears to have ruminants as their reservoirs (Chapman *et al.*, 1997 and Wray *et al.*, 1993), while VTEC has also been isolated from pigs, dogs, cats (Mainil, 1998) horses, sea-gulls and geese (Anonymous, Scientific Veterinary Committee, 1997). The human pathogenic VTEC does not seem to be generally associated with disease in animals, while for example the VTEC serotypes O138, O139 and O141 are associated with porcine oedema and diarrhoea (Cannon *et al*, 1989), but not usually associated with disease in humans. However, since there is no clear definition based on virulence factors of the bacteria apart from the fact that verotoxin production seems to be a necessary but not a sufficient condition for human disease. There is no clear understanding of the microbial ecology of the VTEC bacteria as such. Nevertheless, for the groups referred to as VTEC O157:H7 or VTEC O157, more published data are available, and the comments hereafter pertain to these.These bacteria appear to grow in the temperature interval from 7 to 44.5 °C with an optimum of 37°C (Meng and Doyle, 1998) differing from the conventional *E. coli* where the optimal growth temperature is 40-42 °C.

Heat treatment of 70 °C for at least 2 minutes or equivalent will kill the bacteria, the core temperature must be 70°C for at least 8 seconds. On the other hand freezing and low temperatures do not kill the bacteria.

The bacteria can survive in acid environments where survival up to 2 months at pH=4.5 (Anonymous, Food Safety Authority of Ireland, 1999) and 31 days at pH=3.6 (Kauppi *et al.*, 1996) has been reported. Foods that has been preserved by acidification *e.g.*, salami, fermented meat sausages, apple cider, must therefore be considered as putative vehicles of these bacteria. Moreover, the acid tolerance increases the probability of the VTEC bacteria passing through the stomach barrier if ingested (Danielsson-Tham, 1998).

Another feature of VTEC O157 bacteria is the ability to survive for prolonged periods in the environment (Hancock *et al.*, 1998), thus differing from the traditional view of *E. coli* as an indicator of recent faecal exposure.For example, Bolton *et al.*, 1999 found that VTEC O157 was able to survive for up to 38 weeks straw, breeze blocks, wood surfaces and in water if using bacterial isolates, but shorter periods if the samples containing the bacteria were faeces. Randall *et al.*, 1999 reported that VTECO157:H7 could survive for several months on contaminated grasslands and manure.

(2) Prevalence in animals and food

Prevalence of VTEC O157 has varied in different studies Chapman *et al.*, 1997 found that a bovine, ovine and porcine prevalence in animals of 2.8%, 6.1% and 4%, respectively. No VTEC O157 was found from poultry in that study. In a study of dairy herds it appeared that only 1% of the samples (113/10832) were positive while the 9 out 15 studied herds had one or more positive isolate Hancock *et al.*, 1997. This concurs with findings in Sweden (Vågsholm, 1999) were a individual prevalence of 1-2% in calves and heifers were found when sampled on the farm. However, the the herd prevalence of VTEC O157 was found to be 10%, based on a sample of 249 herds. In a Canadian study Donkersgoed *et al.*, 1999 found that the prevalence of VTEC was around 43% while the prevalence of VTEC O157:H7 was 7.5% in faecal samples taken at slaughter. The authors noted that the prevalence of VTEC was higher in cull cows, while the prevalence of VTEC O157:H7 was highest among calves. The Community report on trends and sources of zoonotic agents in animals, feedstuffs, food and man in the European Union in 1998 (2000) seems to indicate following rule of thumb with regard to prevalences of VTEC O157 in cattle herds 10% or more, in individual bovine animals 1% or more while in beef or minced meat the prevalence is 0-1%.

D. Risk characterisation

(1) Incidence in humans

The annual VTEC incidence has varied between countries within EU during 1997 (Anonymous 1999), in some like Scotland the incidence was close to 100 per million, while the Community average was 7 per million inhabitants. It should be noted that the food borne outbreaks can be large and catch the attention of the media. The sporadic cases would receive less media attention and might also have another epidemiology. In the USA (Griffin and Mead, 1998) certain regions also appear to have a high incidence 80 per million similar to the Scottish situation. In Argentina which has a long history of HUS (Gianantonio, *et al.*, 1964), a high incidence is reported and many HUS patients appear to have VTEC O157 infection (Lopez *et al.*, 1989, Rivas *et al.*, 1998). The VTEC serotype O157:H7 has appeared to most commonly associated with human disease while other

serotypes are also reported associated with human disease.The dominance of the VTEC O157 in the northern and central European countries is contrasted by a reporting of other VTEC serotypes associated with disease in the Mediterranean countries. One interpretation is that a low level of human disease is associated with a multitude of serotypes, while the introduction of one serotype into the human/animal reservoirs and food chain with some additional but unknown virulence factors associated with the serotype O157:H7, will increase the human incidence significantly.

(2) Risk factors

The risk factors can be divided into those factors that increase the risk of bacterial and those factors that will increase the probability of disease if exposed such as host factors (age, health). Of the later age seems to an important factor since children less than 5 years of age and older people are seems to develop more severe clinical manifestations (Reilly, 1998). A possible hypothesis is that the infectious dose is smaller for these groups than for normal healthy adults due to a smaller secretion of gastric acid. Ruminants appear to be the reservoir of VTEC O157 bacteria (Mainil, 1999, Wray *et al.*, 1993, Griffin and Mead, 1998) while the role of other animal species such as pigs appears to be limited, but not refuted. Hence, the risk factors for human exposure is linked to either direct or indirect exposure and ingestion of faecal contents from ruminants or humans, this exposure could be minuscule given the infectious dose being as low as 50 bacteria (Tilden *et al.*, 1996). This exposure could be foodborne through undercooked meats such as hamburgers (Slutsker, 1998), untreated milk (Chapman *et al.*, 1993) and contaminated salad and fruits (Griffin and Mead, 1998). This exposure could result from cross contamination at the primary production stage by faecal contents from wild or domestic animals or cross contamination from raw meat products (Pennington, 1996; Griffin and Mead, 1998; Reilly, 1998). Several Japanese outbreaks were associated with radish sprouts (Nat. Institute of Health and Infectious Disease, 1997). Hence it appears that sprouts might be a risk vegetable since the bacteria might multiply during sprouting (CDC, 1997).

(3) Risk quantification

The risk would follow from the annual zoonosis reporting (Anonymous , 1999) which indicates the global risk experienced and reported during a particular year. Within the EU a total of 1912 cases of VTEC infections and 316 HUS cases were reported giving a incidence of approximately 7 and 1 per million, respectively. The interpretation of these numbers should be done with caution in particular if used for national or regional comparisons since the reporting systems vary between Member States. Nevertheless, the numbers of HUS should be less biased than the numbers of VTEC infections. However, in order to do risk quantification one needs to know the attributable risks for each risk factor and the exposures for each of risk factors to estimate the population attributable risks (Thrusfield, 1995). It is possible to quantify the importance of certain risk factors in outbreak situations, for example Slutsker *et al.*, 1998 reports that undercooked hamburgers were associated with VTEC O157:H7 diarrhoea (OR =4.5, 95% confidence interval 1.6-12.2). However to quantify the risk one needs to know rather the annual exposure rate of undercooked hamburgers per person to quantify the risk experience in the population. To interpret directly from US data and risk assessments to the EU context is problematic. Hence, the second best approach would be to ask each member state to specify the most important risk factors observed during the last years be it from case-control studies or outbreak investigations. From this a semiquantiative risk assessment could be deduced indicating which risk factors contribute most to the reported incidence,

and where *e.g.*, the cooking of hamburgers should produce the biggest reduction in number of cases.

(4) Risks in the future

The risks for human health seems mainly to be connected by lapses in normal hygiene procedures and forgetting lessons of biosecurity learned in the last century. Infections with human pathogenic VTEC has emerged during the last 20 years as a serious disease problem in most countries. From an epidemiological point of view the absence of specific treatments or vaccine at the reservoirs, the low infectious dose involved, the lacking knowledge of transmission routes, and the uncertainty of a laboratory diagnosis, represents the greatest challenges. It is not possible by any microbiological testing procedure at one point of the food chain or the production process to declare a food free of enterohaemorrhagic *E. coli*. Furthermore the confusion caused by different terms used interchangeably of VTEC, EHEC, O157:H7, STEC, STXEC, O157 and VTEC O157 in the scientific literature adds to the confusion with regard to what to look for and control. A common terminology, case definition, diagnostic and reporting procedure would aid in clarifying the epidemiological picture within the Member States. While the VTEC O157 seems to dominate today, other serotypes has been diagnosed in outbreaks such as O26, O103, 0111, O145 (Boudailliez *et al.*, 1997, Blanco *et al.*, 1996, Tossi *et al.*, 1994, Goldwater and Bettelheim, 1998, Meng and Doyle, 1997). The epidemiology of these serotypes is less well known and could represent a reservoir of future food borne pathogens.

While great outbreaks are food or water borne a large number possibly, the majority of cases are often infected through contact with infected animals or human carriers directly or indirectly. This might lead to bias of risk management efforts to the food borne route, while underestimating the public health importance of the non-food borne routes of transmission and preventive efforts there.

## II. RECOMMENDATIONS FOR RISK MANAGEMENT OPTIONS

It might matter more to reduce a common risk factor a little and than to remove an uncommon one completely. Moreover, perhaps priority should be given to measures preventing large outbreaks *i.e.* to avoid contamination of food or water that be a vehicle for the infection to a large population. However, one has always to strike a balance between the wish to protect the public health versus the liberty of the public to eat and produce, hence the issue of proportional measures. The question of whether the preventive measures are being proportionate with the reduction of the public health threat is as always at the end of the day a political one.Risk managers should see the following as suggested menu of options for consideration on a case by case basis.

An useful approach is to divide the transmission routes into 4 categories (a) transmission from person to person ; (b) direct contact with animals (c) food and water borne transmission; and (d) transmission through the environment. Another conceptual approach is to the look at possible risk management interventions at feed, farm, slaughter, food processing, retail, and at home, this approach might bias us to look at ways to control food borne transmission only. It appears that a case by case approach is the most salient with regard to human pathogenic VTEC, one should control the factors that appear to be important for the disease transmission.

The following points are suggested for consideration through the feed/food chain:

A. Farm level

(1) Feed

Garber *et al.*, 1995 found that some interesting but not fully significant (0.05<P<0.15) risk factors appeared to be the sharing of feeding utensils (OR=2.8) and oats (OR=2.9) and whole corn (OR=2.5) fed as calf starter rations. Clover pasture and clover as first forage appeared to be associated with reduced risk of shedding the bacteria. In a study of heifers Herriott *et al.*, 1998 found that the prevalence of heifers with VTEC O157 was significantly higher if feed corn silage.A possible explanation could be that corn silage could be promoting growth of the VTEC O157 bacteria, when removed from the silo and mixed with other feed ingredients and stored for a couple of days. The addition of ionophores in the feed also seemed to be associated with a higher prevalence in heifers. This is consistent with earlier findings that ionophores tend to favour gram negative bacteria (Schelling, 1984). Cray *et al.*, 1998 investigated the dietary stress of withholding feed to calves, with regard to *Escherichia coli* shedding. It was found that calves, for which the feed was withheld 48 hours before inoculation of the VTEC O157 , were more susceptible compared with those that had feed withheld after inoculation. Gyles, 1999 suggested that any management changes that promoted instability of the intestinal flora and reduced the production of volatile fatty acids in the rumen, would increase the shedding of VTEC O157:H7. Zhao *et al.*, 1998 proposed to use probiotic bacteria to reduce the shedding of VTEC O157:H7. Most of the probiotic bacteria (17 out of 18) were not verotoxin producing *E. coli*. In Sweden, one observation is that calves that are let out on pasture seems to shed less VTEC O157 for an extended period of 2-3 months (Jonsson 2000). The pasture available for each calf this seemed to be the crucial parameter.

(2) Calf management at weaning

Garber *et al.*, 1995 found in a case control study that weaning was a critical phase for calves shedding *Escherichia coli* O157:H7. The prevalence of calves shedding the bacteria increased from 1.4% to 4.8% or by a factor of 3. The grouping of calves before weaning appeared to be a significant risk factor (OR=9, P=0.005). Herds in which calves housed in groups were also at higher risk OR=7.8 (p=0.01) and 4.2 (p=0.07), in the winter and summer season, respectively. Hence, it appears that farmers should group their calves after weaning not before, and if possible to house them individually as long as possible.

(3) Direct contacts

The separation of people pick-nicking and cattle on pastures should be encouraged to limit possible environmental exposure. For example, the use of cattle pastures for pop concerts, fairs and markets has been incriminated as the source of VTEC outbreaks, should not be recommended. Moreover, in regions where the incidence of VTEC infections in humans has been considerable, one would suggest that cattle should not pasture together with people on the beaches. It might be noted that in Sweden, children less than 5 years of age are not recommended to visit farms during the summer seasons of 1998 and 1999.

(4) Manure handling

No association was found of manure handling practices and the risk of the calves shedding the bacteria in the study by Garber *et al.*, 1995. This was surprising since earlier studies by Kudva, *et al.*, 1998 and Wang *et al.*, 1996 found that the VTEC O157:H7 bacteria could survive for several months Randall *et al.*, 1999 found that VTEC O157 was able to survive for up to 38 weeks straw, breeze blocks, wood surfaces and in water if using bacterial isolates. However, the survival periods were shorter if the environments were contaminated with faeces containing the VTEC O157. Wray *et al.*, 1999 suggested that one should be careful if extrapolating from growth models to the survival of the same bacteria in faeces. Predictive models based on the survival of VTEC O157 in faeces would be helpful in devising good manure handling practices.

Thus, to avoid outbreaks it seems that the protection of the wholesomeness of food to be eaten raw (*e.g.*, radish sprouts) and drinking water is essential. Hence, one conclusion would be that manure should be disposed of in such a way that neither drinking water nor growing vegetables or berries foreseen eaten without heat treatment could be contaminated.

Juice produced from fallen fruits or berries and possibly contaminated by manure (picked up from the ground with *e.g.* ruminants pasturing in the orchard), should never be sold unless pasteurised.

B. Transport, slaughter, processing and retail

The report of the Scientific Veterinary Committee on VTEC (1997) included several recommendations such as:

- clean animals at slaughter
- better transport conditions of slaughter animals
- review of dressing an d evisceration process
- hygienic production of milk and milk products
- hygiene and cold chain maintained throughout the food chain
- decontamination of carcases
- education of food safety for those working in the food safety chain
- special attention to risk groups with regard food handling
- more research

Moreover, the drinking of unpasteurised milk appears as a risk factor in many outbreak investigations. Traditional hygienic recommendations cannot guarantee freedom from VTEC in unpasteurised milk. Those drinking unpasteurised milk or milk products should therefore be taking an informed risk, and a compulsory labelling procedure should be considered. Children and elderly being the most susceptible groups should avoid drinking unpasteurised milk.

The proper heat treatment of meat preparations *e.g.* minced meat preparations such as hamburgers, or steaks such as roast beefs would eliminate this route of transmission for human pathogenic VTEC, heating the core of the beef or burger to more than 70°C for 2 minutes (Irish Food Agency, 1999)

C. Home and vulnerable groups

For person to person transmission the public health authorities (Mead and Griffin, 1998) could consider several actions:

- to advice patients on the importance of hand-washing and avoiding cross contamination when preparing food;
- to advice on that children should stay at home from kindergarten when having bloody diarrhoea (and ensuring that parents receive compensation to stay at home with the children) and only going back after 2 negative faecal samples for VTEC;
- to advice people working or visiting farms to wear appropriate protective clothing,
- to ensure that a proper investigation takes place for each outbreak to trace the sources and ways of transmission, and
- to ensure that reliable surveillance systems covering the whole population is in place, since it is only then one can rapidly respond to new patterns of outbreaks.

## III. MONITORING AND RESEARCH PRIORITIES

The priorities should be the following for the monitoring:

- There is an urgent need for common terminology for the disease and bacteria (VTEC, EHEC STXEC, STEC or O157), case definitions, microbiological procedures and reporting requirements.
- The prevalence in food producing animals and food should be examined annually.
- The aggregate statistics produced by the monitoring and surveillance should be analysed at the Community level, since the number of cases might be to small for meaningful analyses at the national or regional level.
- The notification and reporting systems for human disease needs to be improved and equivalent throughout the EU
- The diagnostic procedures in humans, food and live animals should be harmonised within the Community, ensuring one is talking about the same bacteria.
- On the basis of a working reporting system it should be possible to assess any changes in the human incidence of VTEC within the EU with regard to time, region and individual factors (such as age). Moreover, the results of interventions could then be assessed throughout the food chain.

The priorities for research would include to:

- identify the clinical importance and the sources of non-O157 VTEC
- improve of the diagnostic methods for all human pathogenic VTEC serotypes,
- identify of host specific factors in the VTEC pathogenesis, why does a few people get sick,
- identify of human disease specific virulence factors of VTEC,
- identify animal reservoirs of the VTEC bacteria causing human disease,
- quantify the importance of different transmission routes *i.e.* their attributable risks,

- assess the impact of calf management and feeding of shedding of the human pathogenic VTEC,
- assess the impact of transport and slaughter practices on the shedding of the human pathogenic VTEC, and to
- develop predictivemodels for all human pathogenic VTEC in food and environment

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### *13.1.4. Annex I.d : Cryptosporidium*

## I. RISK ASSESSMENT

A. Hazard identification

*Cryptosporidium* is a waterborne coccidian parasite known to infect humans (Fayer, 1994). Having been firstly identified in 1908 in mice, *Cryptosporidium* was subsequently noted as a causative agent of diarrhoeal illness in turkeys, lambs and calves. It was not until 1976, however, that *Cryptosporidium* was diagnosed in humans and since then *C. parvum* has been established as a significant agent causing diarrhoea in humans worldwide.However, due to its association with HIV-infected individuals (Dubey *et al.*, 1990; Fayer, 1997), awareness of this parasite really came to the fore in the late 1980s. Our concept of cryptosporidiosis has subsequently changed from that of a rare, largely asymptomatic infection, to an important cause of enterocolitis and diarrhoeal illness in several species, including humans.

The incidence of cryptosporidiosis in the population has been documented within ranges of 0.6 to 20% with significantly higher incidences reported in parts of Asia, Africa and South America, compared to more developed countries (Rose and Slifko, 1999). Seroprevalence studies however have revealed that 30% of the worldwide population has been exposed to this parasite (Rose, 1997).

In England and Wales the numbers of laboratory reported cases of cryptosporidiosis was 3560 in 1986 (population 49.8 million), reaching a peak of 7768 cases in 1989 (population 49.8 million), and the numbers for 1998 were 3745 (population 52.2 million).

(1) Characteristics of the organism

*C. parvum* is an obligate intracellular–extracytoplasmic coccidian protozoan that carries out its parasitic life cycle in one host. Following the ingestion of thick-walled oocysts (cyst-forming sporozoites), they excyst in the small intestine and free sporozoites then penetrate the microvilli of the host enterocytes, where the mature zygotes are developed. Oocysts are developed from these fertilised zygotes and are subsequently released in the faeces. These oocysts are resistant to environmental factors and the infection is spread to other hosts when they are ingested (Jay, 1992).

*Cryptosporidium* sp. infect many herd animals (cows, goats, sheep, deer and elk). The initial assumption that each *Cryptosporidium* species was host-specific has now been recognised to be incorrect and it is currently believed that the same strain of *Cryptosporidium* can infect both humans and young calves. However, strains infecting avian and murine hosts are not thought to be capable of infecting humans (Shield *et al.*, 1990).

The oocysts of *C. parvum* are spherical to ovoid and average 4.5 to 5.0 μm in diameter. The oocysts have been reported to remain viable in the environment for several months in cold, moist conditions (Current, 1998). The oocysts are resistant to most chemical disinfectants, especially ozone and chlorine-containing compounds used in the treatment of drinking water (Campbell *et al.*, 1982).

In general, *Cryptosporidium* is of particular concern for four reasons: (i) the oocyst is extremely resistant to disinfection and cannot be killed with routine water-disinfection

procedures; (ii) the disease is not effectively treatable with antibiotics; (iii) the risk of mortality ranges between 50 and 60% in the immunocompromised population; and (iv) animal and human faecal wastes are associated with transmission of the disease to humans (Rose and Slifko, 1999).

(2) Reservoir

Human cryptosporidiosis may be acquired by a variety of routes of transmission including zoonotic, person to person, water, nosocomial, or food. Transmission from host to host is always via the oocyst stage of the life-cycle through the faecal-oral route. Oocysts of *C. parvum*, unlike many other coccidia, are sporulated when shed and are therefore thought to be infectious immediately (Fayer, 1994).

With regard to reservoir hosts, many mammals have been found naturally infected with *C. parvum*. These include wild mice, the house mouse, and rats (Perryman, 1990), domestic cats, dogs, ferrets, raccoons, rabbits, and monkeys (Riggs, 1990), pigs (Kim, 1990), cattle, sheep, goats, farmed red deer, wild ruminants including fallow deer, roe deer, sika deer, mule deer, Eld's deer, axis deer, and barasingha deer, water buffalo, Persian gazelles, blackbuck, sable antelope, scimitar horned oryx, fringe-eared oryx, addaxes, impalas, springbok, nilgai, gazelles, eland, and mouflon (Angus, 1990). Cattle, however, have been proposed as the most likely source of zoonotic transmission of this parasite through the deposition of infected faeces (Shield, 1990).

Water is a well documented reservoir of this parasite and waterborne transmission is linked to many outbreaks of cryptosporidiosis. Oocysts have been found in water intended for swimming and drinking as well as surface water from reservoirs, lakes, ponds, streams and rivers. Water surveys have shown the detection of oocysts in small numbers in all water sources, but are more prevalent in surface than ground waters. Human infectious dose studies and models demonstrate that one oocyst carries some probability of causing an infection (Haas *et al.*, 1996). Most faeces that carry oocysts end up in the environment and can be spread to foods by irrigation or by direct contact. Routine wastewater treatment eliminates only a small fraction of oocysts (Lisle and Rose, 1995).

An outbreak of cryptosporidiosis which occurred in Georgia, USA in 1987 was the largest waterborne outbreak ever reported to the US waterborne outbreak surveillance system. An estimated 13,000 people became ill after consuming water from a filtered, chlorinated public water supply that complied with state and U.S. federal standards (Levine and Craun, 1990).

B. Hazard characterisation

(1) Disease

*Cryptosporidium* sp. cause infection in humans and other vertebrates, including mammals, birds, reptiles, and fish. More than 20 species of *Cryptosporidium* have been reported, of which six are considered valid species on the basis of oocyst morphologic features and site of infection (O'Donoghue, 1995; Dubey, 1990 ). *C. parvum* and *C. muris* infect mammals, *C. baileyi* and *C. meleagridis*, infect birds and *C. serpentis* and *C. nasorum* infect reptiles and fish. *C. parvum* is the major species responsible for clinical disease in humans and domestic animals (WHO, 1996).

The disease called cryptosporidiosis has been described as "*cholera-like*" and its symptoms include large volumes of fluid loss, fever and abdominal pain (Rose, 1997).

It is not usually possible to define accurately the incubation period, but in most cases symptoms appear within 3 days to a week, or occasionally longer. In healthy individuals, symptoms involve diarrhoea varying in severity from mild to severe and lasting from several days to more than a month. Within this group it is a self-limiting illness and antimicrobial therapy is not usually necessary (Juranek, 1995 and Varnam and Evans, 1996). Supportive therapy includes fluid replacement and, in chronic cases, parenteral nutrition. No truly effective remedy is available for cryptosporidiosis in the immunocompromised, although a number of compounds show promise including the macrolide antibiotic, spiramycin. Immunomodulation therapy may also be of value, with transfer factor, recombinant interleukin-2 and hyperimmune bovine colostrum, all being used successfully in small-scale trials (Varnam and Evans, 1996). To date the species or strain infecting the respiratory system is not distinguished from the gastrointestinal form.

In immunocompromised people, especially those suffering from AIDS, cryptosporidiosis usually results in a prolonged and life-threatening illness which symptomatically resembles cholera. In many cases, diarrhoea becomes very severe and fluid loss excessive. Passage of 3 to 6 litres per day of watery faeces is common, and as much as 17 litres has been reported. Extraintestinal symptoms may occur, and both respiratory and biliary cryptosporidiosis have been reported (Varnam and Evans, 1996).

Symptom severity may wax and wane, which could be correlated to the intensity of oocysts shedding (Fayer and Ungar, 1986). Oocyst excretion is most intense during the first week, decreasing thereafter and generally ceasing when diarrhoea ceases (Ryan *et al.*, 1994).

Treatment is generally unnecessary for immunocompetent patients, although oral rehydration therapy may be practised in severe cases. A major problem for immunocompromised victims is that no effective anti-cryptosporidial compounds have been identified. Oral paromycin treatment may reduce the intensity of diarrhoea in some patients exist, but need to be confirmed (Murray *et al.*, 1994; Ryan *et al.*, 1994).

(2) Virulence/Pathogenicity

Although cryptosporidiosis is often considered to be a zoonotic disease, person-to-person transmission is now commonly recognised. However, many human infections with *Cryptosporidium* are derived from farm animals, particularly cattle, for which wild rodents may act as a reservoir (Varnam and Evans, 1996).

Infection follows the ingestion of a small number of oocysts (cysts forming sporozoites), typically 4-6μm in size. These banana shaped motile sporozoites are released in the small intestine, where they adhere to enterocytes of the villi and develop into trophozoites beneath the cell membrane. Fertilisation of macrogametes may follow, which results in the production of oocysts. Two types of oocyst can be formed:

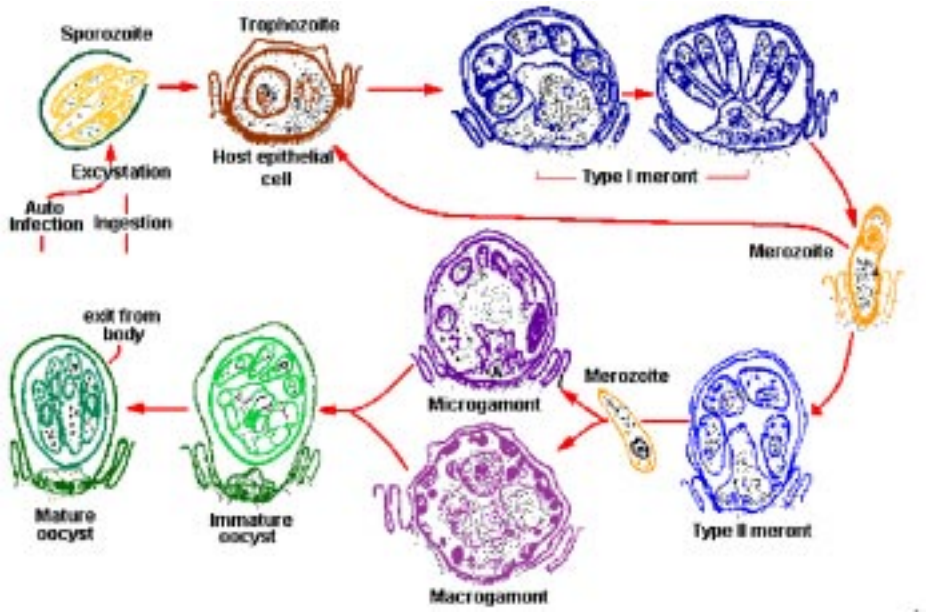
(a) *thin-walled* oocysts which release sporozoites into the host's intestine, causing re-infection ('auto-infection') of the host;

(b) acid-fast, *thick-walled* oocysts which constitute approximately 80% of the total, and are released in the faeces.

The precise mechanism of pathogenesis is unknown, although the diarrhoea produced is of a secretory nature (with possible involvement of an enterotoxin), with damage to the villi and some resulting malabsorption. However invasion beyond the host cell membrane does not usually occur (Eley, 1996)

Sporozoites excyst from an oocyst and enter the microvillus of an epithelial cell, where they differentiate into trophozoites. Trophozoites undergo nuclear proliferation to form type I meronts. A type I merozoite leaves the meront to form either a type I or type II meront. A type II merozoite leaves the meront to form microgametes or a macrogamont. The microgamete fertilizes the macrogamont, which then develops into an oocyst. Oocysts sporulate *in situ* and either release sporozoites for autoinfection or pass from the body in the faeces. (from Fayer & Ungar, 1986)

**Fig. 1 Diagrammatic representation of life cycle of *Cryptosporidium***




(3) Dose-response

Although the minimum infectious dose for humans is not clear, in animal trials, two (of two) primates became infected after ingestion of just 10 oocysts (Jay, 1997)

Dose response data is extremely ambiguous also. For a single isolate of *C. parvum* in healthy human volunteers, the 50% infectious dose ($IC_{50}$) was estimated to be 132 oocysts; one individual was infected by 30 oocysts (Dupont *et al.*, 1995). Other reports based on mathematical modelling algorithms indicate that some persons could become infected with a dose as low as one oocyst (Haas and Rose, 1994).

This data suggests that the infective dose for humans is quite low. There is currently no comparative data linking mouse model and human dose responses.

Although small numbers of oocysts can be recovered from treated drinking water, the significance of this is unknown. Current methods do not allow determination of whether oocysts are viable or infectious, and recovery methods are acknowledged to be poor and inefficient. Hence it is not known if the number of oocysts present in drinking water

constitutes a dose sufficient to cause human illness. While low level transmission may occur as a result of low oocyst numbers, there is no data to document this (Juranek, 1995). The health risk, especially for immunocompromised individuals, associated with consumption of public drinking water contaminated with small numbers of *C. parvum* oocysts remains unknown (Juranek, 1995).

C. Exposure assessment

(1) Microbial ecology

- Temperature

Oocysts can survive at refrigeration temperatures, but are killed by freezing (below –20°C) (Casemore, 1989).

- Heat resistance

Oocysts are not heat-resistant. They are sensitive to holding for 20 minutes or more at temperatures above 45°C and are readily destroyed by pasteurisation or heat treatments equivalent to 5-10 minutes at 65-85°C (Fayer, 1994).

In the event of a boil notice, water need only be raised to boiling point and allowed to cool - a prolonged holding time is not required.

The organism is sensitive to desiccation, requiring moisture for survival. Air drying for a few hours at room temperatures should ensure complete destruction of any oocytes present. Oocysts do not survive freeze drying (Casemore, 1989).

- pH, water activity and atmosphere

Little is known about the effects of pH, $A_w$ and gas atmosphere on the survival of *Cryptosporidium* oocysts. However, extremes in pH appear to have a significant effect on survival (Lawley, 1998).

(2) Prevalence in food

Oocysts can survive for up to a year in water, and contaminated water is well established as a vehicle for *Cryptosporidium*. Current water treatment systems cannot guarantee the complete absence of oocysts in mains water at all times (Casemore, 1989; Fayer, 1994).

In Britain, outbreaks of disease considered to be associated with inadequately filtered or contaminated water have occurred in several parts of the country, some outbreaks involving hundreds of confirmed cases. A recent very large outbreak in Milwaukee, in the USA, affected an estimated 403,000 people, when the city water supply became contaminated with this parasite.

Largely because *Cryptosporidium* cannot be cultured in the laboratory, food has only rarely been linked directly to incidents of cryptosporidiosis. However raw sausages, offal and raw milk have been suggested as possible vehicles (Casemore, 1989) . Suspected outbreaks have been reported from travellers who visited Mexico, the United Kingdom and Australia. Suspect foods included salad, raw milk, sausage and tripe (Smith, 1993). The first foodborne outbreak of cryptosporidiosis in the United States was related to apple cider and occurred in 1993. Three other outbreaks have since been reported, one

was associated with chicken salad, one with green onions and another with apple cider (Rose and Slifko, 1999).

Table 1. Foodborne outbreaks of cryptosporidiosis in the United States

| DATE | LOCALE | NUMBER OF CASE/TOTAL EXPOSED | FOOD | VENUE AND CONTRIBUTING FACTORS |
|---|---|---|---|---|
| October 1993 | Maine | 154/284 | Apple cider | Local fair: apples contaminated: dropped apples from farm with livestock were used in cider preparation |
| September 1995 | Minnesota | 15/26 | Chicken salad | Social event: hostess ran a home-day care facility. No specimens were submitted for examination from hostess or children |
| October 1996 | New York | 31/? | Apple cider | Community outbreak associated with apple cider from a mill that purchased only picked apples, as opposed to dropped apples. Dairy livestock were nearby but not in the orchard. Well water tested positive for coliforms and *E. coli*. Apples were washed and brushed prior to pressing. |
| December 1997 | Washington | 54/62 | Possibly green onions | Dinner banquet: no single food item ( out of 18) had a strong association with the illness. Two of the workers at the banqu*et al*so tested positive and had consumed the food. Green onions had not been washed |

[Rose and Slifko (1999)]

Fresh produce that has been washed with contaminated water is also a possible vehicle of the organism. This is probably an important mode of transmission in travellers' diarrhoea (Varnam and Evans, 1996; Casemore, 1989).

Air may also occasionally act as a vehicle for transmission of *Cryptosporidium*. The inhalation of oocysts held in aerosols in animal processing plants and during certain farming practices (such as muck-spreading) may contribute to the occurrence of cryptosporidiosis (Lawley, 1998).

(3) Consumption data

Although the minimum infectious dose for humans is not known, two (of two) primates became infected after ingestion of 10 oocysts (Barer and Wright, 1990).

D. Risk Characterisation

(1) Incidence in human medicine

Occurrence of cryptosporidiosis is worldwide. In developed areas such as the USA and Europe, prevalence of infection was found in <1% to 4.5% of individuals surveyed by stool examination. In developing regions the prevalence is significantly higher, ranging from 3-20% (Benenson, 1995).

*Age distribution*

All age groups may become infected with *Cryptosporidium*. Age distribution of cryptosporidiosis tends to vary geographically and in some Scandinavian countries such as Sweden, the disease is mainly among adults (Atterholm *et al.*, 1987). Elsewhere there appears to be a primary peak of infection among children aged less than 5 years and a

secondary peak in adults aged 20-40 years, possibly reflecting family exposure to children or occupational exposure (Varnam and Evans, 1996). In the UK, at least, cryptosporidiosis is rare in people over the age of 40 years (Casemore, 1989). Although there is agreement that the incidence is high among children aged 1 to 5 years, there are discrepancies concerning the disease in children aged less than 1 year. In the UK some surveys have shown cryptosporidiosis to be less common in children aged less than 1 year, and rare in those under 6 months of age (Palmer and Biffin, 1987; Thomson *et al.*, 1987). This pattern is similar to that reported in the USA, rural Costa Rica, Liberia, Rwanda, Guinea-Bisseau and Haiti (Casemore, 1989). In contrast, other surveys in the UK (Baxby and Hart, 1986), Ireland (Corbett-Feeney, 1987), and other developing countries including Guatemala and India (Mathan *et al.*, Cruz *et al.*, 1988) have shown *Cryptosporidium* infections to be common in children aged less than 1 year. These differences may be related to differences acquired immunity, maternal immunity, exposure and weaning practices (Varnam and Evans, 1996).

*Area Distribution*

The incidence of cryptosporidiosis varies from region to region. In the developed world, surveys have produced contrasting results, with an incidence of less than 1% shown in some areas, contrasting with others where *Cryptosporidium* can be third or fourth most commonly identified pathogen and, at certain times, the most common. In developing countries the incidence of *Cryptosporidium* infection is higher, and there is evidence of hyperepidemicity (Varnam and Evans, 1996). The organism is an important cause of traveller's diarrhoea (Jokipii *et al.*, 1985, Sterling *et al.*, 1986). Unlike most causes of travellers diarrhoea, travel-associated *Cryptosporidium* infections may be acquired by city dwelling West Europeans on visits to country areas in their own, or neighbouring, countries (Palmer and Biffin, 1987).

*Seasonal Variation in the number of cases*

Various seasonal or temporal peaks have been observed in the occurrence of *Cryptosporidium.* For example, in North America the incidence of infection is greatest in spring or late summer, while in Central America and India peak infection occurs during the rainy season (Varnam and Evans, 1996). Studies in the UK and Ireland (Casemore *et al.*, 1986; Corbett-Feeney, 1987) have shown a peak incidence in the spring, and on some occasions, a second peak in late autumn or early winter. These peaks may indirectly reflect rainfall and farming practices. Experience in the UK over a five year period indicates outbreaks, or temporal clusters of apparently sporadic cases, in different parts of the country. These tend to occur at about the same time, but in different localities each year (Casemore, 1987). The underlying reasons for this pattern are not known (Varnam and Evans, 1996).

(2) Risk Factors

The risk factors that have been associated with outbreaks of cryptosporidiosis are consumption of inadequately filtered and contaminated water, certain foods *e.g.* raw sausages, offal and raw milk.

Travel abroad seems to be a common underlying risk associated with becoming infected with *Cryptosporidium* and may be as a result of exposure to contaminated water.

(3) Risk quantification

Quantitative risk assessment is a tool to estimate the risk of illness caused by a given risk factor. Risk assessment methods have been used for risk management in water (Haas, 1996)

Little data is available to enable a comprehensive risk quantification. However the figures that have been gathered in the UK Public Health Laboratory Service (PHLS) indicate that the number of cases of cryptosporidiosis has remained relatively static over the last number of years (www.cdc.phls.co.uk/facts/crypt.htm).

To reduce the risk of cryptosporidiosis more work has to be done to elucidate the causes of the infections. The relative risk of acquiring cryptosporidiosis from drinking water versus contact with animals and person-to-person contacts should be given high priority for further studies.

(4) Risk in the future

More efforts have to be directed into reducing the prevalence of *Cryptosporidium* in water, food and production animals.

## II. RECOMMENDATIONS FOR RISK MANAGEMENT OPTIONS

*Cryptosporidium* has been associated with a number of water and foodborne outbreaks. Control and prevention of this food and water-borne disease can be summarised by our ability to prevent, remove or kill this protozoan contaminant (Rose and Slifko 1999).

The *Cryptosporidium* oocysts are immediately infectious upon excretion from the host (animal or man). Hence it has emerged as a public health risk, causing concern to the food industry, child care centres, recreational swimming pools and hospitals were person to person or environmental contamination can play a role in disease transmission (Barbee *et al.*, 1999).

### A. Farm level

*Cryptosporidium parvum* is found in most ruminants, both domestic and wild, with young animals presenting the gratest risk. The public health risk arises when water courses become contaminated with animal faeces. Therefore protection of water catchments from contamination by animal wastes should be a priority. Water from unprotected catchments is likely to be subject to contamination by *Cryptosporidium* and treatment including effective filtration will be required to remove these organisms to ensure a safe supply. The lower the quality of the source water, the greater the reliance on water treatment processes. (NHMRC, 1999).

Sanitary surveys of water catchments for potential contamination sources should be undertaken together with investigative and event based testing of source water for *Cryptosporidium* to assess risk factors for contamination, to provide a basis for catchment management to reduce these risks and to determine the level of water treatment required. It has been reported that increases in turbidity associated with rainfall events may signal increased numbers of *Cryptosporidium* (Atherholt *et al.*, 1998).

Groundwater from confined aquifers or from depth should be free from contamination by *Cryptosporidium*. However, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination (NHMRC, 1999).

A multiple barrier approach operating from the water catchment area to tap should be implemented to minimise the risk of contamination by *Cryptosporidium* (NHMRC, 1999).

At the dairy level, it is essential that hygienic practices in the milking parlour and prepasteurisation procedures ensure the bacterial load is kept to the minimum. HACCP should be applied pre and post pasteurisation to reduce the possibility of contamination or cross contamination with any pathogens.

B. Slaughter

Strategies to reduce and eliminate faecal contamination designed to target the more common foodborne pathogens such as *Salmonella* and *E coli* O157 will also deal with *Cryptosporidium*. It is important that only potable water comes into contact with carcasses.

C. Secondary Production, commercial caterers, transport and retail

There are a number of approaches for control and management

- Use of non-contaminated water
- Improvement of drinking water treatment
- Implementation of hygienic practices during harvesting and packaging
- Use of hygienic practices for food handlers
- Pasteurisation of juices
- Irradiation of produce

(Rose and Slifko, 1999)

An EU project funded under the 5$^{th}$ Framework (Project No: QLRT 1999.00775) is to commence in January 2000 and will develop methods to identify *Cryptosporidium* in food and where present, assess the subsequent risk to humans.

(1) Prevention

A multiple barrier approach operating from the water catchment area to tap should be implemented to minimise the risk of contamination by *Cryptosporidium* (NHMRC, 1999

(2) Removal and treatment

The design and operation of water treatment plants should be carefully examined where *Cryptosporidium* oocysts are suspected or known to be present in the raw water to ensure that required performance is achieved and maintained. Particular attention should be paid to ensuring optimum coagulation/flocculation, monitoring of turbidity from all filters, appropriate handling of backwash water, minimising turbidity increases during filter start ups and operation of filters to avoid sudden flow surges (Badenoch, 1995). The performance of filtration plants should be monitored continuously and treated water of a constant quality should be produced irrespective of the quality of raw water. Trained and skilled personnel should operate filtration plants. Failure of water treatment processes including failure to meet specified targets for turbidity (or particle counts) should be regarded as representing a risk of oocyst contamination of the drinking water supply.

The integrity of distribution systems should be maintained. The use of unroofed treated water storages within distribution systems should be avoided as these could allow the entry of contamination from birds and small animals, backflow prevention policies should be applied and faults and burst mains should be repaired in a manner that will prevent ingress of contamination (NHMRC, 1999) .

The physical removal of the oocysts and cysts can be achieved during drinking-water processes via sand filtration using coagulant aids (*i.e.* alum, ferric and polymers) (Rose and Slifko, 1999). An absolute 1 μm pore-size membrane filter or smaller has been recommended by the Centre for Disease Control and Prevention (1991) for removal of the protozoan cysts and oocysts.

*Cryptosporidium* oocysts are extremely resistant to disinfection and will not be killed by doses of chlorine that can be used in drinking water. Other disinfectants such as ozone are more effective but are unlikely to provide complete protection against contamination. (NHMRC, 1999) (Rose and Slifko,1999) (WHO, 1996) (Matukaitis, 1997)

Currently there is no data to support or quantify cyst or oocyst reduction caused by washing fruits and vegetables. (Rose and Slifko 1999)

D. Home consumers including vulnerable groups

The public need to be protected by ensuring that public drinking water is free of *Cryptosporidium*. A crisis management plan should be developed for dealing with a water contamination incident or an outbreak of human illness. It should include strategies to alert the public. Such eventualities should be planned for with alternative sources of supply available.Criteria for the issuing of a boil water notice to the public should be defined, as should criteria for lifting such a notice.

The immunocompromised are particularly at risk to what is normally a self-limiting illness to the average healthy person. Public health officials should consider a communication/education program

- to physicians treating the immunocompromised,
- for nursing homes, child care centres and the food industry
- develop a plan to evaluate cases of cryptosporidiosis in the community and
- contribute to the development of public policies that limit contamination of source waters, improve water treatment, and protect public health (Rose, 1997).

A lot or research and a number of guidelines have been produced to help protect children as well as the immunocompromised from *Cryptosporidium* in drinking water as well as in recreational pools (Carpenter *et al.,* 1999) (PHLS, 1998) (Bouchier, 1998). Recommendations include the drinking of bottled water only, washing hands before eating or preparing food and to avoid touching animal stools.

## III. MONITORING

The effectiveness of implemented risk management tools should be validated through monitoring and surveillance (WHO, 1997; Schlundt, 1999). However *Cryptosporidium* is not a notifiable disease in either humans or animals in most Member States. It is not

notifiable under the current Zoonosis Directive (92/117/EEC). Both the frequency and the level of the pathogen and the impact on the number of human cases of disease caused by the pathogen should be included. Programmes for monitoring the effect should be established at all relevant stages in the production of foods where a certain factor for the control of *Cryptosporidium* contamination has been implemented.

While there is no formal monitoring programme in place in Ireland, *Cryptosporidium* has been detected in river samples and marine mussels in County Sligo, Ireland (Chalmers, 1997).

**Comparable data and methods for analysis**

New methods are now available for determining the level of oocyst contamination. Greatest promise is being seen using molecular- and immunology-based methods. Commercially-available monoclonal antibody-based detection kits are available for detection of *Cryptosporidium* sp. and are used in conjunction with electron microscopy (examining fluorescence, size, shape, and presence of internal sporozoites) for detection from water (Rose and Slifko, 1999).

Because these parasites occur in relatively small numbers within the environment, oocyst detection relies upon concentration techniques. Membrane filtration, cartridge filtration and centrifugation all have applications to detect within surface, irrigation and drinking water. The main drawback to all these techniques, however, is the fact that other types of particles are concentrated in addition to the protozoa. Therefore, waters with algae, turbidity, suspended solids, and other materials can interfere with large volume collections (greater than 10 litres) and the detection process.

A more promising technique is immunomagnetic separation using antibodies tagged to a silicon-coated iron-oxide beads and a magnetic system to pull the target oocysts from suspension (Johnson *et al.*, 1995). This has applications for both microscopic detection and polymerase chain reaction. Several immunomagnetic separation kits are now available for *Cryptosporidium* (Dynal, Lake Success, NY; ImmuCell, Crypto-Scan, Portland, Maine).

For recovery from foods, methods used to date have been relatively simplistic and involve physically washing the oocysts from the surfaces of the fruits and vegetables with a detergent solution. Difficulties involving these methods are firstly, the inability to fully remove the oocysts from the food products and secondly that it is only practical to sample a small percentage of the crop or batch (Rose and Slifko, 1999). It may be more appropriate to sample irrigation waters or to develop a large batch-rinse technique and sample the rinse waters.

Molecular methods are being utilised to detect the nucleic acids within the oocysts, and these methods are extremely rapid, sensitive and species-specific. The polymerase chain reaction (PCR) and fluorescent *in situ* hybridisation methods have been successfully used to identify *Cryptosporidium* (Johnson *et al.*, 1995; Lindquist, 1997). These techniques are relatively new but offer a significant application for detection because the probe binds directly to the oocyst and hence, both microscopy and the specificity of the probe can be used for identification and detection (Johnson *et al.*, 1995).

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### *13.1.5. Annex I.e : Echinococcus*

## I. RISK ASSESSMENT

A. Hazard identification

Two species of parasitic organisms of the genus *Echinococcus* are known to occur in Europe, namely *Echinococcus multilocularis* and *E. granulosus*, causing two different chronic diseases, the alveolar and the cystic form of echinococcosis (Schantz *et al.* 1995). Contamination of humans occur by the ingestion of the parasite eggs, either after touching carnivores or after eating food contaminated by carnivore faeces. Despite the similarities shared by their infectious helminthic agents, both diseases behave very differently, both in terms of clinical presentation and prognosis and in terms of transmission cycles and epidemiology. Cystic echinococcosis (also named hydatid disease or hydatid cyst) behaves as a benign tumour, usually located in the liver and/or the lung, and is most often curable; alveolar echinococcosis behaves as a malignant tumour, initially located in the liver, then able to spread to any organ through metastases, and is lethal in the absence of appropriate therapeutic management (Craig *et al.* 1996; Bresson-Hadni *et al*, 2000). Both, however, for different reasons must be considered of Public Health significance in Europe, the former being more frequent and present in Southern Member States of the EU and border countries, the latter rarer and present in Northern Member States of the EU and border countries (for more detailed information on epidemiology of echinococcosis in the world, see Schantz *et al.* 1995; Lucius and Bilger 1995; Schantz *et al.* 1996, Eckert 1997).

(1) Characteristics of the organisms

*Echinococcus* sp are plathelminths, of the cestode genus. As many parasites, *Echinococcus* sp are characterised by a parasitic cycle which involves final hosts and intermediate hosts, each harbouring different stages of the parasite life (Ammann and Eckert, 1996)

*Final hosts*, carnivores for both species, host the *"adult" form* of the parasite, an intestinal worm, also called *taenia*. Numerous (from tens to thousands) adult worms (length averaging 4-6 mm for *E. granulosus*, and 2-5 mm for *E. multilocularis*) live in the small bowel of the carnivores (taeniosis), attached to the small bowel mucosa through hooks and suckers; after 25 to 40 days, their last gravid segment, each containing hundreds of microscopic "eggs" ("6-hooked oncospheres", or "hexacanth embryos", 30-40 μm in diameter), detaches from the non-fertile segments and these egg-containing segments are dispersed with the faeces of the carnivore.

*Intermediate hosts*, that belong to various species depending on the *Echinococcus* species, host the *"larval" form* of the parasite, also called *"metacestode"*. It constitutes a cyst filled with fluid, well separated from the surrounding host tissues, in cystic echinococcosis, and a tumour-like continuously growing polycystic mass, not clearly separated from host tissues, in alveolar echinococcosis. In both cases, parasitic cysts or vesicles become fertile by giving rise to the particular form which will be able to re-create the adult form in the final host; this form, that fills the cysts, is called "protoscolex". Each protoscolex, when eaten by a carnivore, will transform into an adult worm in its intestine.

(2) Reservoir; "cycles" of *E. granulosus* and *E. multilocularis*

**The cycle of *Echinococcus granulosus* in Europe is predominantly domestic** involving dogs as final hosts and sheep, cattle, pig and horses as intermediate hosts (Schantz *et al*, 1995). Wild animals can occasionally be involved in the cycle.Dogs harbour tens to thousands of taenias in their small bowel, without any specific symptom or sign, except anal pruritus in some cases. Thousands of eggs contained in the detached fertile segments of the worms are dispersed on the ground with the faeces of the carnivore and can be eaten by the intermediate hosts when grazing; because of their small size, eggs may also contaminate water. Additional dispersal of eggs by insect has also been shown. Finally, eggs may also be spread in the fur when the dog leaks its anus.

In intermediate hosts, the typical cysts can be observed at examination of viscera at the abattoir, as watery cysts, the diameter of which may range from 1 to 50 cm, single or multiple, in the liver and/or lung. Fertile cysts contain hundreds of protoscolices, that appear as "sand" ("hydatid sand") at the bottom of the cysts. The cycle of the parasite can be completed when dogs have access to infected viscera with fertile cysts, or when they are deliberately fed with these viscera (Schantz *et al* 1995).

**The cycle of *E. multilocularis* in Europe is predominantly sylvatic** involving red foxes as final hosts and rodents as intermediate hosts. In some countries dogs and cats have also been identified as final hosts. Typically, all final host species acquire the infection from the sylvatic cycle by uptake of rodents infected with metacestodes of *E. multilocularis*. Contamination of eatable vegetables or fruits by infected faeces of the infected carnivores and/or fur occur similarly, as described above.In intermediate hosts (voles of various species, and musk rats, in Europe), the disease may be observed as a polycystic abdominal mass; depending on the rodent species, fertility is ensured or not (*i.e.* protoscolices can develop or not); foxes, dogs and cats become infected by eating infected rodents with fertile metacestodes (Ammann and Eckert, 1996).

For both species, food-borne human contamination occurs by eating the eggs dispersed in the environment from adult worms present in the faeces of infected carnivores.

B. Hazard characterisation

(1) Diseases

*Cystic echinococcosis (CE)*

The larval form of *Echinococcus granulosus*, when developing in the intermediate host, including humans, is characterized by its cystic aspect and behaves as a benign tumour in most of cases (Ammann and Eckert, 1996). The disease occurs in both males and females, the sex ratio being different in the different endemic areas, depending on the characterisics of the parasite cycle in this area, and of particular behaviours in the communities. Children are often found infected, because of their closer contacts with dogs or contaminated environment. Cysts may be unique or multiple; main locations are the liver and the lungs; however any tissue or organ may be involved, including brain, bone, spleen, and kidney. This explains that after a silent asymptomatic period (lasting from months to years after contamination), various symptoms and signs are observed, depending on the primary location of the cyst(s), and of the duct or vessel structures which are compressed, obstructed and/or invaded. Major complications occur when the cyst(s) ruptures into bile ducts or peritoneal cavity, for instance, or when it (they) develop(s) in the brain. Because of the immune reaction against the parasite, associated with IgE production, allergic reactions, including anaphylactic shock may occur, especially when the cyst ruptures,

spontaneously, or during surgery and/or puncture.Rupture of the cyst leads to dissemination of the fertile form of the larva, protoscolices, and leads to secondary echinococcosis, dissemination to many tissues and organs; at this stage, surgery, considered as the treatment of choice in CE, is no longer possible.In fact, several advances have modified the treatment of cystic echinococcosis in the past 10 years, including drug therapy, using bendimidazole compounds, and ultrasound-guided puncture of the cyst, aspiration, injection of a scolicide agent, and reaspiration (the PAIR technique) (Akhan *et al.* 1996; Filice *et al.* 1997; Horton, 1997; Vuitton, 1999; WHO-IIWGE, 1996). Mass screening using ultrasound examination and serology are both feasible and efficient (Craig *et al*, 1996); however, the natural history of small cysts disclosed in some subjects at mass screenings is unknown and this should be studied in order to avoid useless and expensive treatment for cysts which could have been cured spontaneously. Puncture, Aspiration, Injection, Reaspiration can now be proposed as complementary therapeutic action following mass screening; efficiency of this cyst management has been assessed, especially in Italy (Filice *et al*, 1997).

*Alveolar echinococcosis (AE)*

AE in humans presents, in most cases, as a liver tumour, which mimics a cancer, progressively invading bile ducts and liver vessels and leading to numerous complications (Bresson-Hadni *et al*, 2000). Metastases may occur, especially in the lungs and in the brain. The latter location seems to be favoured by immunosuppression (Bresson-Hadni *et al*, 1999). The European collaboration in EurEchinoReg made recently available the information on 579 patients with AE throughout Europe, including Turkey (for details on these unpublished data, see the report of the "Eurechinoreg Pilot Project" in Appendix). This is the far largest number of cases ever assembled in the endemic foci of the European Union and border countries. The conditions leading to diagnosis of AE are available for 378 patients in the European countries. Most patients were diagnosed with AE in the age range from 50 to 70. However, the recruitment in Germany and Eastern France indicates a shift towards younger age groups ( < 40 ). The circumstances of AE diagnosis was evaluated. In contrast to the published literature, 70 % of the patients were referred to health centres because they had symptoms, 25% without (the disease was diagnosed by chance, usually because of an ultrasound exam performed for various purposes, and sometimes because of mass screening). In nearly all cases, the primary infected organ was the liver, although primary extrahepatic manifestation of disease occurred in 2,6 %. This figure is higher than previously anticipated. Most impressively, at the time of diagnosis 12,4 % of patients had already metastatic spread of the disease.According to the data set, 9% did not receive specific treatment for AE. This first glance on the data readily indicates the necessity of a centralised European institution such as the EurEchinoReg Registry and of Reference Centres at the European level. Until the beginning of the eighties, the disease was fatal within 5 years in most cases; because of earlier diagnosis and better medical management (including surgery, continuous treatment with albendazole, and ultimately liver transplantation in some cases), the prognosis and quality of life has improved a lot in most of patients; this improvement of the patients' condition was associated with a considerable increase in economic cost because of the medical treatment for life, of the cost of major operations including liver transplantation and of a necessary regular follow-up, all measures being proven very efficient at the individual's health level (WHO-IWGE, 1996; Ammann and Eckert, 1996; Bresson-Hadni *et al*, 2000).

(2) Virulence / pathogenicity

Four species of *Echinococcus* (*E. granulosus, E. multilocularis, E. oligarthrus* and *E. vogeli)* are infectious to humans. However, geographical distribution of these strains makes that only *E. granulosus* and *E. multilocularis* are of concern in Europe.

All studies, until now, have failed to demonstrate any difference in virulence for humans among various isolates of *E. multilocularis,* despite differences in their growth in experimental animals (Eckert, 1997).

For cystic echinococcosis, the epidemiological situation is complicated by the fact that several strains of *E. granulosus* have been identified in Europe exhibiting morphological, biological, genetic and other differences (see Bowles *et al.,* 1995 ; Thompson *et al.,* 1995 ; Eckert and Thompson, 1996). Data on *E. granulosus* strains in Europe are summarised in Table 1. Among other features strains may differ in their life cycles and preferences for certain intermediate hosts.

In addition, different degrees of infectivity appear to exist among the currently recognised *E. granulosus* sub-strains; epidemiological evidence and molecular studies indicate that the sheep, cattle and deer strains are infective to humans; a particular pig strain is also infective, as shown in Poland; according to present knowledge the horse strain may have low or no infectivity; the question of infectivity of other strains warrants further studies (Eckert, 1997).

**Table 1. Strains of *Echinococcus granulosus* in Europe** (after Eckert and Thompson;1996)

| Strain | Final hosts | Intermediate Hosts | Infectivity to man | Known geographical distribution |
|---|---|---|---|---|
| Sheep (S) | Dog<br>(fox, wolf) | Sheep, goat<br>Cattle, pig | Yes | Western, Southern and Southeastern Europe |
| Cattle (CA) | Dog | Cattle | Yes | Belgium, Germany, Luxembourg, the Netherlands, Scandinavian countries ?<br>Switzerland |
| Pig (P) | Dog, (fox ?) | Pig | Yes ? | Austria, Germany, Czech Republic, Hungary, Poland,<br>Slovakia, Balkan countries |
| Horse (H) | Dog | Horse, other equines | low or no | Belgium, Ireland, Italy, Switzerland, UK |
| Cervid (CE) | Wolf, dog | Cervids | Yes | Northern Norway, Sweden, Finland |

Until recently, strains of *E. granulosus* were identified using morphological, biological, biochemical and some other criteria. In recent years molecular techniques have allowed a more precise strain identification at the DNA level (Bowles *et al.,* 1995, Thomson *et al.,* 1995 ; Eckert and Thompson, 1996). Based on the presently available knowledge on life cycles, intraspecific variation and epidemiology of *Echinococcus* species the following conclusions can be drawn : a) the concept of strain diversity within the species *E. granulosus*, previously based on morphological, biological, biochemical and other

features, has been principally confirmed by modern genetic studies. However, detailed comparative studies must be undertaken in a number of endemic areas in order to determine the geographical distribution and uniformity of any nominated species; 2) several molecular techniques are now available which allow the identification of certain *Echinococcus* species and strains using genetic markers; 3) for comparative studies on a global scale a reference laboratory should be established and adequately funded by international organisations (Eckert, 1997).

(3) Dose-response

As in humans, diagnosis of the diseases is usually performed many months or years after contamination, no dose-response evaluation is available. Experimental studies suggest that a single egg can be effective, provided that the host is susceptible.

(4) Immunity

In fact, like many other parasitic diseases, echinococcosis, and especially alveolar echinococcosis, appears now as a "polar disease"(according to the definition given, for instance, in leprosy): its occurrence as a disease, after successful contamination, as well as its severity are different depending on the receptivity of the host, that is linked to host immune defences (Vuitton *et al*, 1990; Wakelin, 1997). Mass screenings have shown that abortive forms did exist, and could well represent the majority of cases (Rausch *et al*, 1987; Bresson-Hadni *et al*, 1994) this could explain the relatively rare incidence of the disease in alveolar echinococcosis (Vuitton *et al*, 1990), and the natural history of most small hydatid cysts that never grow to symptomatic diseases (Frider *et al*, 1999; Larrieu *et al*, 1999b). Multicenter studies of the HLA groups of AE patients in France, Germany and Switzerland suggest that both occurrence and severity of the disease could be genetically determined (Eiermann *et al*, 1998; Godot *et al*, 1999). Experimental studies in infected mice, as well as immunological studies in humans have revealed the importance of cell-mediated immunity in the control of the larval growth, and the role of the cytokine profile of the patient to promote continuous larval growth (Dixon *et al*, 1978, 1982; Vuitton *et al*, 1989; Liance *et al*, 1990; Bresson-Hadni *et al*, 1990; Emery *et al*, 1996; Godot *et al*, 1997; Siracusano and Vuitton, 1997). Various states of immunosuppression, like infection with Human Immunodeficiency Virus, organ transplantation, or chronic auto-immune disease treated with immunosuppressive drugs, or even pregnancy, have been shown to be associated with a faster progression of alveolar echinococcosis (Bresson-Hadni *et al*, 1992; Liance *et al*, 1992; Sailer *et al*, 1997; Bresson-Hadni *et al*, 1999). Similar observations are now reported for cystic echinococcosis (Panosian, UCLA, Los Angeles, CA, personal communication). Because of the increase in the number of patients with such associated conditions in our developed countries, it can be expected that echinococcosis will be more and more observed as an "opportunistic disease". It is of interest that among the first three autochtonous cases of AE reported in Belgium in 1999, two had an immune suppression (one because of AIDS, and the second one because of an immunosuppressive treatment for a chronic arthritis) (Carlier and Losson, personal communication; EurEchinoReg registry data, 1999; see summary report in Annex). Conversely, because of the role of the immune system of the host in disease development, alternative therapeutic options can be developed, using immunomodulation (Harraga *et al* 1999a and b), and should be tested at a European level.

<u>B. Exposure assessment</u>

(1) Microbial ecology

Because of the particular cycles of the parasites, *E. granulosus* and *E. multilocularis* eggs, which are the infectious agents for humans are dispersed in the environment with the faeces of the carnivores and may contaminate various types of food, including fruits and vegetables collected in kitchen gardens or in infected meadows, and drinking water. Protected by an oncospheral membrane, *Echinococcus* eggs are extremely resistant to environmental conditions (Lethbridge, 1980; Gemmell and Lawson, 1986): as an example, *E. multilocularis* eggs may remain infectious at temperatures ranging from –30 to +60°C; they are readily destroyed by heating, but may survive months or even years at low temperature, especially if they are protected against drying; freezing at –20°C does not prevent infectious potency (Thompson, 1995; Schantz *et al*, 1995).

Adult worms (Taenias) of both species are only sensitive to a very limited number of anti-helminthic drugs : nitroscanate must be used on an empty stomach, 2 days apart, at a daily dosage of 200 mg/kg; praziquantel is the drug of choice (single dose of 5-10 mg/kg); it must be stressed, however, that both drugs are not effective against the oncospheres (eggs), contained in the fertile segments, and which are the contaminating forms of the parasites, and that any de-worming, at the individual or population level, must take this fact into account.

The larval forms, especially cysts found in animal viscera at the abattoir, are not infectious to humans, but only to carnivores that will develop the adult form and the eggs.

(2) Prevalence in food

Due to the lack of suitable tools to distinguish *Echinococcus* eggs from eggs of other cestodes not infectious to humans, no data are available on the actual prevalence of the organisms in food or in drinking water. The recent development of appropriate techniques (antigen detection, using ELISA, and/or specific DNA detection using PCR) should make such an assessment possible in the future (Deplazes and Eckert, 1996; Craig *et al*, 1996; Craig, 1997). An indirect estimate of exposure can be given by the prevalence of infected dogs (for CE) and foxes (for AE); data on cyst prevalence at the abattoir also gives an indirect estimate of the potential risk for humans to be infected in a given region or country (slaughtered animals suffer the same disease as humans, and the prevalence of cysts in these susceptible hosts is usually correlated to exposure to eggs and to prevalence in humans. Tables 2 and 3-A and -B give the available prevalences of *E. granulosus* and *E. multilocularis* infections in animals respectively. It must be stressed that, because of different policies and/or compliance in reporting data at the European level, data may be not available for some countries, and data obtained from published sources (*i.e.* from epidemiological studies performed by research teams) may differ greatly from those reported to the EU reporting centre in Berlin (given in Annex II). Details on the epidemiological data on prevalence of infection in final and intermediate animal hosts at the country level in EU Member States will be given together with human epidemiological data in section C (1).

Table 2. Prevalence of *E. granulosus* in animals and its controls in the Mediterranean countries

| Country | Percentage of Infection | | | | | Disease Control |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Goats | Pigs | Dogs | |
| Albania | Highly endemic in humans and animals | | | | | No control programme |
| Bulgaria | 16,47 | 2,75 | -- | 1,64 | 8,10 | No control programme |
| Cyprus | 0,06 | 0,02 | 0,005 | 0,0 | 0,0 | National control programme |
| France | No data available | | | | | Control measures |
| Greece | 15,3 | 21,1 | 9,5 | 0,31 | 0,17 (1993) | National control programme |
| Italy<br>Sardinia | 0,54-0,72<br>23,7 | 4,57<br>23,7 | 5,45<br>23,7 | 0,2-0,4<br>17,6 | <br>16,2 | Control programme |
| Malta | No incidence reported in humans or animals | | | | | |
| Portugal | 0,6-5,7 | 0,6-3,8 | 0,1-1,2 | 1,3-8,9 | 10,4 | Control programme |
| Spain | 22,0 | 42,2 | 12,6 | 7,6 | 13,5 | Notifiable disease Control programme |
| | Data for animals 1986 | | | | | |
| Turkey | 3-40 | 4-59 | 2-30 | -- | 0,32-59,2 | Dog treatment |
| Yugoslavia | 10-40 | 40-80 | -- | Up to 30 | 8,15-65 | Treatment of dogs |

Table 3 A. Examples of the prevalence of *Echinococcus multilocularis* in red foxes (*Vulpes vulpes*) in European countries (according to Eckert, 1997, with modifications)

| Country/Region | Period | No of animals examined | Prevalence % |
|---|---|---|---|
| France (Lorraine) | 1983-1987 | 513 | 14-36 |
| Switzerland : 21 cantons | 1990-1995 | 7059 | 29.0<br>3-53 |
| Liechtenstein | 1990-1992 | 129 | 34.9 |
| Austria : 9 districts | 1989-1995 | 3600 | 1-35 |
| Germany : | | | |
| Bayern | 1988-1992 | 3042 | 27.3 |
| Bad.-W [1](Tübingen) | 1993-1994 | 679 | 44.8 |
| Bad.-W [1](Karlsruhe) | 1889-1990 | 801 | 11.6 |
| Hessen (Kassel) | 1989-1990 | 162 | 29.0 |
| NRW [2](Detmold) | 1989-1990 | 153 | 9.1 |
| Niedersachsen | 1991-1994 | 2412 | 13.5 |
| Thüringen | 1990-1992 | 1128 | 18.2 |
| Brandenburg | 1992 | 339 | 8.5 |
| Schleswig-Holstein | 1994-1995 | 382 | 0.5 |
| Poland : 10 districts | 1994-1996 | 1752 | 1.4<br>0.47-11.3 |

For references see Eckert, 1996 a, b, and Ramisz *et al.* (1996)

[1]Baden-Wurttemberg. [2]Nordhein-Westfalen.

Only published data are given in this table; for data obtained from the EurEchinoReg surveillance programme (EC DGV report), see in the Appendix.

Table 3 B. Examples of the prevalence of *Echinococcus multilocularis* in dogs and cats in European countries (according to Eckert, 1997, with modifications)

| Country/Region | Period | No of animals examined | Prevalence % |
|---|---|---|---|
| Dogs | | | |
| France (Haute-Savoie) | 1978-1983 | 36 | 5.6 |
| Switzerland (Eastern) | 1995 | 661 | 0.3 |
| Cats | | | |
| Germany | | | |
| Baden-Württemberg | 1974 | 207 | 0.5 |
| Baden-Württemberg | 1988 | 498 | 1.0 |
| Baden-Württemberg | 1989 | 170 | 2.9 |
| Thüringen | 1992 | 58 | 3.4 |
| Switzerland (Eastern) | 1995 | 452 | 0.22 |
| France (Haute-Savoie) | 1999 | 80 | 5 |

For references see Althe r, 1996 ; Eckert, 1996 a, b.

(3) Consumption data

Because of the great variety of foods susceptible to be contaminated, and the fact that they usually do not come from commercial circuits, no consumption data are available.Contamination of gardens and water by dog faeces containing *E. granulosus* eggs has been shown (Schantz *et al*, 1995). Preliminary results from a recent sociological study among families living in an area endemic for *E. multilocularis* infection have shown that most individuals actually ate foods from non-fenced kitchen gardens and that collecting berries, dandelions and other vegetables and eating them without cooking was a very common behaviour in these areas (Marcel, 1998, personal communication); such studies should be extended to other areas to give more insight into real exposure, especially through consumption of contaminated vegetables from non-commercial but also commercial sources.

C. Risk characterisation

(1) Prevalence and incidence in humans and in animal hosts

*Cystic echinococcosis (CE)*

*E. granulosus* has an uneven geographical distribution in Europe with very low prevalence in some of the northern and central European countries, with medium endemicity in others and high endemicity in areas of southern and eastern Europe.In northern countries of the EU, most of cases (except some autochtonous cases in the United Kingdom and in the northern part of Scandinavian countries), are imported cases, observed in immigrant workers from endemic areas of the EU (Mediterranean countries), or from Turkey, central Europe border states, and Middle-East countries, or from the Maghreb (Schantz *et al*, 1995; Eckert, 1997). This report will thus mainly focus on the situation in Mediterranean areas because of its Public Health significance, both in Member States of the EU and in border countries. It should be stressed that the situation in border countries of the East-Central Europe has impaired badly during the past 15 years, due to various reasons including changes in the health protection system, lack of enforcement of regulations on slaughter and/or domestic dogs, and lack of hygiene.Echinococcosis/Hydatidosis is highly endemic in the Mediterranean countries. The disease prevalence and its public health importance in each country is variable.Particularities of the situation regarding cystic echinococcosis in the various countries (including central Europe border countries and

Turkey) are given in the following section. Most of the information comes from reports by the WHO Mediterranean Zoonoses Control Centre, recently summarised at the XIXth International Congress of Hydatidology, Bariloche, Argentina, Sept 99, by P. Economides (Economides, 1999; detailed references may be found in this review).

## Bulgaria

Cystic Echinococcosis is a major public health and animal health problem in Bulgaria (Todorov and Boeva, 1999). According to data from the Bulgarian Veterinary Service the prevalence of hydatid cysts among food animals during the early 1960 was as follows: cattle : 58% ; sheep : 78% ; swine : 2,8%. The dog population in the 60's was approximately 1.000.000 and the prevalence of *E. granulosus* varied between 23 and 44 %. In humans the annual surgical incidence of cystic echinococcosis varied from 1,9 to 16,6 per 100.000 population. Very high incidence was observed in eight districts. The average incidence in humans during the period 1950-62 was 6,6 operations per 100.000 people.In 1960-61 an echinococcosis control program was planned to cover the entire country. The program was implemented by the Veterinary and Health Services with the assistance of Local Authorities and other public organisations. The implementation of the control measures contributed greatly to the interruption of transmission in many parts of Bulgaria and has led to a considerable improvement of the situation especially in the incidence in humans. During the years 1971-82 the number of surgical operations per 100.000 people was reduced to an average of 2 (lowest 1,2 – highest 2,7). Unfortunately the incidence in humans during the years 1983-95 showed an increasing trend which became quite marked during the last three years of the period when it reached 6,8 per 100.000. From an analysis of the human cases by age for the three periods it was revealed that the increase during the year 1995 was very high in children 2-16 years. Similar changes were observed in the infestation rate of food animals. Studies in some regions of Bulgaria have shown that the prevalence of *E. granulosus* in dogs was 8-10%. The increased incidence of cystic echinococcosis in humans and animals during the last ten years is attributed to the discontinuation of the control program in 1987 due to the social and political changes in Bulgaria and lack of funds. At present, echinococcosis is considered of serious concern for the Veterinary and Health Authorities of Bulgaria and there are hopes that the control program will be reintroduced.

## Cyprus (Economides, 1994; Economides *et al*, 1998)

Echinococcosis/Hydatidosis (*E. granulosus*) was wide-spread in Cyprus before the 1970s. Almost every mature food animal were infected and hence Echinococcosis/Hydatidosis was also a very serious public health problem. The large number of stray dogs, the uncontrolled slaughter of animals and the disposal of infected offal anywhere in the vicinity of abattoirs or in the fields, as well as the ignorance of the people, were the factors contributing to the perpetuation of this disease in the island. A successful eradication campaign was implemented during 1971-85. This campaign was based upon a drastic reduction of the dog population, with the killing of more than 85.000 stray dogs, and a systematic arecoline dog-testing programme with euthanasia of all positive dogs. These intensively enforced measures drove the campaign to very quick results. The attack phase was shortened to about 12-13 years, as was also the case in Falkland Islands, where the campaign was based upon a 6-weekly dog-dosing programme using praziquantel. Excellent results were observed in humans and food animals in the Government controlled areas and can be favourably compared with successful programmes in other countries. In the areas not under Government control the disease remained endemic with very high incidence in humans and animals. Unfortunately after the Turkish Invasion in 1974 and the

occupation of about 37 per cent of Cyprus the programme continued only in the areas controlled by the Government. Sporadic infection of dogs in villages, even far from the Turkish part of the island, have been disclosed since the beginning of the nineties, and a new increase in the incidence of human cases could be shown. A new programme of control ("emergency plan against echinococcosis") has thus been implemented by the department of Veterinary Services, Ministry of Agriculture, that started in 1994, and led to a marked reduction of cyst prevalence in sheep (by 73.3 %), cattle (by 85.7 %), and goats (by 93 %).

France (FAO/OIE/WHO Animal Health Book, 1997)

Cystic echinococcosis is reported present in food animals, mainly sheep, goats and cattle, especially in the south of France.In man incidence is 4,5-13/100.000 in some areas, including "imported cases" in immigrants, in most of the country (especially in big industrial cities/areas), and autochtonous cases in the Mediterranean area of continental France and in Corsica where incidence is the highest.

Greece
(Charissis, Ministry of Agriculture of Greece, c/o MZPC Greece, personal communication)

At present there are about 3,4 cases of cystic echinococcosis (*E. granulosus*) in humans annually per 100.000 inhabitants; 10-15 years ago this was more than 12/100.000. A control programme has been in force since 1984. This includes testing of faecal samples for *E. granulosus* and systematic administration of praziquantel to shepherd dogs. Also, slaughterhouse facilities are being improved for more effective control of slaughter. Education on the various aspects of this problem is carried out in schools. The prevalence in food animals has been calculated to be 0.56-15.3 % in cattle, 0.06-21.1% in sheep, 9.5% in goats and 0.01-0.58% in pigs, while in dogs it is about 0.2%.

Italy (Gabriele *et al*, 1997; Arru *et al*, 1999; Conchedda *et al*, 1997)

In Italy *E. granulosus* infection is prevalent in all parts of the country with most of cases in the south of the country. Average annual incidence in humans in Italy is about 2/100.000. Infection in sheep varies widely from area to area, *i.e.* from 10 to 96%. In a survey at four public slaughterhouses in a district of the province of Messina, from 1991 to 1993, infection in bovines was 11%, ovines 43%, caprines 3% and porcines 5%. In the Abruzzi Region the mean yearly prevalence at slaughter, from 1990 to 1994 in sheep and goats was 7%. In humans the number of operated persons was 2,4/100.000. Sardinia has always been one of the worst affected areas in the Mediterranean with regard to CE. In 1988, the prevalence was 86.9% in sheep, 23.7% in goats, 22 % in cattle and up to 60 % in swine, causing losses of about 30 thousand million lira a year. Infection of dogs ranges from 13 % (in pets living in houses to 25 % in shepherd dogs). The average annual incidence in man was 20 cases per 100.000 inhabitants in the 1975-1980 period. Various control strategies have been put into action. Control programmes conducted in Sardinia in 1960, 1962 and 1980 have failed. The 1993 program was based on health education, keeping the canine population under control and inspection of slaughterhouse. However, it was not possible to control the disposal of all sheep entrails, which is the main source of infestation of dogs, partly because of the widespread practice of illegal slaughter of sheep by local farmers, who were inclined to do so on account of the low commercial value of the adult animals, often lower to transport and slaughter costs. A new programme includes treatment of dogs and financial incentives for farmers, to make it worthwhile to

send their adult animals to slaughter in public abattoirs and obliged them to hand over the entrails of sheep killed on the farm, to the Public Health Authority for destruction.

Malta (Vella, Veterinary Services, Ministry of Agriculture and Fisheries, Malta, personal communication)

No incidence in humans and animals is reported. The Veterinary Services have full control of slaughterhouses and carry out meat inspection on all animals slaughtered.

Portugal (Rosinha, Servicos de Sande Animal, Ministerio deAgricultura, de Desealvimento Rural e das pescas, Portugal, personal communication)

Human incidence is about 2,2/100.000. The prevalence in animals is reported to be 0,6-5,7% in cattle, 0,6-3,8% in sheep, 0,1-1,2% in goats and 1,3-8,9% in pigs. There is a Plan of Control for the country.

Spain (Garcia, 1994, and personal communication 1999)

In 1985, there were about 1,000 human cases (about 2.5/100.000). Each year there was a decrease *i.e.* there were about 700-600 cases per year in the 1980s and 500-300/year in the 1990s. In 1997 the incidence rate dropped to 0.78/100.000. In animals the epidemiological situation is similar. There is a continuous reduction of infection in all food animals. However, it should be stressed that the prevalence of infection was very high in some regions: in La Rioja, for instance, 82.3 % of sheep were infected with a mean number of cysts per animal of 6.5; it decreased gradually with years to reach 27.4 %, with a mean number of 1 cyst per animal in 1998. These excellent results were achieved after the implementation of national control programmes by the Autonomous Communities of Spain. Until 1985, the main measure for the control of *E. granulosus* was the massive use of anthelminthics of dogs and public health education. During the XIII International Hydatidosis congress which was held in Spain, a complete National strategy for the control of Echinococcosis was developed by national and foreign experts and was given to the Autonomous Community of Navarra for implementation as a pilot programme for the Mediterranean area under a continuous evaluation by WHO, the Mediterranean Zoonoses Control Centre and the Institute Superiore di Sanita in Rome.Gradually the Autonomous Communities of La Rioja (1987), Madrid (1989), Castilla – La Manda (1988), Aragon (1990) and Castilla – Leon (1994) have undertaken the responsibility for the enforcement of similar programmes adapted to their local conditions. All programs were supported financially by the Ministry of Health and Consumer Affairs which is also responsible for their supervision and annual evaluation. After 1996 the autonomous communities are fully in charge for the financing and implementation of the programmes which are considered very successful.

Turkey (Ozcel, 1994)

Incidence in man ranges from 1-20/100.000. The disease is reported in all food animals. The prevalence ranges from 4-59% in sheep, from 3-40% in cattle and 2-30% in goats. In dogs, infection is between 0.32-59.2%. Echinococcosis/Hydatidosis is a major economic and health problem because: about half of the population live in the rural areas which are high-risk areas ; shepherd dogs are usually kept with flocks of sheep or cattle ; home slaughtering is common ; there is a large stray dog population which is difficult to control. There is no national control programme.Dog control and treatment of owned dogs is practised in some areas of Turkey.

Ex-Yugoslavia states ( Katic-Radivojevic and Popovic, 1997; Pavlovic *et al*, 1997)

Cystic echinococcosis is a very serious problem for animals and public health in ex-Yugoslavia. The infection rate of dogs in towns of the coast in Montenegro was found by the same scientists to be 65% which is very high and must be considered as a very important factor for human infections. Similar findings were reported by Pavlovic I. *et al. 1997* in the Belgrade area. Treatment of owned dogs with praziquantel is one of the measures applied by veterinarians. There is no National Control programme.With the recent war and massive movements of people and animals, the problem of cystic echinococcosis will become very serious.

*Alveolar echinococcosis (AE)*

Alveolar Echinococcosis in humans.

Some incidence rates from European countries based on well documented studies are summarised in Table 3. The average incidence rates as referred to the total population of a country is rather low : 0.18 (1970-1983) and 0.10 (1984-1992) cases per 100,000 inhabitants per year, in Switzerland 0.02 and 0.03 in Austria and Bavaria in Germany. However locally, because of the geographically focused nature of the parasitic cycle in nature, the incidence rates may be higher, for example in the Canton Jura in Switzerland with 0.74 or with 1.4 in the Département Doubs in France.This has to be taken into account when examining the actual risk in a given European region.

Table 4. Alveolar echinococcosis in humans ; annual incidence rates per 100.000 inhabitants

| Country | Period | Incidence | Reference |
|---|---|---|---|
| Austria | | | |
| Whole country | 1983-90 | 0.02 | Auer and Aspöck, 1991 |
| France | | | |
| Franche-Comté | 1971-898 | 0.5 | Bresson-Hadni, 1988 |
| Doubs | 1960-92 | 1.4 | Bresson-Hadni *et al.*, 1994 |
| Franche-Comté | 1983-1998 | 0.7 | Bresson-Hadni *et al.*, 2000 |
| Germany | | | |
| Bavaria | 1985-89 | 0.03 | Northdurft *et al.*, 1995 |
| Switzerland | | | |
| Whole country | 1970-83 | 0.18 | Gloor, 1988 |
| Whole country | 1984-92 | 0.10 | Eckert *et al.*, 1995 |
| Canton Jura | 1970-83 | 0.74 | Gloor, 1988 |

Compared with some other infections these incidence rates are low. However, it has to be considered that untreated alveolar echinococcosis is mostly lethal, and treatment is very expensive and unpredictable in outcome (Ammann and Eckert, 1996 ; Bresson-Hadni *et al.*,1997, 1999, 2000). In this connection it may be mentioned that the incidence rate of the Creutzfeldt-Jacob-Disease is about of the same order of magnitude (1.1 per 100,000 inhabitants per year) as alveolar echinococcosis (Anonymous, 1996). Regarding the latter disease health authorities and politicians are deeply concerned but they are rather insensitive to the public health aspects of alveolar echinococcosis.

Humans belong to the group of “accidental intermediate hosts”. In recent years (1980ies and 90ies) autochthonous human cases of AE have been reported and documented from several European countries, namely Austria, France, Germany, Switzerland, and the European part of Turkey (see Ammann and Eckert, 1996; Bresson-Hadni *et al*, 1988, 1997, 2000; Eckert, 1996 a, b; Schantz *et al.*, 1995; Stössel, 1989; Vuitton *et al*, 1990). In addition, sporadic “imported” cases were described from Belgium (Claudon, 1983), and sporadic, not well documented cases, in the Czech Republic (Slais *et al.*, 1979), Poland (Pawlowski, 1996) and Greece (Theodoropoulos *et al*, 1978). However, the establishment of a European Registry of cases in 1998, funded by the European Commission DG V, has allowed a more accurate collection of human cases. So, in addition to previously well recognised EU regions where the disease is endemic in rural areas, human AE cases are now disclosed in regions located at the border of endemic areas, but not previously identified as ”at risk” (the very recent identification of 3 autochtonous human cases in Belgium is undoubtly of concern), in cities of the endemic areas and, even, of the non-endemic areas, in populations not considered ”at risk” in the past, and in central Europe countries at the border of the EU, an area considered up-to now as ”free” of *E. multilocularis* life cycle, between the ”Russian” and the ”European” foci of infection.

In view of the high prevalence rates of *E. multilocularis* in foxes in large regions of Europe the incidence rates of human alveolar echinococcosis are surprisingly low. It has to be underlined that these incidence rates refer to diagnosed clinical human cases. As the incubation period in alveolar echinococcosis is estimated to be very long – from 5 to 15 years (Ammann and Eckert, 1996) – incidence rates may not reflect the actual and current infection risk for humans (better estimated by infection prevalence in final hosts, foxes and , perhaps, dogs and cats. On the other hand, because of the chronic evolution of the disease in EU countries where appropriate treatment is available, prevalence figures are obviously much higher than incidence rates, but give an indirect estimate on the real impact of this disease on Public Health. Table 4 gives the available published data on annual incidence of AE in EU countries. Updated figures are given in the report to DG V by the “Epidemiological European surveillance of alveolar echinococcosis” group (see summary of the report in Annex).

<u>*E. multilocularis* infection in definitive animal hosts</u>

Until the end of the 1980s some areas in France, northern Switzerland, southern Germany, and Austria were known as endemic for *E. multilocularis*. Since 1989 extensive surveys have been carried out in several countries which revealed that *E. multilocularis* occurs further north, south and east than previously anticipated. The data presented in this report are mainly taken from a recent review by Eckert (1997) and from unpublished data presented in the report of the Pilot project on “Epidemio-surveillance of alveolar echinococcosis in Europe” (EurEchinoReg registry) submitted to the European Commission DG V in June 1999 (see summary of the report in Annex III.

At present *E. multilocularis* is known to occur in red foxes in the following 11 countries of Central Europe (Romig *et al*, 1999): Belgium, Luxembourg, France, Switzerland, Liechtenstein, Austria, Germany, Poland (see Malczewski *et al.*, 1999 ) the Netherlands, Czech Republic (Kolarova *et al.*, 1996; Kolarova, 1999) and Slovak Republic (Dubinski *et al.*, 1999) (Fig.1); although it is likely that foxes are also infected in northern Italy and Greece, sound data are not available in these countries. Unpublished results from Spanish teams working on parasitic infections in wild animals in the Pyrenean area suggest that *E. multilocularis* infection is absent from both foxes and rodents in this area. *E.*

*multilocularis* is also endemic in Turkey; human cases of alveolar echinococcosis have been reported predominantly from the Asian part of the country but also from the European Marmara region. The prevalence rates of *E. multilocularis* in red foxes are alarmingly high in some areas where the average rates may reach values of over 40% (Table 2). These rates show a wide variability between countries, regions and even between small territories. The continuing adaptation of wild foxes to living conditions in settlement areas and cities is on record from many areas in central Europe.The most detailed information exists from the city of Zurich in Switzerland where 44% of 'city foxes' were found infected (Deplazes *et al*, 1999b). Similar studies have only been done in Stuttgart (Southwestern Germany), where an average of 20% carried the parasite (Romig *et al*, 1999). This development causes concern that a far bigger section of the human population might be at risk to contract AE than previously assumed. Also, high infection rates of 'city foxes' increase the probability of transmission to pet dogs and cats (via rodents in parks and recreational areas).

There are several regions of Europe where *E. multilocularis* does not seem to occur. Its absence in the Mediterranean area may be caused by unfavourable climatic conditions while its absence in Scandinavia - which has favourable climate, landscape and intermediate host fauna) is not yet clearly explained. *E. multilocularis* infection has, however, been shown in wild rodents living in Svalbard close to the Arctic, but no human cases have ever been reported. The British Isles also appears as an exception since all host animal species and favourable climatic conditions exist; the absence of the parasite may simply be due to the island situation as a barrier against immigration of the species.

Prevalence rates of *E. multilocularis* in dogs and cats are generally lower than in foxes. Some data are presented in Table 3 B. A recent comparative study in Switzerland using coproantigen detection and other methods has shown that among 661 dogs and 452 cats from eastern Switzerland 0.3% and 0.2% were infected with *E. multilocularis* (Alther, 1996). Three among 81 cats have been recently found infected in Eastern France, near Geneva, Switzerland, using autopsy examination (Pétavy *et al*, 2000). It has to be considered that in groups of dogs and cats which usually eat *E. multilocularis*-infected mice these rates may be higher, but detailed studies are lacking. Since the introduction of new diagnostic techniques, such as coproantigen detection and identification of taeniid eggs by PCR (Deplazes and Eckert,1996; Craig, 1997; Deplazes *et al*, 1999a and b), an increasing number of *E. multilocularis* infected dogs has been found. Model calculations considering the sizes of final host populations and other factors indicate that, for instance, in the Canton of Zurich (Switzerland) dogs and cats may carry about 9% and 20% of the total biomass of *E. multilocularis*(Eckert, 1997). In this connection it has to be considered that cats are regarded as less susceptible to *E. multilocularis* and may produce lower numbers of eggs than foxes and dogs. From these data it can be deduced that foxes are predominantly responsible for the contamination of the environment with *E. multilocularis* eggs. As their habitats include both the countryside and increasingly also urban areas, they may disperse infective eggs within various types of human settlements. However, in the above mentioned study (Petavy *et al*, 2000), fertile adult worms were found in cats. Whether or not for the European epidemiological conditions, the closer association of dogs and cats with households may increase the infection risk for humans is unclear. A recent case-control study on risk factors in Austrian patients with AE showed a significant correlation with cat owning (Kreidl *et al*, 1998).

## *E. multilocularis* infections in intermediate animal hosts

In Central Europe six species of Circetidae and one species of Muridae have been identified as intermediate hosts but this list may not be complete (Table 5). The common vole, the vole rat and the muskrat appear to be of special epidemiological significance.In the intermediate hosts the infection rates with *E. multilocularis* metacestodes are normally surprisingly low ranging from <1% up to 7% (Table 4) Both Giraudoux *et al.*, in France (Giraudoux, 1991; Giraudoux *et al*, 1997) and Gottstein *et al.*, in Switzerland (1997) have shown that local hot spots may exist with infection rates in rodents (*Arvicola terrestris)* of up to 39 %. In these areas the high infection rates in rodents were correlated with high prevalence rates of the parasite in foxes of up to 60 % (Gottstein *et al.*, 1997). A significant correlation between cyclic periods of high densities of voles and occurrence of AE in humans, in a given area has also been shown, high densities of rodents being favoured by landscape characteristics and land use by agriculture (Viel *et al*, 1999).

Table 5. Intermediate hosts for *Echinococcus multilocularis* in Central Europe

| Family and species | Countries | % prevalence of E. *multilocularis* infection[1] |
|---|---|---|
| Cricetidae :<br>*Arvicola terrestris* (Vole rat) | <br>CH, D, F<br>CH | <br>0.11-7.35<br>"Hot spot" : up to 39%[2] |
| *Clethrionomys glareolus* (Red-backed vole) | BU[3], D, F | 0.21-0.90 |
| *Microtus nivalis* (Common vole) | D, F | 0.16-8.91 |
| *Microtus nivalis* (Vole) | BU[3] | ? |
| *Ondatra zibethica* (Muskrat) | CH, D, F | <2-3 |
| *Pitymys subterraneus* (Earth vole) | F | 0.49 |
| *Muridae : Mus musculus* (House mouse) | CH, F | Sporadic |

[1]For references : see Eckert, 1996 a, b. [2]Gottstein *et al.*, 1996). [3]Findings questionable.

## Accidental animal hosts for metacestodes

Accidental hosts are animal hosts susceptible to the infection allowing partial or complete development of the metacestode stage of *E. multilocularis* but which most likely do not play a role in disease transmission. In previous times there were only a few reports on infections of accidental host animals with *E. multilocularis* metacestodes. It is of public health interest and concern that in recent years increasing numbers of such infections have been observed in animals, such as dogs, domestic pigs, wild boar, nutria, and monkey species (Table 6). These cases are indicators that disease transmission actually occurs in the respective regions. Of special interest are observations made by Deplazes (unpublished data) that two dogs had a simultaneous infection of the liver with metacestodes and of the intestine with adult stages of *E. multilocularis*. The modes of infection and parasite development in such cases are open for discussion.

Table 6. Accidental host animals for the metacestode stage of *Echinococcus multilocularis* in Central Europe.

| Species | Countries | References |
|---|---|---|
| Dogs (*Canis familiaris*) | Germany,Switzerland | Geisel *et al.*, 1990 ; Eckert, 1989 ; IPZ 1995,1996 |
| Domestic pig (*Sus scrofa domesticus*) | Switzerland | Deplazes *et al.*, (IPZ) |
| Wild boar (*Sus scrofa*) | Germany | Pfister *et al.*, 1993 |
| Nutria (*Myocastor coypus)* | Germany | Worbes *et al.*, 1989 |
| Monkeys (*various species*) | Germany,Switzerland | Reitschel and Kimming, 1994 Baumgartner, 1990 ; IPZ |

(according to Eckert, 1997).

References can be found in Eckert, 1997; IPZ = Institute of Parasitology, Zurich

Domestic pigs from Switzerland (breed : Edelschwein) are susceptible to intraperitoneal infection with *E. multilocularis* tissue (Pool-Wollmer 1993). However, the metacestodes in the liver persisted for only a short time and died out within 3-5 months. In 1993/1994 natural infections of domestic pigs were observed in a farm in eastern Switzerland (Deplazes and Eckert, unpublished data). Transmission of the parasite material from pigs to susceptible rodents did not provide evidence for viability. In a sero-epidemiological survey in this area, 522 breeding sows from 146 farms (3-5 animals per farm) were tested in the EmG11-ELISA for *E. multilocularis*-specific antibodies;15 animals from 7 farms had specific circulating antibodies (Deplazes and Eckert, unpublished data). All farms had in common that pigs were fed with grass from land accessible to foxes.

(2) Risk factors

*Cystic echinococcosis*

In the Mediterranean countries public knowledge about the disease and its transmission patterns is limited. The area allows the migration of animals from one country to another. Although sheep are the major intermediate hosts for *E. granulosus* in these countries, other livestock including cattle, goats, donkeys and pigs, are usually found heavily infected with hydatid cysts. High infection rates (up to 65%) in stray dogs have been recorded. The negligent attitude toward dogs in most Mediterranean countries leaves the dogs frequently exposed to infection and high worm infestation, shepherd, farm, and hunting dogs receive little care from their owners. De-worming of dogs is rare or non-existent.

Domestic animal husbandry, home slaughtering practices (particularly during religious and special ceremonies including in EU countries), poor meat inspection in town slaughterhouses and abattoirs, improper disposal of carcasses and offal coupled with abundance of stray dogs in some countries (especially those which belonged to the ex-URSS–related zone) all contribute to the spread and high endemicity of the disease.Because of cultural habits, Muslim communities, when they are present in a given country are often more at risk than other communities in the same country, but contamination from infected dogs may occur in subjects (especially children) of the same area that share the same environment; and the disease is also commonly observed in areas/countries only inhabited by Christian communities.

Regional cultural behaviours, such as collecting berries, and other vegetables in nature, or harvesting vegetables from open kitchen gardens, make rural people at risk. However, recent trends observed in human behaviour (hiking, hunting, and interest for organic food), in the infection of foxes (foxes infected in big cities, and overall increase in their numbers in Europe), and in animal breeding/rearing practices, could bring more opportunities to human populations to become infected.

The observed increase in the incidence of AE cases from the beginning of the 1980$^{ies}$ can partly be due to the improvement of diagnostic methods, especially the development and wide use of ultrasound imaging techniques. However, studies in animal hosts, and the correlation between human incidence and the land use by agriculture in the endemic regions support the hypothesis of a real increase in the risks to the populations due to an increase in the infectious forms of the parasites (the eggs) in the environment.

Within the recognised distribution range of *E. multilocularis*, the prevalence rates vary enormously. In most regions, hilly landscapes with cool climates and extensive agriculture seem to be most favourable for the parasite (Gottstein *et al*, 1997; Viel *et al*, 1999). One of the key factors may be the abundance of suitable intermediate hosts, especially common voles (*Microtus arvalis*) and water voles (*Arvicola terrestris*). The occurrence and population size of these species depends on the presence of unploughed grassland (pastures and meadows) which, in turn, is the type of landscape most typical for mountainous regions with cool climate where intensive agriculture is unfeasible. A correlation of *E. multilocularis*-prevalence in foxes and population densities of the above mentioned rodent species has been demonstrated in France (Giraudoux 1991), as well as a correlation between the number of human cases and the existence of cyclic high densities of rodents (Viel *et al.* 1999). High densities of rodents appear to occur only in those areas where land use is characterised by the absence of plough fields, and permanent meadows/pastures for cow-breeding, as increasingly developed in middle altitude plateaux of the Jura and Alps areas (Viel *et al.* 1999). This type of agriculture has been favoured by the EU agricultural policies since the 1960$^{ies}$ in nearly all areas where AE is now of concern. Similar landscape changes leading to high densities of rodents and increase in AE cases has also been observed in PR China because of deforestation. This is a striking example of unexpected consequences on Public Health of political /economic decisions with an impact on environment.

In a few regions, longitudinal studies using sufficient samples sizes allow a direct comparison between prevalence rates of fox infection at different periods. The largest sets of data exist for Baden-Württemberg and Rhineland-Palatinate where definite prevalence increases could be demonstrated (Romig *et al*, 1999; and summary of the EurEchinoReg Report to European Commission-DG V, in Annex). The contamination of the environment is not only influenced by prevalence rates, but to an even larger extent by the absolute number of foxes occurring in a certain area. Although many approaches have been made to estimate the fox population density, no generally accepted method is available at present. An approximation of temporal changes of fox population densities can be deduced from the hunting indices which are kept by authorities of several countries. As an example, the hunting indices of Baden-Württemberg show an increase by the factor 3 between 1989 and 1996. Similar data exist from other German 'Bundesländer', and evidence for increasing roadside counts exists from France. It is a generally accepted fact that in most parts of Europe the number of foxes has risen drastically (probably favoured by reduced mortality due to successful rabies control

campaigns). With or without a recognisable increase of *E. multilocularis* prevalence, a drastically increased output of the parasite's eggs within the previous decade has most likely occurred in the entire European range of the parasite.

The continuing adaptation of wild foxes to living conditions in settlement areas and cities is on record from many areas in central Europe. Infection of these "urban foxes" by *E. multilocularis*, as mentioned above, causes concern that a far bigger section of the human population might be at risk to contract AE than previously assumed. Also, high infection rates of 'city foxes' increase the probability of transmission to pet dogs and cats (via rodents in parks and recreational areas).

The British Isles are of special concern. Changing quarantine regulation in the U.K. have recently raised the topic of protection against accidental importation of *E. multilocularis*, *e.g.* with pet dogs or cats from the adjacent continent (Craig, and Pollitt, and the Advisory Group on Quarantine, personal communication). Unfortunately, there are very few data Switzerland and from the border regions in western Belgium and northern France, and, although unrecorded, the parasite may be widespread there. Studies in those areas are urgently required to allow a risk assessment and the design of effective protective methods. Moreover, dogs and cats can be imported from other parts of continental Europe that are well known as endemic areas. Hence, studies on prevalence in pet animals from these areas are urgently needed.

## II. RECOMMENDATIONS FOR RISK MANAGEMENT OPTIONS

### A. Cystic echinococcosis

Cystic echinococcosis is theoretically an eradicable disease (Arru *et al*, 1999; Economides, 1999; Thomson, 1999). Especially WHO and FAO have proposed guidelines to this purpose. However, because of the numerous factors involved in the maintenance of the cycle, including behavioural and cultural factors that are rather resistant to regulations many well designed control programmes have failed in various countries. The disease remains a threat to human health in some countries of the EU, and in most of the border nations of central Europe.The general rules for an efficient control of the disease are nevertheless rather simple and well known even if their implementation may be problematic in some areas.

(1) Farm level (CE)

Management at the farm level includes the following measures:

- Control of stray dogs populations; spaying of bitches;
- Registration of owned dogs ;
- Testing with arecoline or coproantigen test of dogs in the infected areas ;
- Treatment with praziquantel or an equivalent drug of all dogs in infected villages with hydatid cysts at least 3-4 times every year (and appropriate destruction of the stools, since praziquantel, that is efficient against the adult worms does not kill the infectious eggs);
- Regular use of praziquantel (or an equivalent drug) baits for the treatment of stray dogs and foxes ;

- Control of movements of food animals and dogs from the infected areas to the “clean” ones ;
- Marking and control of movements of animals from infected flocks or herds.

An efficient vaccine for intermediate hosts (*i.e.* usually sheep) has been developed in Australia and tested in limited field trials in various countries (Australia, PR China, South-America); it could represent a part of a control campaign, but its place among other more classical components of such campaigns has not been evaluated ( Lightowlers *et al*, 1996; Craig, 1997; Heath *et al*, 1999).

(2) Slaughterhouse level (CE)

Management at the slaughter house level is essential; it includes the following measures:

- Strict measures against illegal slaughter ;
- Inspection for hydatid cysts of all animals slaughtered ;
- Burial or safe destruction of cadavers or offal of food animals ;
- Training of personnels involved in the programme.

(3) Secondary production, commercial caterers, transport and retail (CE)

It does not concern meat, since infected meat (containing cysts) is not infectious to humans; however other types of food may be contaminated, especially vegetables, if dogs have access to gardens where vegetables are grown for human consumption, or if they have access to such food during their transport or retail. The following measures should thus be implemented in those areas where CE is endemic:

- Fencing of kitchen gardens (family and commercial) to prevent any access of dogs to vegetables aimed at human consumption
- Control of stray dogs, especially around outside market facilities

(4) Home-consumers (CE)

Echinococcosis is tightly linked to human behaviour towards dogs and food, cultural habits, and misunderstanding of the real risks of such behaviours and habits for their own health. Health education is a major component of any control programme and should be carefully adapted to the particularities of the various communities. Health education includes:

- Education of the public;
- Mass screening in the population: especially using ultrasound exams, it may be a part of education campaigns.
- Education of dog owners for the proper feeding and treatment at least 3-4 times per year of the dogs.

Phases of a control programme (CE)

Based on control programmes undertaken during the second half of the 20$^{th}$ century it seems that control can be divided into 4 or 5 phases ; namely the Preparatory or Planning,

Attack, Consolidation and, if appropriate, the Maintenance of Eradication Phases (Schantz *et al.* 1995).

- During the **Attack Phase**, control measures are applied non-discriminately to the entire host population at risk. Examples of this are mass dog-dosing campaigns and the introduction of restrictive regulations on dog-feeding practices.
- In the **Consolidation Phase** "at risk" areas or farms are identified through monitoring and surveillance and control measures are targeted at these only.
- The **Maintenance of Eradication Phase** can be entered once the parasite has been eradicated. Here all specific activities are disbanded and vigilance is employed, mainly through the normal meat inspection services together with border controls to prevent reintroduction. The major objective, where control is feasible – but not eradication – is to transform permanently from the costly "Attack" to the less costly "Consolidation" phase as soon as it is technically possible to do so.

A close co-operation between authorities in agriculture/veterinary services, health, education, police and law is necessary. Depending on the programme to be adopted, areas in which legislation may be needed include ; (i) meat inspection and effective disposal of offal at abattoirs and prevention of clandestine leakage of offal ; (ii) banning dogs from abattoirs and closure if necessary ; (iii) prevention of feeding raw offal to dogs including inspection of offal disposal facilities on farms or other premises where sheep are killed ; (iv) control of dogs including registration, submission of dogs for dosing and elimination of unwanted dogs, and (v) quarantine of premises with infected livestock. Any implementation of a control programme should begin with a sound evaluation of the local social, cultural and behavioural conditions.

B. Alveolar echinococcosis

(1) Environment level (AE)

Because of the sylvatic cycle of *E. multilocularis,* control approaches have been rather scarce until now. A pilot project supported by the Ministry of Agriculture of the RFA has been designed and implemented in an endemic area of Southern Gremany, using baiting of foxes with praziquantel, an anti-helminthic drug active against the adult stage of cestodes (Schelling *et al*, 1997; Tackmann *et al*, 1999). Preliminary results only are available, that suggest that the approach is feasible; however a full efficacy would need the treatment of foxes on a large area, as has been achieved for rabies vaccination, and frequently repeated campaigns of treatment, and this raises some doubts on the practical potential of such a treatment of foxes.

(2) Farm level (AE)

Incidence of AE is higher in farmers than in general population (Vuitton *et al*, 1990); AE could be considered as an occupational disease, although, until now, no regulation has been set up in any EU country; inclusion of AE among formally recognised occupational diseases in farmers is currently considered in Germany.

The extremely rare involvement of farm animals in the parasitic cycle generally precludes any specific action that could deal with farm production, at least for now. However, reports from Hokkaido, Japan, show very frequent infection of pigs, even in industrialised production settings, and cases of pig and cow infection have been reported in Switzerland;

in both countries, infected pigs had been fed with grass accessible to foxes (table 6).Current tendency to go back to traditional pig-rearing practices, as is observed in Germany and some other countries, could lead to pig infection; and even if there is some doubts that these pigs could participate in the parasitic cycle, because of the lack of fertility of the lesions in pigs, and of the well organised slaughtering in the Northern EU Member States concerned by this disease, this new trend in pig-rearing could bring a new risk.

(3) Slaughterhouse level (AE)

For the reasons cited above, slaughterhouses are not concerned by AE control, excepted if changes in food animal-rearing give opportunity to food animals to participate in the parasitic cycle.

(4) Secondary production, commercial caterers, transport and retail (AE)

All considerations, given for CE in paragraph A (3) apply to AE. The recent trend observed in fox infection by *E. multilocularis* in cities, as well as involvement of dogs and cats in the parasitic cycle could increase the risk of contamination of vegetables grown for the commercial circuit and thus dramatically extend the range of potential consumers at risk. Particular attention should thus be exerted towards protection of gardens in the endemic areas.

(5) Home-consumers (AE)

In the absence of sound data on domestic animal infection in most of EU countries, precise recommendations unfortunately cannot be given to the exposed population. However, basic recommendations concerning consumption of raw berries, fruits and vegetables collected in nature or in non-fenced kitchen gardens should be made available to all the population at risk, including populations living in cities. Information on AE is currently very scarce and, if given, not well understood by the consumers; new communication media, such as Internet, should be used to improve health education on zoonotic parasitic diseases.

## III. SURVEILLANCE AND MONITORING

A. Cystic echinococcosis

Comparable data and methods for monitoring *E. granulosus* infection in humans and animal hosts is needed.

Epidemiological monitoring and surveillance are important to evaluate the results of any control programme and its cost/efficiency ratio (Batelli, 1999; Larrieu *et al*, 1999a). Meat inspection at the abattoir usually includes observation of viscera and record of cysts. However, this is correctly notified only in those countries which have actively implemented a control programme.Data obtained at the EU level are usually non- reliable; some countries do not give any data on CE incidence in food animals; and very often data available from other sources (scientific studies) differ from those given from meat inspection system. Situation of CE in humans as a notifiable disease markedly differs between countries and the correlation between abattoir data and hospital records is generally impossible.Moreover, in many countries where both CE and AE may occur, differential diagnosis is not performed (either in food animals or in humans) and that

creates a big confusion, since the diseases and their parasite cycles are very different and thus their management options also differ. A common EU system of monitoring and surveillance, at the abattoir level, for CE, is urgently needed.

Surveillance of dog infection was particularly difficult, time-consuming and hazardous for the dogs until recently. It relied on arecoline purgation and microscopic examination for taenias. Recent development of coprotests, detecting parasite antigens using ELISA techniques, makes surveillance and monitoring of dog infection easier and should be used in a co-ordinated manner in EU Member States. Commercial coprotest kits have become available and this should allow comparisons between countries and regions and sound evaluation of control campaigns.

A co-ordination of monitoring and surveillance and control campaigns at the European level would also improve the efficiency of the campaigns at the country level. This problem occurs in border regions, where the migrations of wildlife and dogs can contribute to the failures of some campaigns when similar measures are not taken in the border countries.

B. Alveolar echinococcosis

Various aspects of the functioning of the parasite cycle in nature are unknown, including the actual role of domestic carnivores in human contamination. For these reasons, in view of the severity of human alveolar echinococcosis in humans, continuous monitoring of the epidemiological situation is an urgent need, at least until a clearer picture of the infection risk for humans and other epidemiological factors are given. In this context the following new aspects are of interest.

(1) Methods for monitoring *E. multilocularis* infection in final hosts

*General aspects and classical methods*. An accurate determination of the prevalence of *E. multilocularis* in foxes, dogs, cats and other final hosts is an essential requirement for establishing epidemiological base-line data, for surveillance and for estimating the potential infection risk of humans in endemic areas. Until recently, the most reliable technique for the diagnosis of the *E. multilocularis* infection in final hosts was the parasitological examination of the small intestine at necropsy. All the recent studies on the prevalence of *E. multilocularis* in foxes have been carried out with this method which has a sensitivity of about 85 % (Deplazes and Eckert, 1996). However, this technique cannot be applied to living animals, it is costly and requires a great deal of work.

*New techniques*. Therefore, alternative techniques have been evaluated in some laboratories (Deplazes and Eckert, 1996; Craig, 1997; Deplazes *et al*, 1999)). Extensive studies have shown that detection of serum antibodies in foxes has only a very limited value in epidemiological surveillance and monitoring (Deplazes and Eckert, 1996 ; Eckert 1997). On the other hand, it has been clearly shown (Alther, 1996, Deplazes and Eckert, 1996) that coproantigen detection by ELISA – combined in some cases with egg detection by PCR – is a realistic alternative for parasite detection. These techniques are principally ready for practical application, and they are already successfully used in research laboratories in France, Germany, Switzerland, Czech and Slovak Republics. These tests should now be made available for other laboratories and for large-scale examinations. Provided that technical and financial obstacles can be overcome, then the large-scale use of a uniform coproantigen ELISA – occasionally supplemented by egg identification by PCR – could form a new basis for monitoring and surveillance of the *E.*

*multilocularis* infection in final host populations. If such studies would be performed in representative areas a relatively simple system for continuous monitoring and surveillance could be developed.

(2) Monitoring and surveillance of disease transmission (AE)

Many basic aspects of egg transmission either directly from the final host to humans or via the environment are still unclear. The main reason is that until recently the eggs of *E. multilocularis* could not be differentiated from the eggs of *E. granulosus* and *Taenia* species as they are morphologically identical. A breakthrough has now been achieved by the development and application of a highly sensitive and specific PCR-technique for the detection of *E. multilocularis* eggs in faecal samples (Bretagne *et al*, 1993; Mathis *et al*, 1996; Monnier *et al*, 1996). As this technique is also applicable to environment samples it should now be possible to study in detail mechanisms and ways of egg dispersal and transmission.

While parts of the parasite's transmission ecology begin to appear as sketches, many questions are still unanswered. The further collection of animal host data is a prerequisite to the understanding of the epidemiological situation and, ultimately, the infection risk of humans.

(3) Monitoring of the infection in human populations (AE)

Monitoring and surveillance of the *E. multilocularis* infection in human populations is particularly difficult due to the very low prevalence.Serological tests (ELISA, Western blotting) have successfully been used in primary screening of approximately 715.000 persons in Japan (Suzuki *et al.,* 1996) and in about 20.000 persons in France (Bresson-Hadni, 1994 and personal communication). For secondary screening imaging techniques were employed, primarily ultrasound scanning (US) (Bresson-Hadni *et al.*, 1995; Suzuki *et al.,* 1996 ; Schantz *et al*, 1996). It has been clearly demonstrated that by a mass screening programme cases of human alveolar echinococcosis are detected much earlier with the important consequence that complete surgical resection of liver lesions was possible more often, (Sato *et al.*, 1993). Similar mass screening programmes (partially in combination with imaging) have also been used in several countries for epidemiological surveys (see Schantz *et al.*, 1995; Craig *et al.*, 1996) and the value of US imaging for mass screening has been assessed (see also, Craig, 1999).

A prospective registration of all AE cases in EU Member States and border countries has been initiated by a network of researchers from 9 institutions of EU Member States, together with 6 sentinel centres in border countries. This EurEchinoReg project was supported as a pilot project for one year (1998) by the European Commission-DG V. Entirely new issues regarding AE in Europe were disclosed by this limited project, such as the emergence of autochtonous human cases in regions or countries previously not recognised as endemic, the increase in the prevalence of infection in foxes within the last ten years. In addition, the extension of the geographic area concerned by fox infection, the infection of foxes living in big European cities, and the infection of dogs and cats and of food animals. Because of the severity and the financial and social cost of the disease, all these considerations should prompt EU authorities to include AE in the group of communicable diseases under monitoring and surveillance at the EU level and support epidemiological research.

Figure 1. Evolution of the geographic distribution of *Echinococcus multilocularis* infection in the final hosts, foxes, in Europe within the last decade of the XXth century

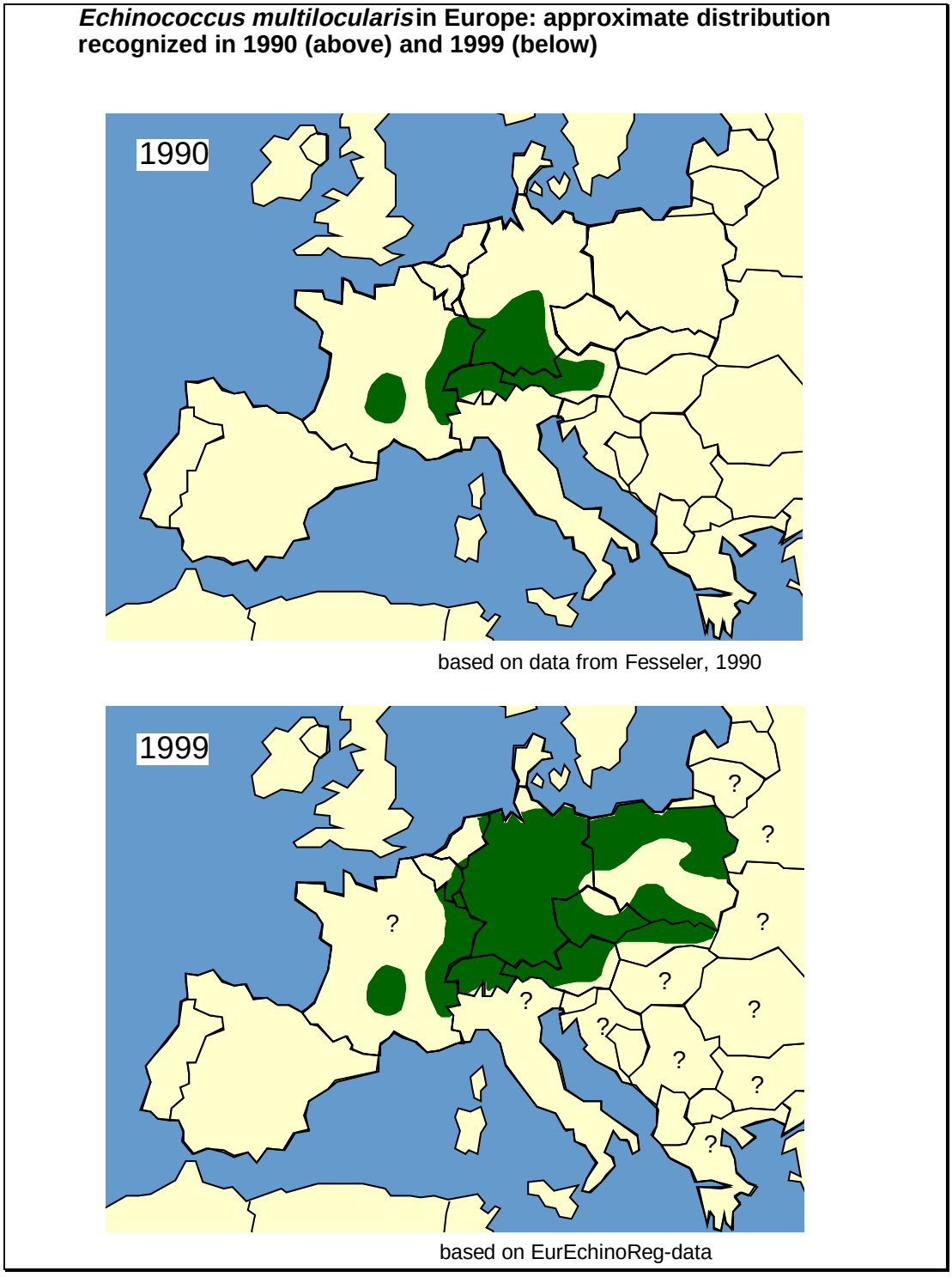
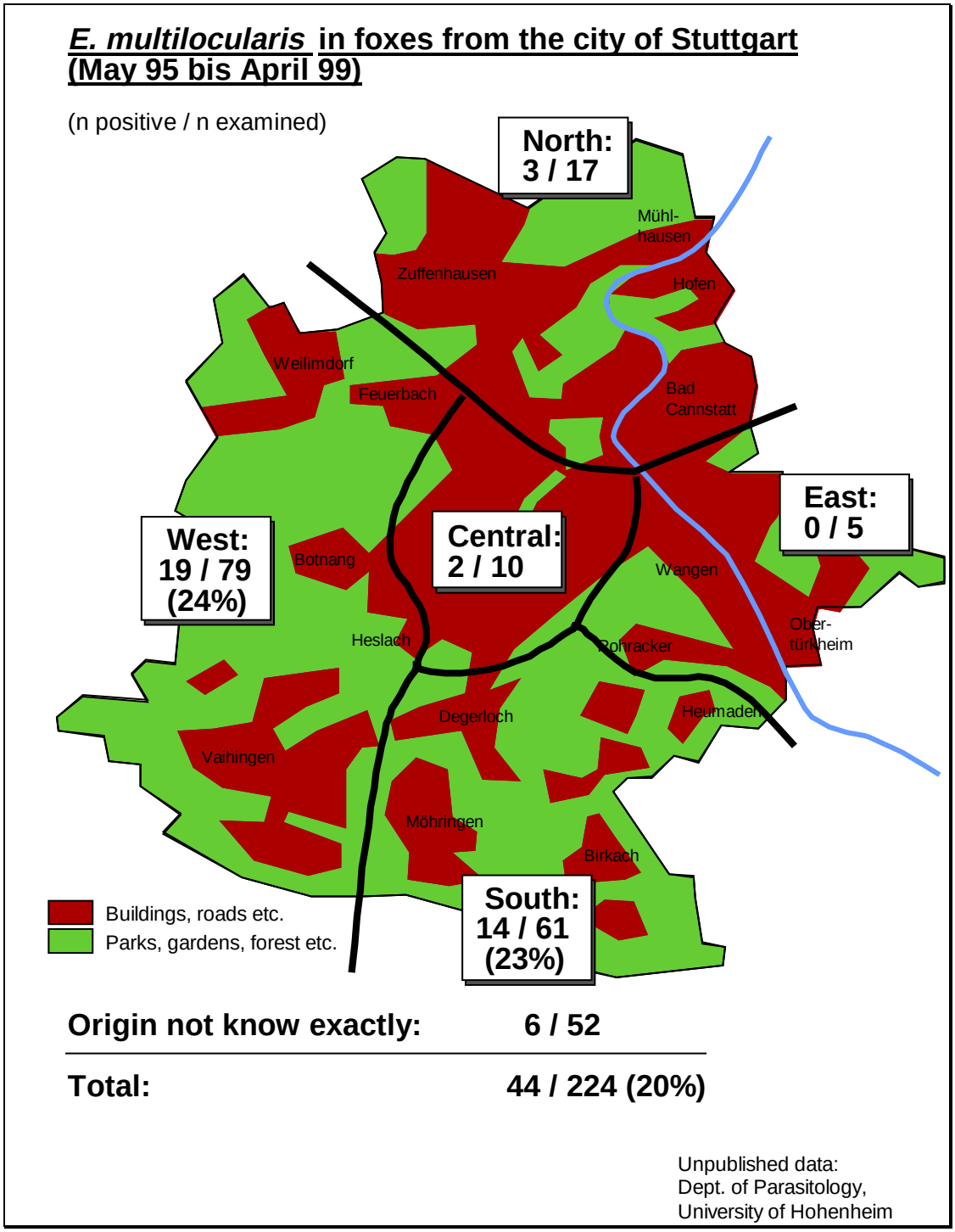


Figure 2. Prevalence of *Echinococcus multilocularis* infection in foxes, in the city of Stuttgart, Germany, in 1998-1999



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## 13.2. Annex II : Available data from Member States reports

### *13.2.1. Annex II a : Human Consumption Data*

Consumption data

**Self-sufficiency in certain agricultural products, 1996** ***(% of human consumption)***

| | EU 15 | BL EU | DK | DE | EL | ES | FR | IRL | IT | NL | AU | PT | SF | SV | UK |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Milk products** | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| - Fresh milk products (excl. cream) | 101 | 140 | 102 | 112 | 97 | 97 | 102 | 100 | 93 | 86 | 100 | 98 | 100 | 100 | 98 |
| - Whole-milk powder | 232 | 324 | 542 | 182 | 0 | 25 | 624 | 3200 | 2 | 444 | 112 | 100 | 2030 | 101 | 124 |
| - Skimmed-milk powder | 129 | 245 | 215 | 313 | 0 | 90 | 134 | 850 | 0 | 19 | 125 | 200 | 114 | 124 | 126 |
| - Concentrated milk | 137 | 204 | -: | 123 | -: | 126 | 59 | 0 | 4 | 341 | 107 | 0 | -: | 104 | 138 |
| - Cheese | 107 | 38 | 335 | 95 | 80 | 85 | 118 | 404 | 90 | 298 | 85 | 92 | 114 | 91 | 68 |
| - Butter | 109 | 154 | 171 | 81 | 43 | 93 | 98 | 811 | 77 | 258 | 102 | 127 | 111 | 108 | 95 |
| - Margarine | -: | -: | 144 | 113 | -: | 97 | 64 | 108 | -: | 126 | 100 | -: | -: | -: | 96 |
| **Eggs** | 102 | 141 | 105 | 73 | 97 | 102 | 100 | 97 | 99 | 253 | 84 | 97 | 124 | 100 | 95 |
| **Meat ( excl. offal )** | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Total meat ( incl cutting room fat), | 107 | 175 | 355 | 82 | 61 | 105 | 116 | 318 | 78 | 219 | 107 | 85 | 100 | 96 | 86 |
| of which: - Total beef/veal | 116 | 169 | 190 | 126 | 24 | 109 | 129 | 1238 | 72 | 165 | 148 | 70 | 98 | 83 | 85 |
| - Pigmeat | 106 | 212 | 449 | 77 | 55 | 107 | 102 | 161 | 67 | 251 | 100 | 81 | 101 | 102 | 71 |
| - Poultrymeat | 109 | 127 | 230 | 60 | 85 | 96 | 157 | 104 | 105 | 193 | : | 98 | : | : | 94 |
| - Sheepmeat and goatmeat | 83 | 18 | 33 | 46 | 87 | 100 | 50 | 354 | 57 | 113 | 75 | 70 | 59 | 55 | 101 |

Abbreviations:
: Not available
EU 15 Total of the Member States of the EU (1995)

BLEU Belgo-Luxembourg Economic Union
Source: Statistical Office of the European Communities (EUROSTAT)

## Consumption data

**Human consumption of certain agricultural products, 1996 (kg / head population)**

| | EU 15 | BL EU | DK | DE | EL | ES | FR | IR L | IT | NL | AU | PT | SF | SV | UK |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Milk products** | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| - Fresh products | 104,6 | 82.0 | 143,2 | 90,7 | 67,2 | 133,4 | 100,1 | 176,4 | 66,7 | 130,7 | 96,4 | 101,8 | 198,2 | 150.0 | 129,6 |
| - Cheese | 15,9 | 15,3 | 17,0 | 18,8 | 23.0 | 7,2 | 23,4 | 6,6 | 19.0 | 13,7 | 14,4 | 7,2 | 14,8 | 15,8 | 8,6 |
| -Butter ( expressed in product weight ) | 4,5 | 5,7 | 2,1 | 7,3 | 0,7 | 0,7 | 8,1 | 3,6 | 2,6 | 4,3 | 5,1 | 1,5 | 8,5 | 5,8 | - |
| - Margarine (fat) | -: | 0.0 | 7,4 | 6.0 | -: | 2,4 | 3,1 | 3,3 | -: | 9,1 | 1,5 | -: | -: | -: | 4,4 |
| **Eggs** | 12,5 | 14,5 | 14,1 | 13,7 | 10,7 | 13,5 | 15,5 | 7,5 | 10,3 | 12,2 | 13,9 | 8,2 | 11.0 | 12,5 | 10,7 |
| **Meat ( excl. Offal)** | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Total meat ( incl cutting room fat), | 87,9 | 97.0 | 100,2 | 86,8 | 81,8 | 107,6 | 99.0 | 89,4 | 84,6 | 94,6 | 95,9 | 82,3 | 64,6 | 64,5 | 71,1 |
| of which: - Total beef/veal | 18,8 | 21,5 | 18,3 | 15,3 | 22,9 | 13,1 | 26,3 | 13.0 | 23,7 | 21,2 | 20.0 | 13,9 | 19,2 | 18,6 | 14,2 |
| - Pigmeat | 41,7 | 47.0 | 64,9 | 54,8 | 24,7 | 58,4 | 36.0 | 37,9 | 35.0 | 48,7 | 57,2 | 37.0 | 33,2 | 35,4 | 23,7 |
| - Poultrymeat | 20,5 | - | - | - | 19,8 | 26,4 | 24,4 | 31,3 | 18,6 | 21,7 | 15,7 | 24,7 | 10,1 | 9,3 | 26,5 |
| - Sheepmeat and goatmeat | 3,7 | 2,1 | 1,1 | 1,1 | 14.0 | 5,8 | 5,3 | 6,6 | 1,7 | 1,5 | 1,2 | 3,7 | 0,4 | 0,8 | 6,4 |

Abbreviations:

: Not available

EU 15 Total of the Member States of the EU (1995)

BLEU Belgo-Luxembourg Economic Union

Source: Statistical Office of the European Communities (EUROSTAT)

Consumption data: German study on food consumption

| **Frequency of food intake** [1] | | | |
|---|---|---|---|
| **Meat** | **males** | **females** | **all** |
| daily | 38,1% | 24,8% | 30,4% |
| weekly | 46,2% | 50,7% | 48,8% |
| monthly | 12,8% | 18,9% | 16,3% |
| rarely, never | 2,9% | 5,6% | 4,5% |
| **n** | **4563** | **6345** | **10908** |

| **Poultry** | **males** | **females** | **all** |
|---|---|---|---|
| daily | 1,0% | 0,8% | 0,9% |
| weekly | 7,2% | 7,9% | 7,6% |
| monthly | 61,2% | 59,1% | 60,0% |
| rarely, never | 30,6% | 32,2% | 31,5% |
| **n** | **4515** | **6312** | **10827** |

| **Eggs** | **males** | **females** | **all** |
|---|---|---|---|
| daily | 17,8% | 17,4% | 17,6% |
| weekly | 40,4% | 43,6% | 42,3% |
| monthly | 33,4% | 30,9% | 32,0% |
| rarely, never | 8,4% | 8,1% | 8,2% |
| **n** | **4539** | **6320** | **10859** |

| **Milk** | **males** | **females** | **all** |
|---|---|---|---|
| daily | 48,0% | 52,3% | 50,5% |
| weekly | 19,7% | 20,6% | 20,2% |
| monthly | 13,1% | 10,7% | 11,7% |
| rarely, never | 19,1% | 16,4% | 17,6% |
| **n** | **4557** | **6326** | **10882** |

[1)] One person > 14 years per household

n Number of households involved

Consumption data: German study on food consumption (ctd.)

| **Daily intake (g), means** [2)] | | | |
|---|---|---|---|
| | **males** | **females** | **all** |
| **Meat (total)** | **91,6** | **71** | 81,3 |
| beef | 19,2 | 14,8 | 17 |
| veal | 1,6 | 1,3 | 1,45 |
| pork | 35,8 | 26,7 | 31,25 |
| game | 0,9 | 0,6 | 0,75 |
| other meat | 1,5 | 1,2 | 1,35 |
| poultry meat | 15 | 12,8 | 13,9 |
| minced meat | 14,9 | 11,2 | 13,05 |
| offal | 2,7 | 2,4 | 2,55 |
| **Eggs** | **32,6** | **27,5** | 30,05 |
| **Milk** | **196,7** | **169,6** | 183,15 |

[2)] 7-day protocols

Source:

Adolf, T., Schneider, R., Eberhardt, W., Hartmann, S., Herwig, A., Heseker, H., Hünchen, K., Kübler, W., Matiaske, B., Moch, K.J., Rosenbauer, J. Ergebnisse der Nationalen Verzehrsstudie (1985-1988) über die Lebensmittel- und Nährstoffaufnahme in der Bundesrepublik Deutschland. Verbundstudie Ernährungserhebung Risikofaktoren Analytik. VERA-SCHRIFTENREIHE. W. Kübler, H.J. Anders, W. Heeschen (Eds)

### *13.2.2. Annex II.b Prevalence Data in Animals and Food*

**Abbreviations:**

| | |
|---|---|
| **Invest.** | Number of epidemiological units investigated |
| **% Salm** | Share *Salmonella* sp. . positive samples |
| **% S.Ent**. | Share *S.enteritidis* positive samples |
| **% S.Typ**. | Share *S.typhimurium* positive samples |
| **% S.E.** | Share *S.enteritidis* of all *Salmonella* isolates |
| **% S.T**. | Share *S.typhimurium* of all *Salmonella* isolates |
| **% Campy** | Share thermophilic *Campylobacter* positive samples |
| **% C.jejuni** | Share *C.jejuni* of all *Campylobacter* isolates |
| **% C.coli** | Share C.coli of all Campylobacter isolates |
| **% *Echinococcus*** | Share *Echinococcus* positive samples |
| **% E. *granulosus*** | Share *E. granulosus* positive samples |
| **% E. *multilocularis*** | Share *E. mulitlocularis* positive samples |
| **% VTEC** | Share Verotoxin producing *E. coli* positive samples |
| **% EC O157** | Share *E. coli* O157 positive samples |

**Source of information:**

Report on trends and sources of zoonotic agents in the EU, 1996. Community Reference Laboratory for the Epidemiology of Zoonoses, Berlin, Germany:

Report on trends and sources of zoonotic agents in the EU, 1997. Community Reference Laboratory for the Epidemiology of Zoonoses, Berlin, Germany:

Report on trends and sources of zoonotic agents in the EU, 1998. Community Reference Laboratory for the Epidemiology of Zoonoses, Berlin, Germany:

## 13.2.2.1. *Salmonella*

### *Layers and products thereof*

Countries which run a monitoring programme

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Layers (flock based data)** | | | | | | | | | | | | |
| Belgium [12] | - | - | - | - | - | - | - | - | 61 | 29,5 | 21,3 | 1,6 |
| Denmark | 422[4] | 3,1 | 2,4 | 0,5 | 431[5] | 14,2 | | - | 1145[8] | 10,2 | 8,9 | 0,5 |
| Denmark | - | - | - | - | 136[6] | 19,1 | - | - | - | - | - | - |
| Denmark | - | - | - | - | 96[7] | 27,0 | - | - | - | - | - | - |
| Finland | 1900 | 0,2 | 0 | 0,1 | 1600 | 0,1 | 0 | 0,1 | 3638 | 0 | 0 | 0,03 |
| France | 1323 | 19,0 | 1,3 | 5,0 | 5392 | - | 0,6 | 0 | 1380 | - | 0,5 | 1,1 |
| Ireland | 303 | - | 0 | 0 | 128[1] | 7,0 | 0 | 0 | 521 | 2,7 | 0,8 | 0 |
| Ireland | - | - | - | - | 208[2] | 1,0 | 0 | 0 | - | - | - | - |
| Netherlands [9] | - | - | - | - | 114 | 19,3 | 3,5 | 0 | 207 | 23,7 | 15,9 | 0,5 |
| Netherlands [10] | - | - | - | - | - | - | - | - | 1631 | - | 11,1 | 0,4 |
| Netherlands [11] | - | - | - | - | 602 | 6,6 | 6,3 | 0,3 | 2828 | - | 5,8 | 0,2 |
| Sweden | 1040 | 0,6 | 0 | 0 | 813 | 0,9 | 0 | 0 | 817 | 0,6 | 0 | 0 |
| **Egg products (raw materials)** | | | | | | | | | | | | |
| Ireland | - | - | - | - | - | - | - | - | 78 | 0 | - | - |
| Netherlands | 171 | 45,0 | 36,3 | - | 157 | 48,4 | 42,0 | - | - | - | - | - |
| **Egg products (final products)** | | | | | | | | | | | | |
| England | - | - | - | - | 279 | 0 | - | - | - | - | - | - |
| Ireland | - | - | - | - | - | - | - | - | 88 | 0 | - | - |
| North. Ireland | 763 | 0,4 | - | - | 849 | 0 | - | - | 409 | 0 | - | - |
| Sweden | - | - | - | - | - | - | - | - | 144 | 0 | - | - |

[1] Caged layers
[2] Free range / perchery layers
[4] Monitoring programme: 100 cloacal swabs per herd
[5] Table egg production, serological surveillance. *Salmonella* surveillance in DK increased considerably from 1996 to 1997
[6] Rearing flocks for table egg production, serological surveillance
[7] Rearing flocks for table egg production, examined only bacteriologically until September 1997
[8] Rearing and production flocks; compulsory routine programme.
[9] Monitoring programme of RIVM
[10] Plan of Approach
[11] Samples taken for diagnostic purposes
[12] Herd based data

## Investigations in other Member States (sampling procedure not described)

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Layers (flock / farm based data)** | | | | | | | | | | | | |
| Germany [5] | 4082 | 2,7 | 1,3 | 0,7 | 15526 | 1,1 | 0,5 | 0,1 | 4305 | 1,3 | 0,7 | 0,2 |
| Germany [6] | - | - | - | - | 279 | 8,6 | 3,9 | 0,7 | - | - | - | - |
| Greece [5] | - | - | - | - | 42 | 7,1 | 7,1 | 0 | - | - | - | - |
| Italy [5] | 368 | 11,7 | 10,1 | 1,6 | - | - | - | - | 28 | 10,7 | - | 7,1 |
| **Layers (animal / sample based data)** | | | | | | | | | | | | |
| Germany [7] | - | - | - | - | 1416 | 1,5 | 0,8 | 0 | - | - | - | - |
| Greece [7] | 146 | 21,2 | 0 | 0,7 | - | - | - | - | - | - | - | - |
| Italy [8] | - | - | - | - | 1183 | 2,8 | 0,5 | 0,6 | - | - | - | - |
| Portugal [7,8] | 619 | 1,5 | 1,1 | 0,3 | 338 | 2,1 | 1,2 | 0,3 | 253 | 6,7 | 5,5 | 0,4 |
| **Eggs** | | | | | | | | | | | | |
| Austria | 617 | 1,94 | 1,78 | - | 502 | 3,39 | 1,99 | - | 403 | 1,2 | 1,2 | 0 |
| Germany | - | - | - | - | 17905[1] | 1,20 | 0,90 | 0,10 | 17598[3] | 0,41 | 0,21 | 0,02 |
| Germany | 11390[2] | 1,49 | 1,14 | 0,08 | 93243[2] | 0,50 | - | - | 5513[4] | 0,49 | 0,34 | 0,05 |
| Italy | - | - | - | - | 2539 | 3,4 | 1,2 | 0,1 | - | - | - | - |
| Spain | 427 | 0,23 | 0,23 | - | 48 | 0 | - | - | 34 | 0 | - | - |
| **Egg products (raw materials)** | | | | | | | | | | | | |
| Austria | 3 | 33,3 | 33,3 | - | 136 | 0 | - | - | 420 | 1,9 | 0,2 | 0 |
| Germany | 72 | 13,9 | 13,9 | - | 91 | 2,2 | 2,2 | - | 154 | 1 | - | - |
| Greece | - | - | - | - | - | - | - | - | 47 | 23,4 | 2,1 | 6,4 |
| **Egg products (final products)** | | | | | | | | | | | | |
| Austria | - | - | - | - | - | - | - | - | 262 | 0,4 | 0,4 | 0 |
| Germany | 501 | 0,8 | 0,4 | - | 1116 | 5,1 | 1,0 | - | 508 | 0 | - | - |
| Italy | - | - | - | - | 132 | 7,6 | 5,3 | - | - | - | - | - |

1) Routine sampling
2) Whole egg
3) Routine sampling - whole eggs
4) Suspicious samples (samples not taken during routine sampling)- whole eggs
5) Flocks
6) Farms
7) Animal
8) Sample

## Main serotypes in poultry and poultry products poultry meat retail Table SA 49, 1998

| | Broilers | | Poultry meat, retail | | Poultry meat [5] | | Layers | | Eggs | |
|---|---|---|---|---|---|---|---|---|---|---|
| | % S.E. | % S.T. | % S.E. | % S.T. | % S.E. | % S.T. | % S.E. | % S.T. | % S.E. | % S.T. |
| Austria | 48,4 | 9,4 | - | - | 19,9 | 12,7 | - | - | - | - |
| Belgium | 11,4 | 13,6 | 12,2 | 20,4 | 33,6 | 5,5 | 72,2 | 5,6 | - | - |
| Denmark | 24,7 | 18,5 | - | - | - | - | 87,2 | 5,1 | - | - |
| France | - | - | - | - | - | - | - | - | - | - |
| Finland | 0 | 0 | - | - | - | - | 0 | 100[1] | - | - |
| Germany | 57,9 | 0 | 25,0 | 7,8 | - | - | 54,4 | 12,3 | 51,4 [3]<br>70,4 [4] | 5,6 [3]<br>11,1 [4] |
| Greece | - | - | 36,4 | - | - | - | - | - | - | - |
| Ireland | 0,6 | 0 | 29,2 | 4,2 | 2,5 | 0,4 | 28,6 | 0 | - | - |
| Italy | 23,5 | 8,8 | - | - | - | - | - | 66,7[2] | - | - |
| Netherlands | 21,3 | 6,6 | - | - | - | - | - | - | - | - |
| Portugal | 78,5 | 1,0 | 40 | 40 | - | - | 82,4 | 5,9 | - | - |
| Sweden | 0 | 0 | - | - | 0 | 0 | 0 | 0 | - | - |
| Northern Ireland | 68,9 | 3,4 | - | - | - | - | 63,6 | 18,2 | - | - |
| Great Britain | 11,4 | 7,8 | - | - | - | - | 48,6 | 8,1 | - | - |

[1] One isolate
[2] Two isolates
[3] routine samples, whole eggs
[4] suspicious samples, whole eggs
[5] Slaughterhouse and retail level

## *Broiler and products thereof*

### Countries which run a monitoring programme

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Broilers (flock based data)** | | | | | | | | | | | | |
| Austria | 7412 | 5,5 | 2,7 | 0,4 | 8698 | 4,8 | 2,1 | 0,1 | 5029 | 3,4 | 1,4 | 0,2 |
| Belgium [5] | - | - | - | - | - | - | - | - | 122 | 36,1 | 4,1 | 4,9 |
| Denmark [1] | 3963 | 7,9 | 0,1 | 2,6 | 4139 | 12,9 | 2,8 | 4,2 | 4166 | 6,5 | 1,6 | 1,2 |
| Denmark [2] | 4097 | 17,4 | - | - | 4378 | 17,1 | - | - | 4985 | 11,1 | - | - |
| Finland | 2568 | 0,9 | - | - | 2951 | 0,7 | 0 | 0 | 2856 | 0,7 | 0 | 0 |
| Ireland | - | - | - | - | - | - | - | - | 1732 | 20,7 | 0,1 | 0 |
| Netherlands | - | - | - | - | 63 | 25,4 | 1,6 | 0 | 192 | 31,8 | 6,8 | 2,1 |
| Sweden [1] | 3300 | 0,12 | 0 | 0 | 3379 | 0,06 | 0 | 0 | 2935 | 0,03 | 0 | 0 |
| Sweden [3] | 3922 | 0,05 | 0 | 0 | 4235 | 0 | - | - | 4010 | 0,02 | 0 | 0 |
| **Poultry meat at slaughterhouse and cutting plants (sample based data)** | | | | | | | | | | | | |
| Belgium | - | - | - | - | 127 | 28,4 | - | - | - | - | - | - |
| Finland | - | - | - | - | 611 | 3,1 | - | - | 384 | 0,52 | - | - |
| Ireland | 1632 | 22,2 | 0 | 0,6 | 2218 | 22,6 | 0,4 | 0,2 | 2695 | 16,6 | 0,2 | 0,04 |
| Sweden | 581 | 0 | 0 | 0 | 723 | 0 | 0 | 0 | 1138 | 0 | 0 | 0 |
| **Poultry meat at retail (sample based data)** | | | | | | | | | | | | |
| Denmark | 462 | 9,5 | 1,5 | 1,1 | 404 | 5,7 | 0,2 | 0,7 | 286 | 10,6 | - | - |
| Finland | 100 | 3,0 | 0 | 0 | - | - | - | - | 114 | 0,88 | - | - |
| Germany | 3979 | 27,2 | 9,9 | 2,4 | 3062 | 22,2 | 4,9 | 2,2 | 1207 | 22,2 | 5,6 | 1,7 |
| Ireland | - | - | - | - | - | - | - | - | 51 | 47,1 | 13,7 | 1,96 |
| Netherlands | 1196 | 32,8 | 11,5 | - | 1314 | 29,2 | 9,1 | 2,0 | 1010 | 20,2 | - | - |
| North. Ireland | 314 | 12,1 | 0 | 3,5 | - | - | - | - | 31 | 0 | - | - |
| Sweden | - | - | - | - | - | 0 | - | - | [-4)] | - | - | - |
| United Kingdom | 562 | 36,8 | 22,4 | 0,7 | - | - | - | - | - | - | - | - |
| **Poultry meat products (sample based data)** | | | | | | | | | | | | |
| Denmark | - | - | - | - | - | - | - | - | 158 | 0 | - | - |

[1] monitoring before slaughter
[2] neck skin sample, flock based data
[3] neck skin sample, sample based data
[4] Poultry collected in 1998 at retail level in Sweden with unknown country of origin showed 1,2% (n=84)
[5] Herd based data

### Investigations in other Member States (sampling procedure not described)

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Broilers (flock / herd based data)** | | | | | | | | | | | | |
| Germany | - | - | - | - | 129[1] | 7,0 | 3,1 | 1,6 | - | - | - | - |
| Germany | 3119 | 4,2 | 2,4 | 0 | 691[2] | 5,8 | 3,0 | 0,4 | 455 | 4,2 | 2,4 | 0 |
| Italy | - | - | - | - | 754 | 1,1 | - | 0,4 | 1093 | 3,1 | 0,7 | 0,3 |
| **Broilers (animal / sample based data)** | | | | | | | | | | | | |
| Greece | - | - | - | - | 55 | 23,6 | 7,3 | 0 | - | - | - | - |
| Italy | - | - | - | - | 9845 | 0,8 | 0,2 | 0,2 | - | - | - | - |
| Portugal | 2636 | 2,9 | 1,9 | 0,1 | 2387 | 5,2 | 3,8 | 0,1 | 1954 | 10,0 | 7,8 | 0,1 |
| **Poultry meat sampled at slaughterhouse** | | | | | | | | | | | | |
| Austria [3] | 3485 | 20,9 | 7,5 | 2,4 | 80 | 62,5 | 42,5 | - | 1207 | 22,2 | 5,6 | 1,7 |
| Austria | - | - | - | - | - | - | - | - | 124 | 2,4 | 1,6 | 0 |
| Italy | - | - | - | - | 126 | 8,7 | 3,2 | 1,6 | - | - | - | - |
| **Poultry meat sampled at retail** | | | | | | | | | | | | |
| Austria | - | - | - | - | 2779 | 9,1 | 4,3 | 0,5 | 430 | 16,4 | 5,2 | 0,7 |
| Austria [4] | - | - | - | - | - | - | - | - | 1931 | 17,5 | 6,3 | 2,1 |
| Italy | - | - | - | - | 104 | 14,4 | 1, 9 | - | - | - | - | - |
| Greece | - | - | - | - | 69 | 0 | - | - | 198 | 5,6 | 2,0 | - |
| Portugal | - | - | - | - | 34 | 23,5 | 5,9 | 2,9 | 73 | 34,3 | 13,7 | 13,7 |
| **Poultry meat products** | | | | | | | | | | | | |
| Germany | | - | - | - | - | - | - | - | 569 | 4,2 | 0,7 | 0,18 |

[1] Farms
[2] Flocks
[3] Skin samples of suspected flocks
[4] All samples

## *Turkeys and products thereof*

### Countries which run a monitoring programme

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Turkeys (flock based data)** | | | | | | | | | | | | |
| Denmark | - | - | - | - | 355 | 11,8 | 0,9 | 1,7 | 366 | 9,3 | 0,5 | 0,3 |
| Finland | 73 | 0 | 0 | 0 | 85 | 2,4 | 2,4 | 0 | 252 | 0 | 0 | 0 |
| Sweden | 62 | 0 | 0 | 0 | 59 | - | - | - | - | 0 | 0 | 0 |
| **Turkey meat (at retail)** | | | | | | | | | | | | |
| Denmark | - | - | - | - | 257 | 7,0 | - | - | 525 | 4,0 | 0 | 0,2 |
| Germany | - | - | - | - | 992 | 16,8 | 3,0 | 1,4 | 651 | 11,8 | 0,5 | 3,5 |
| **Turkey meat products** | | | | | | | | | | | | |
| Denmark | - | - | - | - | - | - | - | - | 72 | 0 | - | - |

### Investigations in other Member States (sampling procedure not described)

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Turkeys (flock / farm based data)** | | | | | | | | | | | | |
| Austria | 1165 | - | - | - | 1813 | 4,9 | 0,1 | 0,2 | 1777 | 6,1 | 0,2 | 4,1 |
| Germany | 20 | 15,0 | 5,0 | 10,0 | 266 | 5,6 | 1,1 | 2,2 | 1147 | 4,4 | 0,2 | 0,4 |
| France | - | - | - | - | 2376 | - | 0,06 | 1,5 | 608 | - | 0 | 0,3 |
| Italy | - | - | - | - | - | - | - | - | 36 | 2,8 | - | 2,8 |
| **Turkeys (animal / sample based data)** | | | | | | | | | | | | |
| Ireland | 82 | 1,2 | - | - | 54 | 9,3 | - | - | 201 | 5,5 | 0 | 0 |
| Germany | 2279 | 3,9 | 0,4 | - | 2647 | 2,2 | 0,7 | 0,6 | 1276 | 4,1 | 0,2 | 0,4 |
| Italy | - | - | - | - | 3039 | - | 0,3 | 0,03 | 9096 | 0,2 | - | 0 |
| Portugal | 1568 | 2,4 | 0,1 | 0,2 | 807 | 1,5 | 0,3 | 0,3 | 810 | 2,1 | 0,2 | 0,2 |

## *Pigs and pork*

### Countries which run a monitoring programme

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. | Invest. | %Salm | %S.Ent. | %S.Typ. |
| **Pigs (herd / farm based data) - serological tests** | | | | | | | | | | | | |
| Denmark [2] | 17087 | 5,0 | - | - | 16268 | 5,5 | - | - | 16756 | 3,7 | - | - |
| **Pigs (herd / farm based data) - faecal samples** | | | | | | | | | | | | |
| Ireland [5] | - | - | - | - | - | - | - | - | 83 | 54,2 | - | 36 |
| Netherlands [6] | - | - | - | - | - | - | - | - | 41 | 34,2 | - | 20 |
| **Pigs (sample based data) - lymph nodes, carcass swabs** | | | | | | | | | | | | |
| Belgium [7] | - | - | - | - | 221 | 9,95 | - | - | 228 | 8,77 | - | - |
| Finland [4] | - | - | - | - | 6374 | 0,20 | - | - | 6317 | 0,13 | - | 0,05 |
| Finland [7] | - | - | - | - | - | - | - | - | 6265 | 0,05 | - | 0,05 |
| Sweden [4] | 4708 | 0,08 | 0 | 0,08 | 5996 | 0,13 | - | 0,05 | 6988 | 0,14 | 0,03 | 0,10 |
| Sweden [7] | 4696 | 0 | 0 | 0 | 5993 | 0,02 | 0 | 0 | 6998 | 0,04 | - | - |
| **Pork at slaughterhouse and cutting plants** | | | | | | | | | | | | |
| Denmark [8] | 18159 | 1,30 | 0 | 0,80 | 18510 | 1,10 | 0 | 0,70 | 17846 | 1,10 | 0 | 0,60 |
| Denmark [9] | 9979 | 3,00 | - | 2,09 | 9489 | 3,70 | 0,01 | 2,60 | 9091 | 2,69 | - | 1,59 |
| Finland [10] | - | - | - | - | 3741 | 0,03 | - | - | 4427 | 0,05 | - | 0,02 |
| Sweden[10,11] | 5510 | 0 | 0 | 0 | 5909 | 0,03 | 0,02 | 0,02 | 4595 | 0,04 | 0 | 0 |
| **Pork at retail** | | | | | | | | | | | | |
| Denmark | 3371 | 1,81 | - | 1,22 | 2235 | 1,40 | 0,04 | 0,90 | 2660 | 0,71 | 0,04 | 0,19 |
| Germany | 2389 | 7,03 | 0,17 | 4,27 | 2036 | 6,83 | - | 4,76 | 1804 | 3,99 | 0,11 | 2,38 |
| Sweden | 119 | 0 | - | - | 97 | 0,00 | - | - | 1142 | 0,09 | - | - |
| Northern Ireland | - | - | - | - | 104 | 0,96 | - | - | - | - | - | - |
| Engl.&Wales | - | - | - | - | 367 | 0,00 | - | - | - | - | - | - |

1) Clinical outbreaks
2) Serological monitoring programme
3) Serotype distribution
4) Lymph node sample
5) Targeted at mainly category 3 herds
6) Surveillance project IW&V-RIVM
7) Swabs of carcasses
8) Cuts of meat
9) Offal
10) Cutting plants
11) Both pork and beef, approximately 62% are estimated to be sampled from pork scrapings

### Investigations in other Member States (sampling procedure not described)

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ |
| **Pigs (farms / herd based data)** | | | | | | | | | | | | |
| Germany | 223 | 9,87 | 0,90 | 4,93 | 1325 | 8,23 | 0,08 | 6,57 | 6726 | 3,54 | 0,24 | 2,72 |
| Italy | 673 | 3,42 | - | 2,53 | - | - | - | - | 442 | 9,05 | - | 4,07 |
| Netherlands | - | - | - | - | - | - | - | - | 999 | - | - | 14,31 |
| **Pigs (animal / sample based data)** | | | | | | | | | | | | |
| Austria | 817 | - | - | - | 811 | 0,12 | - | - | 724 | - | - | - |
| Germany | 17656 | 1,39 | 0,02 | 0,95 | 11800 | 1,27 | - | - | 9442 | 1,26 | 0,04 | 0,77 |
| Germany | 7624 | 7,76 | 0,09 | 5,65 | 25717 | 4,58 | 0,05 | 2,96 | 10208 | 2,05 | 0,01 | 1,59 |
| Italy | - | - | - | - | 2489 | 8,32 | - | 1,0 | 274 | 9,12 | 0,36 | 4,74 |
| Netherlands | - | - | - | - | 886[2] | 25,17 | - | 21,56 | 2941[3] | 10,8 | 0 | 4,9 |
| Portugal | 553 | 0,90 | 0,18 | 0,72 | 254 | 4,72 | 0,39 | 2,36 | 503 | 4,97 | 0,60 | 1,19 |
| **Pork** | | | | | | | | | | | | |
| Austria | 94 | 2,13 | 1,06 | - | - | - | - | - | 22 | 0 | - | - |
| Germany [1] | - | - | - | - | - | - | - | - | 1595 | 2,76 | 0,06 | 1,50 |
| Italy | - | - | - | - | 828 | 7,00 | 0,36 | 2,05 | - | - | - | - |
| Netherlands | - | - | - | - | 105 | 7,62 | 1,90 | 2,86 | - | - | - | - |

1) Suspicious samples, follow-up samples, hygiene samples
2) Diagnostic samples
3) Surveillance project

## *Cattle and products thereof*

### Countries which run a monitoring programme

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ |
| **Cattle (herd based data) - faecal samples** | | | | | | | | | | | | |
| Denmark [1] | - | - | - | - | 256 | 0,8 | 0 | 0,40 | 286 | 0 | - | - |
| Netherlands | - | - | - | - | - | - | - | - | 411 | 2,0 | - | 0,7 |
| Netherlands | - | - | - | - | 84 | 0 | - | - | 263 | 2,7 | - | - |
| Netherlands | - | - | - | - | 114 | 2,6 | - | - | 148 | 0,7 | - | - |
| **Cattle (herd based data) - lymph nodes and carcass swabs** | | | | | | | | | | | | |
| Finland [2] | - | - | - | - | - | - | - | - | 3189 | 0,31 | - | - |
| Finland [3] | - | - | - | - | 3116 | 0,10 | - | - | 3227 | 0,22 | - | - |
| Sweden [2] | 3571 | 0,03 | 0 | 0,03 | 3989 | 0,08 | 0 | 0,03 | 3990 | 0,05 | 0 | 0,03 |
| Sweden [3] | 3591 | 0 | 0 | 0 | 3976 | 0 | 0 | 0 | 4046 | 0,02 | 0 | 0,02 |
| **Beef sampled at slaughterhouse and cutting plants** | | | | | | | | | | | | |
| Denmark [4] | 1927 | 0,52 | 0 | 0,21 | 2149 | 0,40 | 0 | 0,10 | 2145 | 0,28 | - | - |
| Denmark [5] | 1166 | 1,37 | 0 | 0,09 | 1667 | 1,40 | 0,08 | 0,20 | 1026 | 1,17 | 00,1 | 0,19 |
| Finland [6] | - | - | - | - | 3189 | 0,16 | - | - | 3016 | 0,07 | - | 0,03 |
| Sweden[6,7] | 5510 | 0 | 0 | 0 | 5909 | 0,03 | 0,02 | 0,02 | 4595 | 0,04 | 0 | 0 |
| **Beef sampled at retail** | | | | | | | | | | | | |
| Belgium | - | - | - | - | 53 | 3,8 | - | - | 51 | 3,9 | - | - |
| Denmark | 3306 | 0,91 | - | 0,48 | 2751 | 0,50 | - | 0,20 | 2600 | 0,50 | 0,12 | 0,04 |
| Sweden | 216 | 0 | - | - | 385 | 0 | - | - | 1846 | 0 | - | - |
| Northern Ireland | 21 | - | - | - | 320 | 1,9 | - | - | - | - | - | - |

[1] Monitoring programme
[2] Lymph nodes
[3] Swabs of carcasses
[4] Cuts of meat
[5] Offal
[6] Cutting plants
[7] Approximately 40 % is estimated to be scrapings collected from beef

### Investigations in other Member States (sampling procedure not described)

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ |
| **Cattle (herd / farm based data)** | | | | | | | | | | | | |
| Germany | 1252 | 11,7 | 0,4 | 7,0 | 4441 | 4,77 | 0,27 | 3,17 | 8438 | 3,9 | 0,1 | 2,1 |
| Italy | - | - | - | - | - | - | - | - | 388 | 6,4 | 0,3 | 5,7 |
| Netherlands | - | - | - | - | - | - | - | - | 1676 | - | - | 2,6 |
| **Cattle (animal / sample based data)** | | | | | | | | | | | | |
| Austria | 4633 | 0,11 | - | - | 5407 | 0,09 | - | - | 5507 | 0,09 | 0,02 | - |
| Germany | 41420 | 0,49 | 0,04 | 0,25 | 23302 | 0,69 | 0,03 | 0,52 | 19095 | 0,3 | - | 0,1 |
| Germany | 88346 | 2,8 | 0,01 | 2,4 | 182931 | 3,48 | 0,07 | 2,87 | 116214 | 2,8 | 0,2 | 2,4 |
| Italy | 432 | 0,5 | - | 0,5 | 1098 | 9,3 | 0,5 | 3,3 | 1851 | 1,4 | 0,1 | 1,1 |
| Luxembourg | 298 | 2,7 | - | 2,0 | - | - | - | - | 1073 | 15,6 | - | 1,1 |
| Portugal | 184 | 3,3 | - | 2,7 | 327 | 1,5 | 0,3 | 0,6 | 119 | 9,2 | - | 5,0 |
| Netherlands | - | - | - | - | 2198 | 11,65 | 0 | 2,5 | 4305 | - | - | 1,5 |
| **Beef** | | | | | | | | | | | | |
| Austria | - | - | - | - | 52 | 0 | - | - | 15 | 0 | - | - |
| Germany [1] | 16083 | 0,69 | 0,33 | 0,12 | 1088 | 1,3 | 0,09 | 0,3 | 1032 | 0,8 | 0,1 | 0,3 |
| Germany [2] | 764 | 2,1 | 0,39 | 0,65 | 55 | 1,8 | 1,8 | - | 317 | 0,3 | 0 | 0,3 |
| Italy | - | - | - | - | 1903 | 5,5 | - | - | - | - | - | - |
| Netherlands | - | - | - | - | 109 | 0 | - | - | - | - | - | - |

[1] Beef
[2] Veal

## Main serotypes in livestock and food (other than poultry) 1998

| | Pigs | | Pork | | Cattle | | Beef | |
|---|---|---|---|---|---|---|---|---|
| | % S.E. | % S.T. | % S.E. | % S.T. | % S.E. | % S.T. | % S.E. | % S.T. |
| Austria | - | - | - | - | 20 | 0 | - | - |
| Belgium | 0,95 | 47,7 | 0 | 24,1 | 1,3 | 70,9 | - | - |
| Denmark | 0,9 [1] | 78,6 [1] | 0 [2] | 54,0 [2] | 1,2 [9] | 24,1 [9] | 0 [2] | 10,5 [2] |
| | - | - | 5,3 [3] | 26,3 [3] | - | - | 23,1 [3] | 7,7 [3] |
| France | - | - | - | - | 1,1 | 60,2 | - | - |
| Germany | 3,4 [4] | 61,3 [4] | 2,8 [7] | 59,7 [7] | 10,5 [4] | 45,6 [4] | 13 [10] | 37,5 [10] |
| | 6,7 [5] | 76,9 [5] | 2,3 [8] | 54,6 [8] | 3,7 [5] | 53,4 [5] | - | - |
| | 0,5 [6] | 77,5 [6] | | | 6,1 [6] | 84,5 [6] | - | - |
| Ireland | 0 | 66,7 | - | 100 [4] | - | - | - | - |
| Italy | - | 45 [5] | - | - | 4,0 [5] | 88,0 [5] | - | - |
| | 4 [6] | 52 [6] | - | - | 7,7 [6] | 80,8 [6] | - | - |
| Luxembourg | - | - | - | - | 0 | 7,2 | - | - |
| Netherlands | 0 | 57,1 | - | - | 0 | 56,9 | - | - |
| Portugal | 12 | 24 | - | - | - | 54,5 | - | - |
| Northern Ireland | 0 | 71,4 | 0 | 0 | 0,4 | 46,9 | - | - |
| Great Britain | 0 | 58,5 | - | - | 2,3 | 49,7 | - | - |

[1] isolates obtained from sampling in slaughter-pig herds placed in Level 2 or 3
[2] representative meat samples from the surveillance programme in slaughterhouses
[3] calculated from findings at retail level
[4] samples taken at post mortem health inspection
[5] herd based data
[6] animal based data
[7] routine samples at retail
[8] suspicious samples, follow up samples, hygiene samples
[9] clinical outbreaks
[10] beef

## *Milk and milk products*

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ |
| **Raw milk** | | | | | | | | | | | | |
| Austria | 123 | 0,81 | - | - | 313 | 0 | - | - | 259 | 0 | - | - |
| Germany | 3485 | 0 | - | - | 22119 | 0,01 | - | - | 4674 | 0,06 | - | 0,02 |
| Greece | - | - | - | - | - | - | - | - | 105 | 0 | - | - |
| Italy | - | - | - | - | 4005 | 0,15 | - | - | - | - | - | - |
| North.Ireland | 50 | 0 | - | - | 50 | 0 | - | - | - | - | - | - |
| **Heat treated milk** | | | | | | | | | | | | |
| Austria | 48 | 0 | - | - | 68 | 0 | - | - | 109 | 0 | - | - |
| Germany | 4084 | 0 | - | - | 2033 | 0 | - | - | 1386 | 0 | - | - |
| Ireland | - | - | - | - | - | - | - | - | 10124 | 0 | - | - |
| Italy | - | - | - | - | 122 | - | - | - | - | - | - | - |
| Portugal | 24 | 0 | - | - | 2 | - | - | - | 129 | 0 | - | - |
| North. Ireland | 600 | 0 | - | - | 300 | 0 | - | - | 500 | 0 | - | - |
| **Dairy products** | | | | | | | | | | | | |
| Austria | 798 | 0,25 | 0,25 | - | 936 | 0 | - | - | 1008 | 1,1 | - | - |
| Germany [1] | 11914 | 0,04 | 0,02 | 0,02 | 14789 | 0,01 | 0 | 0 | 10361 | 0,02 | 0,02 | 0 |
| Germany [2] | - | - | - | - | - | - | - | - | 301 | 0 | - | - |
| Germany [3] | - | - | - | - | - | - | - | - | 935 | 0,43 | 0,32 | 0,11 |
| Greece | - | - | - | - | 302 | 0 | - | - | 30 | 0 | - | - |
| Ireland | 4502 | 0,11 | - | - | - | - | - | - | 370 | 0 | - | - |
| Italy | - | - | - | - | 928 | 0,65 | 0,65 | - | - | - | - | - |
| Portugal | 237 | 0 | - | - | 310 | 0 | - | - | 264 | 0,4 | - | - |
| Spain | 19 | 0 | - | - | 24 | 0 | - | - | 84 | 0 | - | - |
| Sweden | - | - | - | - | - | - | - | - | 143 | 0 | - | - |
| North.Ireland | 165 | 0 | - | - | 143 | 0 | - | - | 80 | 1,3 | - | 1,25 |

1) Without raw milk 2) Raw milk products 3) Suspicious samples

# *Other animal derived foodstuff*

| | 1996 | | | | 1997 | | | | 1998 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ | Invest. | %Salm | %S.Ent. | %S.Typ |
| **Minced meat** | | | | | | | | | | | | |
| Austria | 163 | 0,61 | 0 | 0,61 | 58 | 0 | - | - | 70 | 0 | - | - |
| Belgium | - | - | - | - | 589 | 4,41 | - | - | - | - | - | - |
| Germany | - | - | - | - | - | - | - | - | 3389 | 3,6 | 0,1 | 1,9 |
| Germany [1] | - | - | - | - | 870 | 7,59 | - | 2,30 | 1479 | 2,8 | - | 1,6 |
| Italy | - | - | - | - | 470 | 5,53 | 0,21 | 0,85 | 6945 [3] | 5,85 | 0,06 | 0,07 |
| Netherlands [2] | 596 | 7,89 | - | - | 561 | 4,99 | 0 ,36 | 2,32 | - | - | - | - |
| **Meat preparations** | | | | | | | | | | | | |
| Austria | 79 | 3,80 | 3,80 | 0 | 58 | 0 | - | - | 80 | 0 | - | - |
| Germany | 10944 | 5,21 | 0,13 | 2,81 | 12162 | 4,04 | 0,06 | 2,30 | 5123 | 3,08 | - | 1,74 |
| Germany | - | - | - | - | - | - | - | - | 824 | 1,58 | - | - |
| Greece | - | - | - | - | 26 | 0 | - | - | 106 | 0 | - | - |
| Italy | - | - | - | - | 1389 | 6,70 | 1,01 | 1,87 | 6945 [3] | 5,85 | 0,06 | 0,07 |
| Portugal | 102 | 11,76 | 3,92 | - | 31 | 3,23 | - | - | 74 | 0 | - | - |
| **Meat products** | | | | | | | | | | | | |
| Austria | 237 | 3,80 | 2,95 | - | 779 | 1,67 | 0,90 | - | 1311 | 2,2 | 1,4 | 0,1 |
| Denmark [4] | 8411 | 0,01 | - | - | 5144 | 0,06 | 0,04 | - | 2311 | 0,09 | 0,04 | - |
| Denmark [5] | 3342 | 0,03 | - | - | 1837 | 0,05 | - | - | 745 | 0,13 | 0 | 0,13 |
| Germany [6] | 14549 | 1,33 | 0,08 | 0,53 | 11352 | 1,73 | 0,10 | 0,79 | 2840 | 0,60 | 0,04 | 0,11 |
| Germany [7] | - | - | - | - | - | - | - | - | 1238 | 0,48 | 0,08 | 0,16 |
| Germany [8] | - | - | - | - | - | - | - | - | 3796 | 1,37 | - | 0,79 |
| Germany [9] | - | - | - | - | - | - | - | - | 2031 | 0,89 | 0,20 | 0,59 |
| Greece | - | - | - | - | 208 | | - | - | 324 | 1,85 | 0,62 | - |
| Ireland | 1098 | 2,09 | 0,09 | 0,36 | 1309 | 2,98 | - | - | 360 | 5,28 | - | - |
| Italy | - | - | - | - | 704 | 17,33 | 0,43 | 5,26 | 6945 [3] | 5,85 | 0,06 | 0,07 |
| Luxembourg | - | - | - | - | 53 | 3,77 | 1,89 | 1,89 | - | - | - | - |
| Portugal | 292 | 18,15 | 4,45 | 3,08 | 119 | 0 | - | - | 152 | 1,32 | - | - |
| Spain | - | - | - | - | 30 | 0 | - | - | - | - | - | - |
| Sweden [10] | - | - | - | - | - | - | - | - | 779 | 0,13 | 0,13 | 0 |
| Sweden [10] | 288 | 0 | - | - | - | - | - | - | 785 | - | 0,13 | - |
| Netherlands | 531 | 1,88 | - | - | - | - | - | - | - | - | - | - |
| North.Ireland | - | - | - | - | - | - | - | - | 44 | 0 | - | - |
| North.Ireland | - | - | - | - | - | - | - | - | 40 | 0 | - | - |
| England | - | - | - | - | 662 | 0,15 | - | - | - | - | - | - |
| Engl.&Wales | 465 | 0 | - | - | 455 | 0,22 | - | 0,22 | - | - | - | - |

1 Suspicious samples
2 After pos. routine-sample
3 Meat preparations, meat products and minced meat
4 Pork
5 Beef
6 Heat treated, routine sampling
7 Heat treated, suspicious samples
8 Treated other than heat, routine sampling
9 Treated other than heat, suspicious samples
10 Countries of origin unknownn

## 13.2.2.2. Campylobacter

### *Broilers and products thereof*

Countries which run a monitoring programme

| Country | 1996 Invest. | 1996 % Campy | 1997 Invest. | 1997 % Campy | 1998 Invest. | 1998 % Campy |
|---|---|---|---|---|---|---|
| **Broilers (flock based data)** | | | | | | |
| Denmark | 1030 | 35,3 | 1037 | 37,0 | 5943 | 47,1 |
| Sweden | 3398 | 9,3 | 3641 | 9,8 | 3561 | 9,1 |
| Netherlands | - | - | - | - | 189 | 30,7 |
| **Poultry meat (at slaughterhouse)** | | | | | | |
| Denmark | 274 | 38,8 | - | - | - | - |
| **Poultry meat (at retail)** | | | | | | |
| Denmark | - | - | 676 | 33,0 | 819 | 28,8 |
| Finland | 100 | 14,0 | 114 | 10,5 | 114 | 11,4 |
| Sweden | - | - | - | - | 83 | 4,5 |
| Netherlands | 1165 | 36,6 | 1314 | 31,7 | 1009 | 26,9 |
| **Poultry meat products** | | | | | | |
| Denmark | 303 | 0,7 | - | - | - | - |

Investigations in other Member States (sampling procedure not described)

| Country | 1996 Invest. | 1996 % Campy | 1997 Invest. | 1997 % Campy | 1998 Invest. | 1998 % Campy |
|---|---|---|---|---|---|---|
| **Broilers (flock based data)** | | | | | | |
| Germany | - | - | 17 | 47,1 | 102 | 0 |
| **Broilers (animal / sample based data)** | | | | | | |
| Germany | 564 | 12,6 | 677 | 2,5 | 994 | 0,3 |
| Italy | 21 | - | 71 | 9,9 | 190 | 0 |
| Portugal | - | - | 3 | 0 | 2 | 1 pos. |
| Netherlands | - | - | 47 | 44,7 | - | |
| **Poultry meat (at slaughterhouse)** | | | | | | |
| Germany | 420 | 6,0 | 812 | 20,1 | 675 | 17,2 |
| Ireland | 2 | 0 | - | - | - | - |
| Spain | 7 | 3 pos. | 8 | 2 pos. | - | - |
| **Poultry meat (at retail)** | | | | | | |
| Austria | - | - | - | - | 21 | 33,3 |
| Ireland | - | - | - | - | 19 | 5,3 |
| Italy | - | - | 68 | 1,5 | 16 | 0 |
| Spain | - | - | - | - | 5 | 3 pos |
| **Poultry meat products** | | | | | | |
| Germany | - | - | 40 | 2,5 | 24 | 0 |

Share *C.jejuni* of all *Campylobacter* isolates

| Country | 1996 Isolates | 1996 % C.jejuni | 1997 Isolates | 1997 % C.jejuni | 1998 Isolates | 1998 % C.jejuni |
|---|---|---|---|---|---|---|
| **Broiler** | | | | | | |
| Denmark | 364 | 84,9 | 384 | 76,0 | 2799 | 85,1 |
| Germany | 71 | 21,1 | 17 | 41,2 | - | - |
| Sweden | 316 | 98,1 | - | - | - | - |
| **Poultry meat** | | | | | | |
| Austria | - | - | - | - | 7 | 40 |
| Belgium | - | - | 97 | 44,3 | 150 | 78,8 |
| Finland | - | - | - | - | 13 | 92,3 |
| Germany | 25 | 44,0 | 163 | 74,8 | 116 | 67,2 |
| Luxembourg | - | - | - | - | 120 | 85,8 |
| Portugal | - | - | 84 | 48,8 | - | - |

### *Cattle and products thereof*

## Countries which run a monitoring programme

| Country | 1996 Invest. | % Campy | 1997 Invest. | % Campy | 1998 Invest. | % Campy |
|---|---|---|---|---|---|---|
| **Cattle (herd based data)** | | | | | | |
| Denmark | 93 | 43,0 | 96 | 51,0 | 85 | 47,1 |
| Netherlands | - | - | - | - | 192 | 48,4 |

## Investigations in other Member States (sampling procedure not described)

| Country | 1996 Invest. | % Campy | 1997 Invest. | % Campy | 1998 Invest. | % Campy |
|---|---|---|---|---|---|---|
| **Cattle (herd based data)** | | | | | | |
| Germany | 56 | 0 | 287 | 0,4 | 4617 | 0,5 |
| **Cattle (animal based data)** | | | | | | |
| Finland [1] | 481 | 0 | 367 | 0 | 31 | 0 |
| Germany | 10471 | 10,6 | 10051 | 10,2 | 9808 | 9,6 |
| Italy | 51 | 0 | 344 | 8,4 | 347 | 1,4 |
| TheNetherlands | - | - | 141 | 1,4 | - | - |
| Portugal | 114 | 0,9 | 91 | 1,1 | 100 | 0 |
| **Beef** | | | | | | |
| Austria | 4 | - | 4 | - | 2 | 0 |
| Belgium | 62 | 9,7 | 31 | - | - | - |
| Denmark | 198 | 2,0 | 516 | 0,7 | - | - |
| Sweden | - | - | 100 | 0 | - | - |
| North.Ireland | - | - | 320 | 15,0 | - | - |
| Italy | - | - | - | - | 104 | - |

## *Milk and milk products*

| Country | 1996 Invest. | % Campy | 1997 Invest. | % Campy | 1998 Invest. | % Campy |
|---|---|---|---|---|---|---|
| **Milk, raw** | | | | | | |
| Austria | 36 | 2,8 | 73 | 0 | 95 | 0 |
| Germany | 510 | 0 | 799 | 0,4 | 1559 | 0 |
| Italy | - | - | 19 | - | - | - |
| **Heat treated milk** | | | | | | |
| Germany | 42 | 0 | 35 | 0 | 29 | 0 |
| **Dairy products** | | | | | | |
| Austria | - | - | 49 | 0 | 98 | 0 |
| Germany | 122 | 0 | 89 | 1,1 | 59 | 0 |
| Ireland | - | - | - | - | 17 | 0 |
| Spain | - | - | - | - | 40 | 0 |

## Share C.jejuni of all Campylobacter isolates

| Country | 1996 Isolates | % C.jejuni | 1997 Isolates | % C.jejuni | 1998 Isolates | % C.jejuni |
|---|---|---|---|---|---|---|
| **Cattle** | | | | | | |
| Denmark | 93 | 92,5 | 49 | 96,0 | 40 | 90,0 |
| Germany | - | - | - | - | 25 | 76,0 |
| **Beef** | | | | | | |
| North.Ireland | - | - | 48 | 60,4 | - | - |

## *Pigs and products thereof*

### Countries which run a monitoring programme

| Country | 1996 Invest. | 1996 % Campy | 1997 Invest. | 1997 % Campy | 1998 Invest. | 1998 % Campy |
|---|---|---|---|---|---|---|
| **Pigs (herd based data)** | | | | | | |
| Denmark | 310 | 48,4 | 319 | 59,0 | 318 | 68,6 |
| Netherlands | - | - | - | - | 38 | 97,4 |

### Investigations in other Member States (sampling procedure not described)

| Country | 1996 Invest. | 1996 % Campy | 1997 Invest. | 1997 % Campy | 1998 Invest. | 1998 % Campy |
|---|---|---|---|---|---|---|
| **Pigs (herd based data)** | | | | | | |
| Germany | - | - | 196 | 0,5 | 1440 | 0,6 |
| Italy | - | - | 61 | 13,1 | - | - |
| **Pigs (animal based data)** | | | | | | |
| Finland | - | - | - | - | 54 | 37,0 |
| Germany | 1893 | 5,3 | 1629 | 8,0 | 1873 | 5,5 |
| Italy | - | - | - | - | 56 | 1,8 |
| Portugal | - | - | 18 | 28 | - | - |
| **Pork** | | | | | | |
| Austria | - | - | - | - | 1 | 0 |
| Belgium | 49 | 2,0 | - | - | - | - |
| Denmark | 177 | 1,7 | 433 | 1,0 | - | - |
| Finland | - | - | 97 | 0 | - | - |
| Germany | - | - | 165 | 0 | - | - |
| Italy | - | - | - | - | 55 | 0 |
| Sweden | - | - | - | - | 16 | 0 |

### Share C.coli of all Campylobacter isolates

| Country | 1996 Isolates | 1996 % C.coli | 1997 Isolates | 1997 % C.coli | 1998 Isolates | 1998 % C.coli |
|---|---|---|---|---|---|---|
| **Pigs** | | | | | | |
| Denmark | 150 | 95,3 | 188 | 94,7 | 218 | 92,2 |
| Finland | - | - | - | - | 20 | 95,0 |
| Germany | - | - | 131 | 91,2 | - | - |
| **Pork** | | | | | | |
| Belgium | - | - | 30 | 50 | 90 | 30 |

## *Other animal derived food*

| Country | 1996 Invest. | 1996 % Campy | 1997 Invest. | 1997 % Campy | 1998 Invest. | 1998 % Campy |
|---|---|---|---|---|---|---|
| **Meat** | | | | | | |
| Germany | 22 | 0 | 286 | 0 | 92 | 2,2 |
| Italy | - | - | 22 | 0 | - | - |
| Sweden | 129 | 0 | - | - | - | - |
| **Meat preparations** | | | | | | |
| Germany | 294 | 0 | 254 | 0 | 105 | 0 |
| Italy | - | - | 99 | 0 | 57 | 0 |
| Portugal | 41 | 61,0 | - | - | - | - |
| Sweden | - | - | - | - | 24 | 0 |
| **Meat products** | | | | | | |
| Germany | 94 | 0 | 163 | 0 | 86 | 0 |
| Ireland | - | - | - | - | 47 | 0 |
| Italy | - | - | - | - | 145 | 0 |
| Portugal | 36 | 11,1 | 67 | 6,0 | - | - |
| Spain | - | - | - | - | 19 | 0 |
| Sweden | - | - | - | - | 43 | 0 |
| Unit. Kingdom | 465 | 0,22 | 455 | 0 | - | - |

## 13.2.2.3. ***Echinococcus***

### ***Sheep and goats***

Countries where E. *granulosus* is prevalent (Mediterranean region of the EU)

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Greece [1] | 83595 | 35,2 | - | - | 107 3411 | 2,6 |
| Greece [2] | 45740 | 5,4 | - | - | 557273 | 1,5 |
| Italy [1] | - | - | - | - | 113 7398 | 6,6 |
| Italy [2] | - | - | - | - | 42 113 | 1,5 |
| Portugal | - | - | 141 | 12,1 | 30 | 6,7 |
| Spain | - | - | - | - | 14447390 | 1,5 |

[1] sheep
[2] goat

Investigations in other Member States

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Belgium | 146270 | 0,02 | 128181 | 0 | 125162 | 0 |
| Finland | - | - | 79021 | 0 | - | - |
| France | - | 812 findings | - | - | - | - |
| Germany | 735 | 0 | 338 | 0,3 | - | - |
| Sweden | - | 0 | | 0 | | 0 |
| North.Ireland | - | - | 400000 | 0 | 550661 | 0 |
| Great Britain | 17104824 | 1,0 | 15974414 | 0,8 | - | - |

### ***Cattle***

Countries where E. *granulosus* is prevalent (Mediterranean region of the EU)

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Greece [1] | 2224 | 37,1 | - | - | 91118 | 1,71 |
| Greece[2] | 10245 | 2,7 | - | - | - | - |
| Italy | 517542 | 0,6 | 892891 | 0,3 | 1447866 | 0,4 |
| Spain | - | - | - | - | 2088397 | 1,4 |

[1)]Dairy
[2)]Not Dairy

Investigations in other Member States

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Belgium [1] | 294154 | 0 | 317112 | 0 | 321371 | 0 |
| Belgium [2] | 770440 | 0,004 | 747291 | 0,001 | 644735 | 0,0008 |
| Finland | - | - | 419677 | 0 | - | - |
| Germany | 4868 | 0,1 | 446 | 0 | - | - |
| Sweden | 490745 | 0 | - | - | - | - |
| North.Ireland | - | - | 500000 | 0 | 502553 | 0 |
| Great Britain | 2090131 | 0,5 | 1911262 | 0,2 | - | - |

1) Calves
2) Adult cattle

### ***Pigs***

Countries where E. *granulosus* is prevalent (Mediterranean region of the EU)

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Greece | 22189 | 1,4 | 1358813 | 0,02 | 422426 | 0,004 |
| Italy | 1468194 | 0,003 | 2023178 | 0,02 | 6893587 | 0,010 |
| Portugal | - | - | - | - | 4 | 3 findings |
| Spain | - | - | - | - | 25191876 | 0,1 |
| Spain [1] | - | - | - | - | 151789 | 0,5 |

1) Slaughter at home

## Investigations in other Member States

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Belgium | 11344912 | 0,1 | 11010013 | 0,02 | 11344076 | 0,0002 |
| Finland | - | - | 2208707 | 0 | - | - |
| Germany | 257399 | 0 | 335 | 0 | - | - |
| Sweden | 3853201 | 0 | - | - | - | 0 |
| North.Ireland | - | - | 1400000 | 0 | 1434934 | 0 |
| Great Britain | 12650830 | 0,01 | 13783089 | 0,002 | - | - |

# ***Wildlife***

## Countries reporting on *Echinococcus* findings

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| Country | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* | Invest. | % *Echinococcus* |
| Belgium | 194 | 0 | 200 | 0 | 14 | 0 |
| Denmark | - | - | - | - | - | 0 |
| Finland | - | - | - | - | 63895 | 0 |
| Sweden | 68120 | 0 | - | - | - | 0 |
| Great Britain | 5959 | 0 | 4973 | 0 | - | - |

## Countries reporting on E. *granulosus* findings

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| Country | Invest. | % E. *granulosus* | Invest. | % E. *granulosus* | Invest. | % E. *granulosus* |
| Spain | - | - | - | - | 1964 | 0,4 |

## Countries reporting on E. *multilocularis* findings in foxes

| | 1996 | | 1997 | | 1998 | |
|---|---|---|---|---|---|---|
| Country | Invest. | % E. *multilocularis* | Invest. | % E. *multilocularis* | Invest. | % E. *multilocularis* |
| France | - | - | - | - | 133 | 45,9 |
| Germany | 10656 | 10,6 | 5915 | 7,0 | 3107 | 22,3 |
| TheNetherlands | - | 0 | 272 | 1,8 | 114 | 0 |

## 13.2.2.4. Verotoxin producing *E. coli* (VTEC)

### *Cattle and products thereof*

Countries which run a monitoring programme

| | All age groups | | | Dairy cattle | | | Calves | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| Cattle (herd / farm based data if nothing else stated) | | | | | | | | | |
| Belgium [2] | 467 | - | 7,3 | - | - | - | - | - | - |
| Denmark | 248 | - | 0,4 | - | - | - | - | - | - |
| Germany | 28 | 0 | - | - | - | - | - | - | - |
| Sweden | - | - | - | 125 | - | 5,6 | 2000[1] | - | 0,4 |
| Netherlands | 419 | - | 5,0 | 267 | - | 4,9 | 152 | - | 5,3 |

[1] samples
[2] data out of a research project

| | Cattle (herd level) | | | Beef at slaughterhouse | | | Beef at retail | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| Belgium | 467 [4] | - | 7,3 | 6010 | 0,2 | - | - | - | - |
| | - | - | - | 299 | - | 0 | - | - | - |
| Denmark | 248 | - | 0,4 | - | - | - | 1100[1] | - | 0,1 |
| | - | - | - | - | - | - | 1584[2] | 0,1 | - |
| Finland | | | - | 359[3] | 1,4 | 1,4 | 93[1] | 0 | - |
| | - | - | - | - | - | - | 10[2] | - | 0 |
| Germany | 28 | 0 | - | - | - | - | 222 | 1,8 | - |
| | 149 | 47,7 | - | - | - | - | 1122[1] | 0,9 | - |
| | 70 | 61,4 | - | - | - | - | 485[2] | 0,2 | - |
| Sweden | 125 | - | 5,6 | 334[1] | - | 0 | - | - | - |
| | - | - | - | 543 [2] | 0 | 0 | - | - | - |
| | | | | | - | - | | | |
| | - | - | - | 482 [2] | 0,21 | 0,21 | - | - | - |
| | - | - | - | 650 [2] | 0,31 | 0,31 | - | - | - |
| Netherlands | 419 | - | 5,0 | 334 | - | 0 | - | - | - |

[1] 1997
[2] 1996
[3] Parts rejected at meat inspection
[4] data out of a research project

| | 1996 | | | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| **Cattle (animal based data)** | | | | | | | | | |
| Denmark | - | - | - | 130 | - | 3,0 | - | - | - |
| Finland | - | - | - | 2455 | - | 0,8 | - | - | - |
| Germany | 2487 | 9,5 | - | 3006 | 10,3 | - | 1159 | 8,7 | - |
| Germany [2] | - | - | - | 774 | 17,7 | - | - | - | - |
| Germany [3] | - | - | - | 13 | 7,7 | - | 209 | 0 | - |
| Sweden | 198 | - | 2,5 | - | - | - | - | - | - |
| Sweden | 1585 | | 1,0 | 200 | - | 0,3 | 125[4] | - | 5,6 |
| Sweden [5] | | | | 3072[5] | - | 1,2 | - | - | - |
| Netherlands | 1154 | 6,3 | - | - | - | - | - | - | - |
| Netherlands [1] | 484 | 5,4 | 5,8 | - | - | - | - | - | - |
| Netherlands [3] | 214 | 0,5 | 0,5 | 1437 | 4,8 | - | - | - | - |
| **Beef sampled at retail** | | | | | | | | | |
| Ireland | - | - | - | - | - | - | 1265 | 0 | 0 |
| North.Ireland | - | - | - | 320 | - | 0,6 | - | - | - |

## Investigations in other Member States

| | 1996 | | | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| **Cattle (herd / farm based data)** | | | | | | | | | |
| Italy [3] | - | - | - | 146 | 8,9 | - | 282 | 10,6 | - |
| Italy | 29 | - | 20,7 | - | - | - | - | - | - |
| North.Ireland | - | - | - | - | - | 3 isolates | - | - | - |
| **Cattle (animal based data)** | | | | | | | | | |
| France | - | - | - | 851 | 26,1 | 0 | - | - | - |
| Greece [1] | 302 | - | - | 599 | 14,0 | - | - | - | - |
| Italy | - | - | - | - | - | - | 2631 | 0 | - |
| Italy [2] | - | - | - | 430 | 0 | - | 38 | 0 | - |
| Italy [3] | - | - | - | 336 | - | 4,5 | 477 | 0 | - |
| Luxembourg | - | - | - | 47 | - | 6,4 | - | - | - |
| Portugal | 27 | 11,1 | - | - | - | - | 93 | 0 | - |
| Scotland | - | - | 9 isolates | 585 | 19,2 | - | - | - | - |
| North.Ireland | - | - | - | - | - | - | 166 | 21,1 | 21,1 |
| Great Britain [7] | 377 | - | 5,8 | - | - | - | - | - | - |
| **Beef** | | | | | | | | | |
| Austria | 425 | 0 | 0 | | 3,1 | 0 | - | - | - |
| Greece | - | - | - | 28 | 10,7 | - | 8 | 0 | - |
| Italy | - | - | - | 888 | 0,5 | - | - | - | - |
| TheNetherlands | 325 | - | 1,2 | 162 | - | 0 | - | - | - |

1) all age groups
2) dairy cattle
3) calves, veal
4) follow up study
5) One infected herd
6) Parts rejected in meat inspection
7) Follow up of human infections

## *Milk and products thereof*

| | 1996 | | | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| **Raw milk (sample based data)** | | | | | | | | | |
| Austria | 1348 | 0,3 | 0,1 | 489 | 0 | - | 221 | 0 | - |
| Finland | - | - | - | 2 | 0 | - | 32 | 0 | - |
| Germany | 6191 | 0,1 | - | 6066 | 0,2 | - | 2425 | 0,4 | - |
| Greece | - | - | - | - | - | - | 105 | 0 | - |
| Ireland | - | - | - | - | - | - | 98 | 0 | - |
| Italy | - | - | - | 202 | 0 | - | - | - | - |
| Netherlands | - | - | - | 1017 | - | 0 | - | - | - |
| North.Ireland | - | - | - | 60 | - | 0 | 2 | 0 | - |
| **Heat treated milk (sample based data)** | | | | | | | | | |
| Austria | 666 | 0 | - | 11 | 0 | - | - | - | - |
| Germany | 190 | 0 | - | 256 | 0 | - | 62 | 0 | - |
| Greece | - | - | - | 253 | 0 | - | 36 | 0 | - |
| Italy | - | - | - | 268 | 0 | - | - | - | - |
| **Milk products (sample based data)** | | | | | | | | | |
| Austria | 2414 | 0 | 0 | 411 | 0,2 | 0 | 23 | 0 | - |
| Finland | - | - | - | 4 | 0 | - | 58 | 0 | - |
| Germany | 3435 | 0 | - | 1498 | 0 | - | 645 | 0,3 | - |
| Greece | - | - | - | 324 | 24,4 | - | 781 | 8,1 | - |
| Ireland | - | - | - | - | - | - | 11 | 0 | - |
| Italy | - | - | - | 914 | 1,0 | - | - | - | - |
| Portugal | 18 | - | 0 | - | - | - | - | - | - |
| Sweden | 14 | 0 | 0 | - | - | - | - | - | - |

## *Sheep and goats*

| | 1996 | | | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| **Sheep and goats (herd / flock based data)** | | | | | | | | | |
| Finland | - | - | - | - | - | - | 4 | 1 isolate | - |
| Germany | - | - | - | 9 | 3 isolates | - | 121 | 65,3 | - |
| Italy | - | - | - | 113 | 0 | - | - | - | - |
| Sweden | - | - | - | 3 | - | 2 isolates | 583 | - | 0,9 |
| North.Ireland | - | - | 1 isolate | - | - | 1 isolate | - | - | - |
| **Sheep and goats (animal based data)** | | | | | | | | | |
| Finland | - | - | - | 20 | 0 | - | - | - | - |
| Germany | 99 | 0 | - | 176 | 5,1 | - | 87 | 58,6 | 0 |
| Greece | - | - | - | 3565 | 5,2 | - | - | - | - |
| Italy | - | - | - | 3463 | 0,03 | - | 3697 | 0 | - |
| Portugal | 18 | 0 | - | 43 | 4,7 | - | 188 | 0 | - |
| Netherlands | 1 | 1 isolate | - | - | - | - | - | - | - |
| Scotland [1] | - | - | - | 109 | - | 16,5 | - | - | - |
| Engl.&Wales [1] | - | - | - | 58 | - | 12,1 | 86 | 11,6 | - |
| Great Britain | 107 | - | 31,8 | - | - | - | - | - | - |

1 Connection with human cases suspected

## *Pigs and pork*

| | 1996 | | | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| **Pigs (herd based data)** | | | | | | | | | |
| Germany | 42 | 0 | - | 1 | 0 | - | - | - | - |
| Sweden | - | - | - | 4 | - | 2 isolates | 2446 | - | 0,1 |
| **Pigs (animal based data)** | | | | | | | | | |
| Finland | - | - | - | - | - | - | - | 3 isolates | - |
| Germany | 376 | 0,5 | - | 1097 | 0 | - | 154 | 0 | - |
| Italy | - | - | - | - | - | - | - | - | - |
| Italy | - | - | - | 286 | 5,2 | - | 1099 | 4,0 | - |
| Portugal | 273 | 8,4 | - | 391 | 5,1 | - | 524 | 4,4 | - |
| Netherlands | 32 | 0 | - | - | - | - | 41 | - | 2,4 |
| North.Ireland | - | - | 0 | - | - | 1 isolate | - | - | - |
| Engl.&Wales | - | - | - | 4 | - | 1 isolate | 11 | 45,5 | - |
| Great Britain | 30 | - | 0 | - | - | - | - | - | - |
| **Pork (sample based data)** | | | | | | | | | |
| Austria | 490 | 0,2 | 0 | 287 | 3,8 | 0 | - | - | - |
| Belgium | - | - | - | 179 | - | 0 | - | - | - |
| Denmark | 524 | 0,4 | - | 300 | - | 0 | - | - | - |
| Finland | - | - | - | - | - | - | 1 | 0 | - |
| Germany | 231 | 0 | - | 79 | 0 | - | 28 | 0 | - |
| Greece | - | - | - | - | - | - | 7 | 0 | - |
| Italy | - | - | - | 309 | 5,8 | - | - | - | - |
| Luxembourg | - | - | - | - | - | - | 4 | 0 | - |
| TheNetherlands | 262 | - | 0,8 | - | - | - | - | - | - |

## *Meat and meat products (several animals species)*

| | 1996 | | | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 | Invest. | % VTEC | % EC O157 |
| **Meat (sample based data)** | | | | | | | | | |
| Germany | 1121 | 0,3 | - | 2677 | 0,8 | - | 554 | 6,7 | - |
| Luxembourg | - | - | - | 153 | - | 0 | - | - | - |
| **Minced meat (sample based data)** | | | | | | | | | |
| Austria | 522 | 0 | 0 | 185 | 0 | - | - | - | - |
| Belgium | - | - | - | 509 | - | 0 | - | - | - |
| Finland | 138 | - | 0,7 | 28 | 3,6 | 3,6 | 115 | - | 0 |
| Greece | - | - | - | 26 | 38,5 | - | 275 | 5,5 | - |
| Ireland | 70 | - | - | - | - | - | 22 | 0 | - |
| Italy | - | - | - | 428 | 0 | - | - | - | - |
| Netherlands | 255 | - | 0,4 | 471 | - | 1,7 | - | - | - |
| **Meat preparations (sample based data)** | | | | | | | | | |
| Austria | 302 | 0 | 0 | 74 | - | - | - | - | - |
| Germany | 1033 | 0,4 | - | 1631 | 1,6 | - | 1059 | 2,9 | - |
| Greece | - | - | - | 37 | 21,6 | - | - | - | - |
| Spain | - | - | - | - | - | - | 18 | 0 | 0 |
| Sweden | 60 | 0 | 0 | - | - | - | - | - | - |
| **Meat products (sample based data)** | | | | | | | | | |
| Austria | 2337 | 0,2 | 0,04 | 543 | 1,8 | - | - | - | - |
| Finland | - | - | - | 180 | 0 | - | 51 | - | 0 |
| Germany | 2426 | 0,2 | - | 1162 | 0,7 | - | 510 | 0,4 | - |
| Greece | - | - | - | 213 | 5,2 | - | - | - | - |
| Ireland | - | - | - | - | - | - | 56 | 0 | - |
| Luxembourg | - | 0 | - | - | - | - | 85 | 1,2 | - |
| Portugal | 19 | - | 0 | - | - | - | - | - | - |
| Spain | - | - | - | - | - | - | 38 | 0 | 0 |
| Netherlands | 302 | - | 0,3 | 26 | - | 0 | - | - | - |
| Engl.&Wales | 465 | - | 0 | 455 | - | 0 | - | - | - |

*13.2.3. Annex II.c Human incidence data*

## Human incidence of salmonellosis

Data are grouped according to the national provisions for reporting
Laboratory confirmed cases are notifiable
Outbreak related cases are notifiable
Reports are based on laboratory isolates (without a requirement to notify all cases)
The source of reporting is not defined
Beside this, the requirement to notify foodborne infections (on a clinical basis, *i.e.* without the causative agent) in Austria (until May 1996), Italy, England, Wales and Northern Ireland.

## Countries where salmonellosis is notifiable

| | Salmonellosis cases | | | | Incidence rate per 100000 inhabitants | | | |
|---|---|---|---|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 | 1998 | 1997 | 1996 | 1995 |
| Austria | 7236 | 7488 | 7209 | 8903 | 106,7 | 107,0 | 108,3 | 114,0 |
| Denmark | 3880 | 5015 | 3259 | 3654 | 73,3 | 95,0 | 63,1 | 70,8 |
| Finland (all cases) | 2740 | 2964 | 2847 | 3326 | 52,0 | 58,0 | 56,6 | 66,1 |
| Finland (domestic cases) | 574 | 825 | 527 | 1280 | 11,0 | 10,0 | 17,0 | 25,0 |
| Germany | 97529 | 105340 | 109449 | 115649 | 119 | 128,4 | 133,7 | 141,0 |
| Italy | 6789 | 15198 | 14751 | 11746 | 11,8 | 26,5 | 20,5 | 20,5 |
| Portugal | 186 | 177 | 205 | 191 | 1,9 | 1,8 | 2,0 | 1,9 |
| Sweden (all cases) [1)] | 4300 | 4286 | 4098 | 3562 | 48,6 | 48,5 | 46,3 | 40,4 |
| Sweden (domestic cases) | 453 | 585 | 618 | 558 | 5,1 | 6,7 | 7,0 | 6,3 |

1) Includes healthy carriers; about 90% of cases were acquired abroad

## Countries, where outbreak related cases are notifiable

This regulation exists at least in France and Spain, but not data are available.

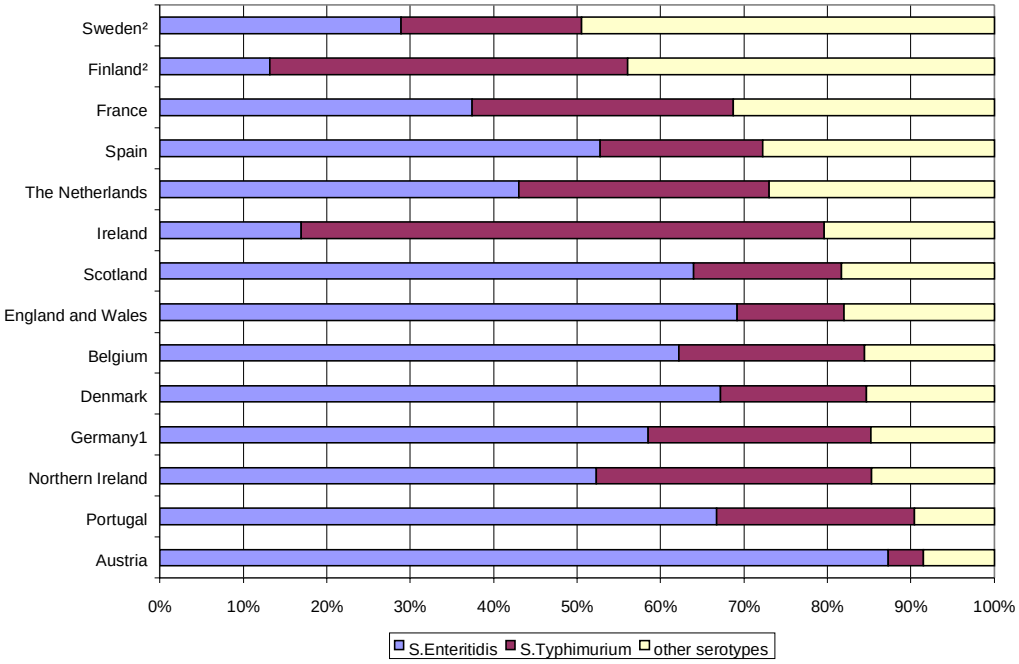
## Countries, where reports are based on laboratory isolates

| | Salmonellosis cases | | | | Incidence rate per 100000 inhabitants | | | |
|---|---|---|---|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 | 1998 | 1997 | 1996 | 1995 |
| Belgium | 13803 | - | - | 11294 | 135,7 | - | - | 112,7 |
| France | 16523 | 19174 | 17152 | 17705 | 28,2 | 33,5 | 30,0 | 30,9 |
| Greece [2)] | 918 | 326 | 389 | 25 | 9,2 | 3,2 | 3,8 | 0,2 |
| Ireland [3)] | 1265 | 1056 | 992 | 767 | 34,9 | 29,1 | 30,8 | 21,6 |
| Luxembourg | - | 307 | 248 | 220 | - | 76,6 | 61,9 | 54,9 |
| Spain [4)] | 4868 | 2867 | 2529 | 4208 | 12,4 | 7,3 | 6,5 | 10,8 |
| The Netherlands | 2263 | 2557 | 2889 | 3018 | 14,5 | 16,4 | 19,1 | 19,9 |
| Scotland | 2109 | 3349 | 3266 | 3107 | 41,2 | 65,5 | 63,0 | 60,5 |
| Northern Ireland | 558 | 431 | 434 | 446 | 33,3 | 26,0 | 26,2 | 27,0 |
| England and Wales | 23420 | 32169 | 30272 | 29717 | 44,9 | 61,8 | 58,0 | 57,6 |
| Total | 188387 | 203537 | 201296 | 217538 | 50,5 | 70,3 | 69,5 | 75,1 |

2) Notification of confirmed cases is required but only cases from hospitals are available (accidentally diagnosed)
3) Returns from each of 8 Health Board regions
4) Microbiological information system based on hospitals notification

## Pattern of Human salmonellosis (CRL-E data)

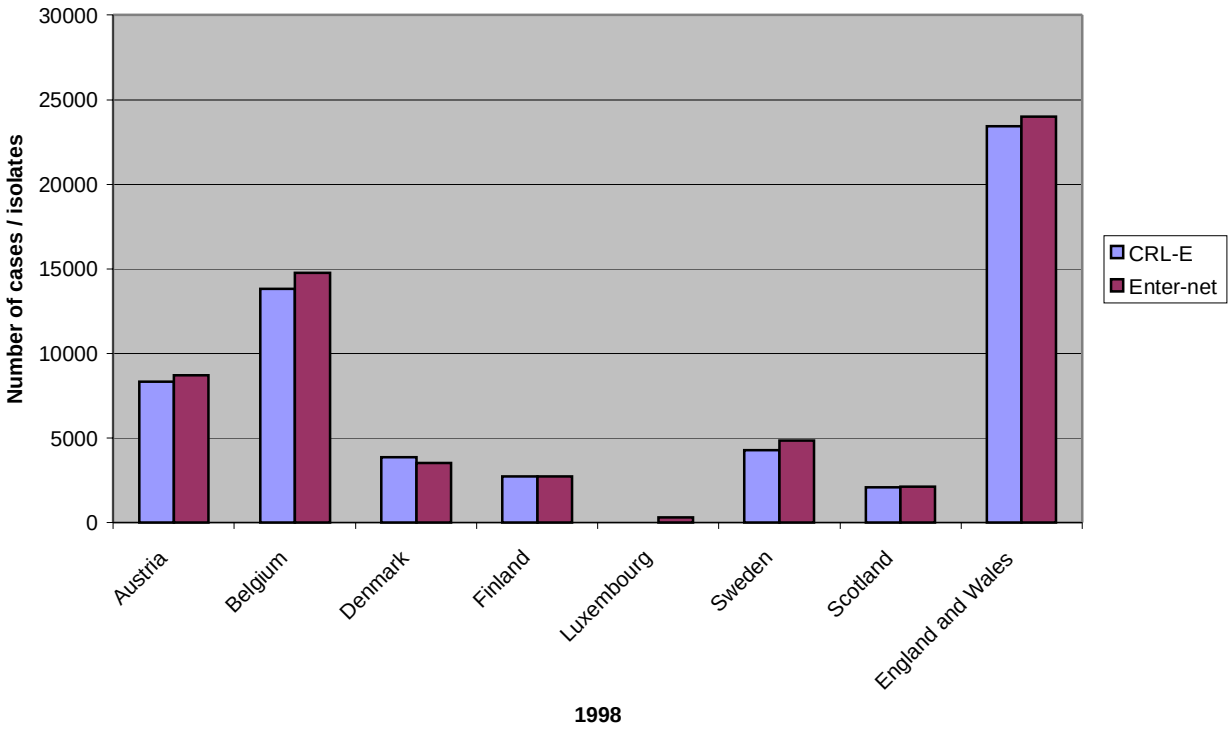
**Table SA 12. Main Salmonella serotypes in human salmonellosis, 1998**




1) Not all Länder 2) based on domestic cases

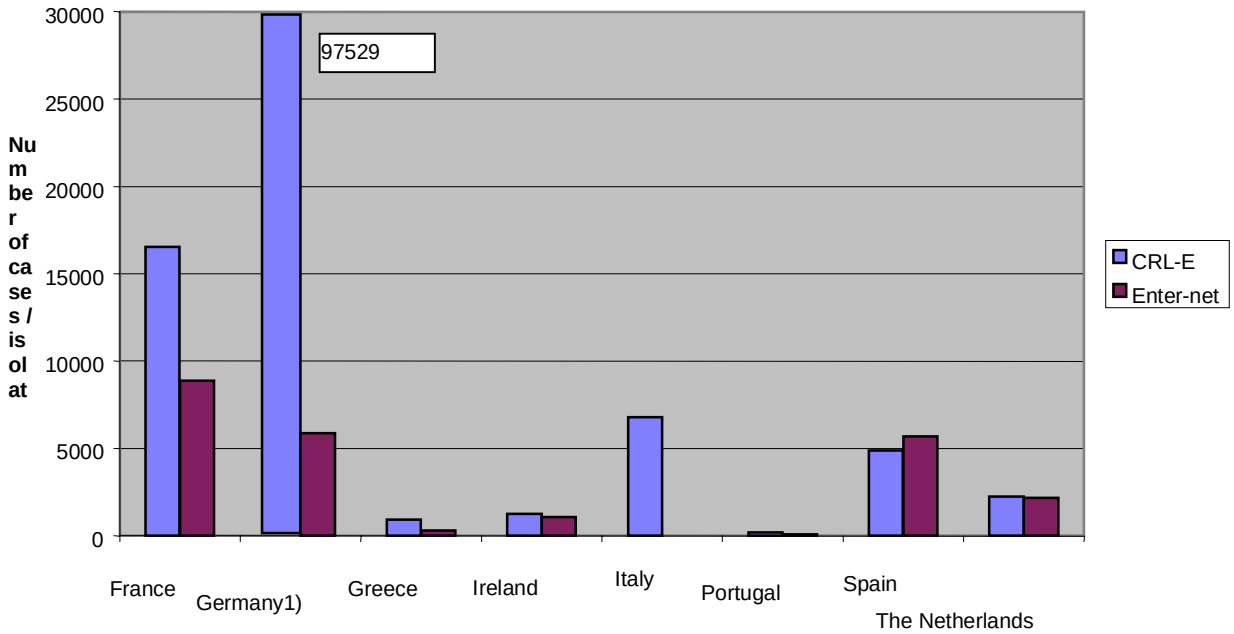
## Comparison of data collected in the frame of the zoonoses directive and Enter-net

**Human salmonellosis in the EU**
**Countries from which reports are considered relatively complete in Enter-net**




**1998**

**Human salmonellosis in the**
**Countries for which data are considered incomplete in**




**1998**

## Human incidence of campylobacteriosis

Data are grouped according to the national provisions for reporting

1) Laboratory confirmed cases are notifiable
2) Outbreak related cases are notifiable
3) Reports are based on laboratory isolates (without a requirement to notify all cases)
4) The source of reporting is not defined

Beside this, the requirement to notify foodborne infections (on a clinical basis, *i.e.* without the causative agent) in Austria (until May 1996), Italy, England, Wales and Northern Ireland.

Countries where **campylobacteriosis** is notifiable

| | Campylobacteriosis cases | | | | Incidence rate* | | | |
|---|---|---|---|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 | 1998 | 1997 | 1996 | 1995 |
| Austria | 2454 | 1667 | 917[1] | - | 30,4 | 21,0 | 10,3 | - |
| Denmark | 3372 | 2666 | 2973 | 2601 | 63,6 | 50,0 | 57,6 | 50,0 |
| Finland [2] | 2851 | 2404 | 2629 | 2273 | 55,9 | 47,0 | 52,3 | 44,6 |
| Germany [3] | 33235[6] | 13095[5] | 10124[5] | 6600[4] | 75 | 70,0 | 54,0 | 37,0 |
| Sweden (domestic cases) | 2586 | 1828 | 1814 | 2551 | 29,23 | 20,66 | 20,51 | 28,84 |
| Sweden (total) | 6544 | 5306 | 5081 | 5580 | 74,0 | 60,0 | 57,4 | 63,2 |

* per 100.000 inhabitants
[1] 1.6.96-31.12.96
[2] Findings notified by the laboratories
[3] Reportable in some Länder (counties) only; incidence rate is calculated for the inhabitants in these regions
[4] Data are related to 6 Länder of Germany (neue Bundesländer, Berlin)
[5] Data are related to 7 Länder of Germany (neue Bundesländer, Berlin, Saarland)
[6] Data are related to 10 Länder of Germany

Countries, where only outbreak related campylobacteriosis cases are notifiable

This regulation exists in no country.

Countries, where reports are based on laboratory isolates

| | Campylobacteriosis cases | | | | Incidence rate* | | | |
|---|---|---|---|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 | 1998 | 1997 | 1996 | 1995 |
| Belgium | 6610 | - | 4991 | 4879 | 65,0 | - | 49,8 | 48,7 |
| Greece | 136 | 26 | 16 | - | 1,3 | 0,26 | 0,2 | - |
| Ireland [7] | 1318 | 943 | 646 | 644 | 36,1 | 26,0 | 20,1 | 17,8 |
| Luxembourg | - | 106 | 129 | - | - | 26,4 | 33 | - |
| Spain [8] | 4328 | 3711 | 3557 | 3225 | 11,0 | 9,5 | 9,1 | 8,2 |
| The Netherlands | 3489 | 3661 | 3737 | 2871 | 22,4 | 23,5 | 24,7 | 18,3 |
| Scotland | 6375 | 5528 | 5218 | 4377 | 124,4 | 108,2 | 85,3 | 85,3 |
| Northern Ireland | 774 | 778 | 653 | 557 | 46,2 | 46,8 | 39,0 | 34,0 |
| England and Wales | 58058 | 50201 | 43240 | 43902 | 103,2 | 96,5 | 85,1 | 85,1 |
| Total | 129544 | 90092 | 82994 | 70909 | 50,6 | 38,1 | 33,7 | 31,1 |

* per 100.000 inhabitants
[7] Returns from Health Board regions
[8] Sistema Microbiologica Informacione

Limited information available on the *Campylobacter* species shows that about 60-80% of the cases are caused by *C.jejuni* and up to 20% by *C.coli*

## Human incidence of echinococcosis

In reporting echinococcosis no distinction is made between alveolar and cystic echinococcosis.

Countries where **echinococcosis** is notifiable

| | Echinococcosis cases | | | |
|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 |
| Denmark | 0 | 0 | 0 | 0 |
| Finland | 1 | 0 | 0 | 0 |
| Spain [1] | 283 | 312 | 396 | 362 |
| Sweden [2] | 7 | 7 | 6 | 3 |

[1] Data based on Epidemiological Notifiable Diseases Surveillance System
[2] Laboratory based reports; no known domestic cases

Countries, where only hospitalised cases are notifiable

| | Echinococcosis cases | | | |
|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 |
| The Netherlands [3)] | 36 | 52 | 24 | 28 |

3) Cases reported in context of diagnosis

Countries, where reports are based on laboratory isolates

| | Echinococcosis cases | | | |
|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 |
| Portugal | 34 | 44 | 53 | 39 |
| Spain | 16 | 31 | 27 | 39 |
| Scotland | 0 | 0 | | 0 |
| England and Wales | 11 | 14 | 43 | 12 |

Countries, where the source of the data is not specified

| | Echinococcosis cases | | | |
|---|---|---|---|---|
| Country | 1998 | 1997 | 1996 | 1995 |
| Greece | 122 | 101 | 43 | - |
| The Netherlands [4)] | 23 | | | |

4) Serological positive cases; only RIVM figures, data not included into calculation of totals

Notification is not mandatory in Denmark and Germany.
No data are reported from Austria, France, Germany and Luxembourg

# Human incidence of Verotoxin producing *E. coli*

| | HUS Cases | | | | Clincial cases (non HUS) | | | | All cases | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Country | Clin | Conf. | O157 | Non-O157 | Clin | Conf. | O157 | Non-O157 | Clin | Conf. | O157 | Non-O157 |
| Austria | | 11 (0,14) | 10 (0,12) | 1 (0,01) | | 17 (0,21) | 16 (0,20) | 1 (0,01) | | 28 (0,35) | 26 (0,13) | 2 (0,21) |
| Belgium | - | - | - | - | - | 40 | - | - | - | - | - | - |
| Denmark | - | 3 (0,06) | 2 (0,04) | 1 (0,02) | - | 31 (0,59) | 4 (0,08) | 27 (0,51) | - | 34 (0,65) | 6 (0,12) | 28 (0,53) |
| Finland | 5 (0,1) | 5 | 5 | 0 | 39 (0,7) | 39 | 13 | 26 | 44 (0,8) | 44 (0,8) | 31 | 13 |
| France | 74 (0,64) | 25 | 24 | 1 | - | - | - | - | 74 (0,64) | 25 | 24 | 1 |
| Germany | - | - | - | - | - | - | - | - | 644 (0,79) | - | - | - |
| Ireland[2)] | 1 (0,03) | - | - | - | 6 (0,16) | 58 (1,59) | 58 (1,59) | - | 7 (0,19) | 58 (1,59) | 58 (1,59) | - |
| Portugal | 2 | 2 | 2 | - | - | - | - | - | - | - | - | - |
| Sweden | - | - | - | - | - | - | 72 (0,8) | - | - | - | 72 (0,8) | - |
| Spain | - | 1 | 1[1)] | - | - | - | - | - | - | - | - | - |
| Netherlands | 19 | - | - | - | - | - | - | - | - | - | 31 | - |
| Scotland | - | - | - | - | - | - | - | - | - | - | 216 (4,2) | - |
| Northern Ireland | - | - | 5 (0,3) | - | - | - | 19 (1,13) | - | - | 24 (1,43) | 24 (1,43) | - |
| England and Wales | - | - | - | - | - | - | - | - | - | 890 (1,7) | 890 (1,7) | - |

[1)] imported from Mauretania

[2)] Returns from 4 out of 8 Health Boards

Clin - clinical cases; Conf. - confirmed cases

## 13.3. Annex III : Networks

### *13.3.1. Annex III.a : Campynet*

A Network for the standardisation and harmonisation of
Campylobacter Molecular Typing Methods (CAMPYNET).

D.G. Newell[1], S.L.W. On[2], J.A. Wagenaar[3], R. Madden[4], B. Duim[3], J. van der Plas[5].

1. Veterinary Laboratories Agency (Weybridge), UK
2. DVL, Copenhagen, DK
3. ID-Lelystad, Lelystad, NL
4. Queen's University, Belfast, UK
5. TNO-Zeist, NL

A network was established on the 1st October 1998 to harmonise and standardise molecular typing techniques for *C. jejuni/coli*. The Network is funded by the European Commission for 3 years and formally comprises 24 participants from 11 countries. The project is planned in two phases. In phase 1 a reference set will be established and standard operating procedures for fla-typing, PFGE, AFLP and data handling will be recommended. In phase 2 the technologies will be transferred to all participant laboratories. Six working groups have been established to implement this plan.

Co-ordinator of CAMPYNET: Diane G. Newell (dnewell.cvl.wood@gtnet.gov.uk)

WG 1: collection, characterisation, selection and distribution of reference strain set: Jaap A. Wagenaar, (J.A.Wagenaar@id.wag-ur.nl)

WG 2: recommendation of procedures for fla-typing: Bob Madden

(madden robert/science fsd newforge@dani.gov.uk)

WG 3: recommendation of procedures for PFGE: Stephen On (sto@svs.dk)

WG 4: recommendation of procedures for AFLP: Birgitta Duim (B.DUIM@ID.DLO.NL)

WG 5: evaluation of software for data-handling: Stephen On (sto@svs.dk)

WG 6: investigation of alternative and new techniques: Jan van der Plas (vanderPlas@voeding.tno.nl)

At this moment the reference strain set is completed. This strain set will be made available, at cost, to non-participant laboratories on request in the future. Comparisions of various typing procedures are ongoing in several laboratories and a website has been established (http://www.svs.dk/campynet/)

Further progress will be reported.

### *13.3.2. Annex III.b : Echinoreg*

EurEchinoReg : European Network for
Concerted Surveillance of Alveolar Echinococcosis
SOC 97 20239805F01 (97 CVVF1-057-6)

**Background and objectives**

Alveolar echinococcosis (AE) is a rare and chronic but lethal zoonosis which mimics a cancer although it is due to a larval cestode, *Echinococcus multilocularis*. Major changes in the diagnosis and management of the disease for the past 15 years have made it a potential concern for Public Health despite a relatively low prevalence.

Because of the very particular life cycle of the cestode and the nature of the ecological factors which allow its maintenance in nature, the disease is found in geographically limited endemic areas which had not been markedly modified for the past decades. However, changes in population behaviour, migrations of people and families have increased the exposure to infectious eggs of *E. multilocularis* and, as a consequence, made the diagnosis of AE in non-endemic areas more frequent. On the other hand, because of a marked increase in the number of foxes in Europe for the past 10 years, and changes in fox behaviour that currently leads to their presence in towns and cities, the risk of human infection in areas not previously recognised as endemic has to be considered.

Because of its geographic distribution which is linked to specific parameters including altitude, climate, agricultural practices, human and animal behaviour, AE is unequally present in the various regions of a given country but it ignores borders… Moreover, the time-lag between changes in the environment and animal hosts and their consequences on the prevalence of the disease can be long (more than 10 years) and a systematic surveillance can be of help to anticipate any emergence of the disease in new areas or increase of the disease in the already recognised endemic areas, by studying animal infection and disclosing human cases.

A concerted European approach to its study seemed thus typically adapted to add value to any action in this field, and a pilot project appropriate to set up a formal network from teams otherwise informally linked by bicentre projects and occasional *meetings.*

The aims of the pilot project were:

*To collect reliable epidemiological and clinical data on AE cases in humans, in countries of the EU where the disease is endemic or suspected to become endemic.*

*To collect reliable epidemiological data on adult stages of the parasite in definitive animal hosts, and of the larval stage in intermediate hosts in the same countries.*

*To set up a network for epidemiological surveillance and elaborate an agreed European system for case definition and staging.*

*To promote a better information on the disease, its prevention and its treatment*

*To facilitate international staff exchanges and training of physicians, surgeons, veterinarians, PhD students and post-doctoral researchers.*

**Main results**

The European collaboration in EurEchinoReg made available the information on 579 patients with alveolar echinococcosis ( AE ) throughout Europe including Turkey. This is the far largest number of cases ever assembled in the endemic foci of the European Union and border countries. Main results, as available in June 1999, are shown in tables and figures V-A1-3, from the final report of the pilot project to the European Commission DGV (June 15, 1999). The conditions leading to diagnosis of AE are available for 378 patients in the European countries. Most patients were diagnosed with AE in the age range from 50 to 70. However, the recruitment in Germany indicates a shift towards younger age groups ( < 40 ). In parallel, the prevalence for the fox-tapeworm has steadily been increasing during the last 10 –15 years indicating probably the recent increased risk to the public. The circumstances of AE diagnosis was evaluated. In contrast to the published literature, 70 % of the patients were referred to health centres because they had symptoms, 25% without. In nearly all cases the primary infected organ was the liver, primary extrahepatic manifestation of disease occurred in 2,6 %. This figure is higher than previously anticipated. Most impressively, at the time of diagnosis 12,4 % of patients had already metastatic spread of the disease. According to the data set 9% did not receive specific treatment for AE. This first glance on the data readily indicates the necessity of a centralised European institution such as the EurEchinoReg Registry. The implementation of the infrastructure was therefore the action which is now the platform of communication between different EU countries. Based upon the experience of the pilot project, new questionnaires adapted to case reporting by family physicians and/or by the patients themselves were developed. The detailed questionnaires will be used in the future for the study of a cohort of patients in order to better study and evaluate the treatment strategies. A regular follow-up of all registered patients will be undertaken every two years by sending a specific questionnaire to the patients family physician or Reference Centre physician, upon agreement by the patient. Because of the short time-period available for the collection of cases, not all complete files of AE patients have been entered in the register ; especially, epidemiological data are still missing for many cases, and correctness of the diagnosis of AE for some cases reported from non-endemic areas has to be checked.

The definitive evaluation will only be achieved at the beginning of 2000 but the data collected so far point out to the occurrence of AE in a EU country which was not previously known as endemic, Belgium, and of infected foxes in a country where they were not infected in the past, the Netherlands. Human cases and fox infection also exist now in the border countries of central Europe. Fox infection in EU and Swiss cities was demonstrated.

During its first year, the pilot programme has achieved a series of goals: (1) the infrastructure of a network has been established, (2) actions have been taken to set up national reference centres, (3) a common definition of AE cases and a common staging system (PNM) have been elaborated and evaluated, (4) updated maps of endemic areas have been drawn, (5) new trends in the incidence of human cases and animal infection have been clearly disclosed.

Surveillance of emerging AE cases in non-endemic areas or in ”low-risk” populations appears necessary from both the observation of isolated AE cases in these settings during the past few years and the trends in the infection of foxes in endemic and non-

endemic areas. The Web Site www.eurechinoreg.org should facilitate the information on the disease in these areas so that both populations and physicians can be aware of it and refer new cases to the Register. For this purpose, the Web Site will be linked to other medical Web Sites, and especially to the "Orphanet" Web Site on rare and orphan diseases, set up by the French Inserm (http://orphanet.infobiogen.fr), and as soon as it is developed, to the European rare diseases database (programme of Community action 1999-2003 on Rare Diseases in the context of the framework for action in the field of public health). The network would take benefit of its inclusion in a wider system for epidemiological surveillance and control of communicable diseases at the EU level.

Table V-A1: Case registration in the EurEchinoReg-Network, as of May 1999

*1a) Total number of cases according to the year of diagnosis*

| Years of first diagnosis | Austria | France | Germany | Switzerland | Poland | Greece | Turkey |
|---|---|---|---|---|---|---|---|
| < 1971 | 3 | 6 | 10 | - | - | - | |
| 1971 - 1980 | 5 | 14 | 16 | 2 | - | - | |
| 1981 - 1990 | 21 | 91 | 26 | 40 | 1 | - | |
| > 1990 | 20 | 42 | 46 | 25 | 7 | 1 | |
| Total N | 51 | 153 | 98 | 67 | 8 | 1 | 201* |

* patients were diagnosed between 1980 - 1998

*1b) Available data sets of all registered cases*

| Data sets from | Patients alive [N] | | Deceased [N] | | epidemiological data set available [N] | consent to be registered nominally [N] |
|---|---|---|---|---|---|---|
| countries using the standard clinical questionnaire | Men | women | Men | women | | |
| Austria | 12 | 22 | 10 | 7 | 34 | 8 |
| France | 50 | 62 | 24 | 17 | 22 | 22 |
| Germany | 42 | 40 | 10 | 6 | 68 | 64 |
| Poland | 3 | 5 | - | - | - | ? |
| Greece | 1 | - | - | - | 1 | ? |
| countries using different data lists | | | | | | |
| Switzerland | 24 | 30 | 6 | 7 | - | ? |
| Turkey | 201 | | - | - | - | ? |
| Total* N men / women | 132 | 159 | 50 | 37 | | |
| Total N / % alive / deceased | 492 / 85% | | 87 / 15% | | | |
| All patients | 579 | | | | 125 | 94 |

* No. of patients from Turkey not included

*Due to the differences in patient recruitment in each country, the characteristics of patients registered to date are not representative for the whole European patient cohort; therefore, in-depth analysis of the data or comparison between countries is not yet meaningful.*

Fig. V-A-2 Age of AE-patients at first diagnosis.

Proportions of age class by countries (N = 375)

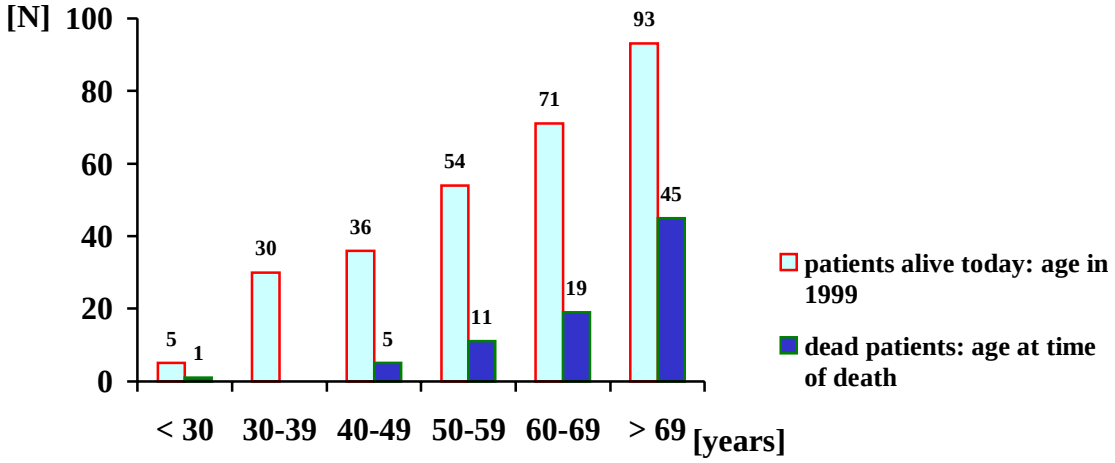



Fig. V-A-3 Age of patients alive today, and of deceased persons at time of death.

**No. of patients for age classes (N = 370)**

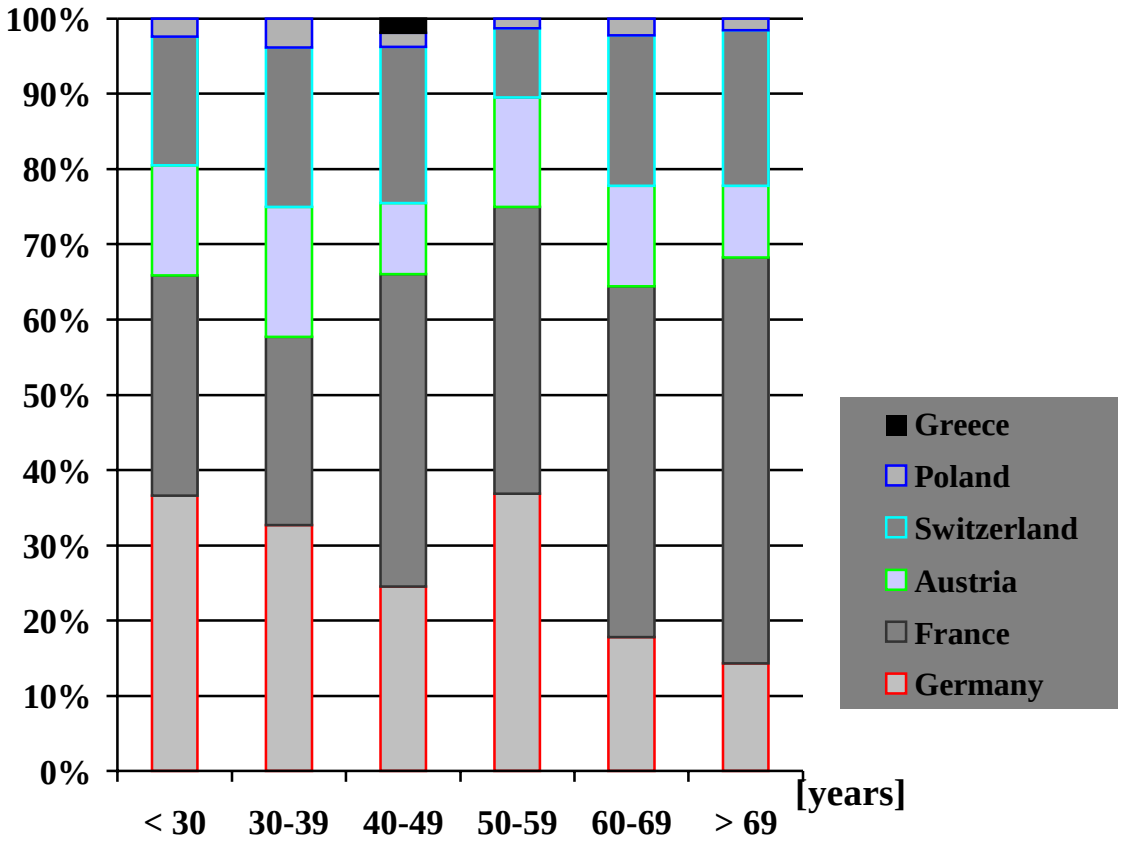


Table V-A-4:Clinical parameters of the AE patients in the EurEchinoReg-Registry, as of May 1999, Total N = 378

| Variables | Patients N | (%) | data missing |
|---|---|---|---|
| • Patients alive | 291 | (77.0) | |
| • Gender | | | |
| men | 182 | (48.1) | |
| women | 196 | (51.9) | |
| • Age at time of diagnosis | N = 371 | (98.1) | 7 (1.9) |
| [years] median / range | 56 / 7 - 86 | | |
| mean / std dev | 53.09 / 16.44 | | |
| • Diagnosis / circumstances | | | 20 (5.3) |
| symptoms | 268 | (70.9) | |
| by chance | 68 | (18.0) | |
| screening | 22 | (5.8) | |
| • Techniques applied for diagnosis | | | 26 (6.9) |
| imaging techniques (liver-ultrasound, -MRT, -CT) | 293 | (77.5) | |
| antibody detection | 253 | (66.9) | |
| histology/parasitology | 209 | (55.3) | |
| • Manifestation of the disease *at first diagnosis* | | | |
| 1. Liver | | | |
| right lobe only | 159 | (42.1) | |
| left lobe only | 44 | (11.6) | |
| right + left lobe | 118 | (31.2) | |
| location of lesion in the liver not documented | 47 | (12.4) | |
| 2. Primary extrahepatic manifestation | 10 | (2.6) | |
| 3. Invasion of organs adjacent to the liver | 99 | (26.2) | |
| 4. Occurrence of distant metastases | 47 | (12.4) | |
| • Therapy | | | 3 (0.8) |
| surgery + chemotherapy | 173 | (45.8) | |
| chemotherapy only | 129 | (34.1) | |
| surgery only | 39 | (10.3) | |
| none | 34 | (9.0) | |
| Liver transplantation* | 8 | (2.1) | |

* The number of these patients is included in the groups with "surgery"

Table V-A-5: Epidemiological parameters of the AE patients in the EurEchinoReg-Registry, as of May 1999, Total N = 125

| Variables | Patients N (%) | | |
|---|---|---|---|
| • Patients alive | 121 (96.8%) | | |
| • Gender | | | |
| men | 57 (45.6%) | | |
| women | 22 (54.4%) | | |
| • Age at time of diagnosis | N = 124 | | |
| [years] median (range) | 50 (8 - 86) | | |
| mean (std dev) | 48.06 (16.25) | | |
| | yes | no | data missing |
| • Family history of AE | 2 (1.6%) | 111 (88.8%) | 12 (9.6%) |
| • Vacations spent in endemic areas other than home country | 56 (44.8%) | 53 (42.4%) | 16 (12.8%) |
| • Occupation in agriculture, forestry or gardening | 72 (57.6%) | 41 (32.8%) | 12 (9.6%) |
| duration in lifetime < 10 years | 6 (8.3) | | |
| ... > 10 years | 58 (80.6%) | | |
| ... unknown | 8 (11.1%) | | |
| at time of diagnosis | 34 (47.2%) | | |
| • Ownership of pet animals | 93 (74.5%) | 16 (12.8%) | 16 (12.8%) |
| dogs only | 23 (24.7%) | | |
| cats only | 19 (20.4%) | | |
| dogs+cats | 51 (54.8%) | | |
| patient cared for pets himself | 45 (48.4%) | 28 (30.1%) | 20 (21.5%) |

## Conclusions-Achievement of the aims of the project

The pilot project European Network for Concerted Surveillance of Alveolar Echinococcosis ”EurEchinoReg” showed that a European register of human AE case and of epidemiological data on the infection of intermediate and definitive hosts by *Echinococcus. (E. ) multilocularis* was not only feasible but worthwhile.

In addition to previously well recognised EU regions where the disease is endemic in rural areas, **human AE cases are now disclosed :**

- in regions located *at the border of endemic areas,* but not previously identified as ”at risk”
- *in cities of the endemic areas* and, even, of the non-endemic areas, in populations *not considered* ”at risk” in the past
- *in central Europe countries* at the border of the EU, an area considered up-to now as ”free” of *E. multilocularis* life cycle, between the ”Russian” and the ”European” foci of infection.

An active functioning of *E. multilocularis* life cycle, manifested by a significant infection of foxes, is currently observed

- *with an increased prevalence,* in previously studied areas of endemic regions of EU countries (averaging 60-70% of foxes in most ”at risk” spots)
- *in cities* of the endemic areas of EU and Switzerland (*i.e.* Stuttgart, Zürich)
- *in ”new” regions* of the EU countries endemic for *E. multilocularis* infection
- in a EU country considered up-to-now as ”free” of *E. multilocularis, the Netherlands.*
- in central Europe countries at the border of EU

**Diagnosis and treatment management of AE cases in EU countries do not always follow the consensus guidelines** given by expert groups (*i.e.* the WHO Informal Working Group on Echinococcosis) : especially, confusion between ”alveolar” and ”cystic” echinococcoses (the management and prognosis of which are markedly different) was frequent ; and use of chemotherapy was often misconducted.

The European Network in the Pilot Project, contributed to give a clear **case definition** ; guidelines for diagnosis ; **common staging** (**PMN**) for disease assessment ; and accurate information to the professionals and the public, especially through the development of the Web Site www.eurechinoreg.org**.**

Thanks to the EurEchinoReg pilot project, **national referral centres** were established, and progress was made towards incorporation of AE within the national surveillance system. **Exchanges between European teams have markedly increased** and exchanges of students to achieve the goals of the AE-EU Network have already begun and will be intensified in the future.

Systematic and concerted registration of AE cases and animal epidemiological data confirm that **AE is actually becoming an emerging disease** in new rural areas and in cities of EU countries and Switzerland, and in central Europe countries.

Despite the usually low prevalence of the disease, **AE severity and cost makes any increase or emergence a concern for public health**, and a prolonged epidemiological surveillance should be done.

### *13.3.3. Annex III. 3: Enter-Net*

## The Enter-net Surveillance network

Introduction

Enter-net is the EU-wide network for the surveillance of human *Salmonella* and Vero cytotoxin producing *Escherichia coli* (VTEC) infections linking the national enteric reference centres and the national communicable disease surveillance centres in the EU. The network developed as a concerted action in 1994, initially to standardise the definitive typing of *Salmonella* and collect a standard set of data on all isolates. To enable comparisons across Member States and identify and track food contamination incidents and outbreaks of humans disease it was essential to harmonise typing methods and the collection of epidemiological data. Enter-net has made great progress with *Salmonella* and the same phage typing system for S. *enteritidis* and S. *typhimurium* is now in use. The project grew to include antibioitic resistance monitoring and more recently molecular typing. *E coli* O157 was added in 1997. Enter-net has now created international databases for both salmonellosis and infection with VTEC. It has grown from a research project to a developing public health surveillance network. From 2000 the core international surveillance activity of Enter-net is funded by DG SANCO as part of the Commission's response to its communicable disease Network Decision.

Many factors affect comparisons of disease incidence between the Member States and need to be considered when examining data. These differences include: the proportion of the population presenting to the health service when they have a diarrhoeal illness, the proportion that have a faecal specimen examined, the approach of the front line laboratories in looking for pathogens (ie not all labs look for *E coli* O157), the proportion of isolates that are referred for definitive typing (not all countries have a national reference laboratory). Furthermore the epidemiological approach to investing sporadic cases and outbreaks, to identify associated cases and to establish the routes of transmission are also very different.

A vast amount of microbiological and epidemiological expertise exists in the Member States and this collaboration has enabled a sharing of this expertise. Great strides have been made and now all countries are contributing data to the central database. With the constant movement of people and foodstuffs across the EU pathogens can rapidly become widely disseminated. It is imperative that the EU has an effective surveillance network to both identify outbreaks and monitor trends. Establishing the burden of disease resulting from *Salmonella* and VTEC and is essential if we are to develop effective public health priorities and interventions.

Trends in *Salmonella* infections

Between 1994 and the end of September 1999, 515,000 reports of human *Salmonella* isolates have been included in the database.

The proportion of the total of laboratory-confirmed human *Salmonella* isolates in each EU country that has been reported to Enter-net varies enormously between countries. All cases reported to the National Reference Centre (NRC) are incorporate d into the international database. Broadly speaking two categories of countries can be described those from which most laboratory-confirmed infections are reported to the NRC and those from which a minority of the infections (usually less than half) are reported. Data from the former group of countries can be used for calculating rates and for estimating the overall

impact of salmonellosis, while data from the other countries are valuable for indicating secular trends.

In a few countries a very high proportion of *Salmonella* cases are known to have been acquired abroad **(Table 1).** In particular, just over a half of the cases from Sweden are reported as definitely being associated with travel and for most of the remaining cases there is no information on travel. Therefore, when comparing rates between countries it must be acknowledged that the overall rate for Sweden does not reflect the incidence of salmonellosis acquired indigenously.

Detailed data on the phage types of S. *enteritidis* and S. *typhimurium* are available from six countries so it is possible to show the relative distribution of the major phage types for these countries only. However, the figures may be understated, as data on phage-type was not fully completed for some of the countries in the earlier years of the Enternet project.

The incidence of human salmonellosis varies considerably between EU countries **(Table 2).** In 1998 and 1999, the incidence in Belgium rose considerably to over three times that in the United Kingdom, where incidence fell, or Finland, where incidence was static. Austria is another country in which the annual incidence per million population was over 1,000, although the trend was downwards since 1995. In Denmark the incidence rose by 80% to 900 per million in 1997 and fell again the following year to 670.

It is difficult to interpret the trend in incidence in those EU countries for which data are incomplete **(Table 3).** Changes in the referral pattern of isolates to the reference laboratory may explain the apparent doubling of reports in Germany between 1996 and 1997. In France the data suggests there may have been a peak in incidence in 1997, similar to what occurred in Denmark.

In most countries S. *enteritidis* was the commonest serotype reported **(Table 4)**. Only from France and Ireland and S. *typhimurium* reported more often than S. *enteritidis*. The proportion of isolates that was S. *typhimurium* was much greater in Belgium, France, Germany, Ireland, the Netherlands, and Spain, compared to other countries. *Usually S. virchow* was the third commonest serotype reported, apart from Belgium, France and Spain, where *S. hadar* was third in frequency. Of the S. *enteritidis* isolates, phage type 4 predominated in Austria, Germany, and the UK, but in Spain and Sweden it was much less common. Similar variations occurred between countries in the proportion of S. *typhimurium* isolates that was DT104 from 62% in the UK to only 18% in Spain.

There were marked differences in the trend in S. *enteritidis* incidence between countries **(Table 5 and Figure 1)**. Incidence doubled in Belgium from 500 to 985 per million between 1995 and 1999. Elsewhere, incidence was static or in slow decline apart from Denmark where it would appear that much of the 1997 peak was due to S. *enteritidis* (Figure 1).

The incidence of S. *typhimurium* has stayed more constant over the five yars in most countries although the incidence in Belgium was much greater than elsewhere (Table 6 and Figure 2). In the UK there has been a steady decline in S. *typhimurium* incidence.

The average annual number of *Salmonella* isolates for the period 1995 to 1999 for the seven countries from which reports were relatively complete was 53, 686. The combined population of these countries is 85.3 million, giving an annual rate of 630 laboratory confirmed isolates per million. Applying this rate to the total EU population of 373 million produces an estimate of 235,000 for the annual number of laboratory-confirmed cases of salmonellosis in the Community.

Notified laboratory confirmed cases are an underestimate of the total number of *Salmonella* infections in a country as only a proportion of patients present to the health service, only a proportion have a feacal specimen examined, only a proportion of isolates are definitively typed and only a proportion may be reported nationally. The ratio of actual to laboratory confirmed cases will be different in each member state. A study in England estimated it to be 3.2 to 1, and for the United States it was reported to be 38 to 1. Using the English ratio the annual total for cases would be 752,000, and using the American ratio it would be 8,931,000 cases. Therefore, over the past five years the impact of salmonellosis throughout the EU may have been between 750 thousand and 8.9 million cases of human infection each year.

Conclusions

The data presented on *Salmonella* illustrates both the power and the limitations of the surveillance data that is available within the Enter-net database. The data show marked differences between selected European countries in the overall incidence of salmonellosis, in the distribution of different serotypes and phage types, and in incidence trends. In countries from which a minority of laboratory confirmed cases are reported to the international database, the minority that is reported may not be representative of all cases in that county. Using a number of questionable assumptions a range has been derived, of 750,000 to 8,9000,000, within which probably lies the number for the current annual total for human *Salmonella* cases throughout the EU.

In particular the group of countries, from which the overall EU rate of laboratory confirmed cases was estimated, did not include any Mediterranean countries.

While there are some limitations to the database, the quality of the data is consistently improving and it should be remembered that the data presented has not illustrated the effectiveness of the Enter-net surveillance system at addressing it’s primary purpose of rapid recognition of international outbreaks. Numerous outbreaks have been indentifed and dealth with since the inception of Enter-net and the collaboration between the key players in each member state is ensuring that the EU is developing an effective public response to outbreaks. Todays outbreak in one member state can be another members problem tomorrow. Efforts are ongoing to improve the quality of data available to Enter-net and the capablility of the surveillance system.

The Enter-net network has evolved from humble beginnings and can become an effective surveillance and early warning system integrated into the mainstream of public health protection.

Sharing of expertise, training of staff, antibiotic resistance monitoring and developing standard molecular typing techniques are ongoing. Consideration needs to be given to incorporating of other enteric pathogens of public health significance into the surveillance system.

Creating similar databases of definitively types isolates from animals and from food and linking all three would create a public health infrastructure that could track contamination through the food chain to the animal reservoir or other source whether within the EU and third countries. Typing methods now exist to permit more and more specific comparison of strains making effective traceability a reality.

Tables and Figures

From " The incidence of salmonellosis in the EU – Report from the Enter-net surveillance network"

Table 1. Proportion of human *Salmonella* isolates in each country reported as travel associated.

| Country | Reported as travel associated (%) | Assumed not to be travel associated (%) | No information |
|---|---|---|---|
| **Austria** | 2 | 99 | - |
| **Belgium** | 1 | - | 99 |
| **Denmark** | 12 | 83 | 7 |
| **England & Wales** | 11 | 88 | - |
| **Finland** | - | - | 100 |
| **France** | - | - | 100 |
| **Germany** | - | - | 100 |
| **Greece** | 1 | 2 | 97 |
| **Ireland** | 2 | 9 | 89 |
| **Italy** | 1 | 20 | 80 |
| **Luxembourg** | 2 | 45 | 53 |
| **The Netherlands** | 6 | 1 | 93 |
| **Portugal** | 0 | 0 | 100 |
| **Scotland** | - | - | 100 |
| **Spain** | - | - | 100 |
| **Sweden** | 52 | 3 | 45 |

Table 2. Trend in human salmonellosis in selected EU countries from which reports are relatively complete

| | **Pop$^n$** | **1995** | **1996** | **1997** | **1998** | **1999*** |
|---|---|---|---|---|---|---|
| **Austria** | (8.1) | 9,822 | 9,391 | 8,919 | 8,745 | 8,127 |
| **Rate** | | 1,210 | 1,160 | 1,100 | 1,080 | 1,000 |
| **Belgium** | (10.2) | 10,647 | 11,891 | 11,868 | 14,776 | 15,740 |
| **Rate** | | 1,040 | 1,170 | 1,160 | 1,450 | 1,540 |
| **Denmark** | (5.3) | 3,116 | 2,822 | 4,786 | 3,532 | 4,057 |
| **Rate** | | 590 | 530 | 900 | 670 | 770 |
| **England & Wales** | (51.1) | 30,565 | 29,859 | 33,276 | 24,005 | 20,955 |
| **Rate** | | 600 | 580 | 650 | 470 | 410 |
| **Finland** | (5.2) | 0 | 0 | 2,891 | 2,729 | 2,631 |
| **Rate** | | 0.0 | 0.0 | 560 | 520 | 510 |
| **Luxembourg** | (0.4) | 0 | 0 | 302 | 303 | 344 |
| **Rate** | | 0.0 | 0.0 | 760 | 760 | 860 |
| **Scotland** | (0.5) | 3,170 | 3,277 | 3,330 | 2,138 | 1,832 |
| **Rate** | | 640 | 650 | 670 | 430 | 370 |
| **Sweden** | (8.8) | 4,016 | 4,523 | 4,845 | 4,868 | 5,353 |
| **Rate** | | 460 | 510 | 550 | 550 | 610 |

Rate per million population * 1999 full year estimates calculated from 1999 actuals to end September plus average october through December from previous years'data**.**

Table 3. Human *Salmonella* isolates reprted from countries for which data are incomplete*

| Country | 1995 | 1996 | 1997 | 1998 |
|---|---|---|---|---|
| **France** | 8,835 | 8,981 | 10,284 | 8,885 |
| **Germany** | 2,932 | 2,878 | 5,947 | 5,870 |
| **Greece** | 0 | 0 | 0 | 330 |
| **Ireland** | 0 | 845 | 919 | 1,075 |
| **Italy** | 12,417 | 9,543 | N/A | N/A |
| **Netherlands** | 0 | 2,889 | 2,556 | 2,195 |
| **Portugal** | 259 | 260 | 274 | 91 |
| **Spain** | 3,222 | 4,887 | 5,223 | 5,705 |

* A minority of human cases confirmed in local laboratories are referred to the national reference centre and reported internationally

Table 4. Relative distribution of the common serotype and phage types – **annual average 1995-1998**

| EU 95-98 | *Salmonella Enteritidis* | | *Salmonella typhimurium* | | *Salmonella virchow* | *Salmonella hadar* | Other serotypes | Total |
|---|---|---|---|---|---|---|---|---|
| | (all) | % PT4 | (all) | % DT104 | | | | |
| Austria | 7,615 | 69 | 435 | 29 | 200 | 165 | 805 | 9,220 |
| Belgium | 6,800 | | 3,300 | | 160 | 515 | 1,525 | 12,295 |
| Denmark | 2,220 | | 745 | | 50 | 600 | 490 | 3,565 |
| England & Wales | 18,605 | 69 | 5,065 | 62 | 1,175 | 595 | 3,985 | 29,425 |
| Finland | 1,115 | | 485 | | 90 | 95 | 1,035 | 2,810 |
| France | 2,420 | | 2,515 | | 620 | 805 | 2,885 | 9,245 |
| Germany | 1,900 | 78 | 1,505 | 40 | 35 | 75 | 890 | 4,405 |
| Greece | 175 | | 70 | | 1 | 5 | 75 | 325 |
| Ireland | 190 | | 515 | | 15 | 5 | 225 | 945 |
| Italy | 4,325 | | 2,540 | | 80 | 55 | 3,985 | 10,980 |
| Luxembourg | 180 | | 70 | | 5 | 5 | 45 | 305 |
| The Netherlands | 1,190 | | 805 | | 45 | 55 | 520 | 2,610 |
| Portugal | 140 | | 35 | | 2 | 3 | 35 | 220 |
| Scotland | 1,870 | 72 | 600 | 62 | 120 | 65 | 330 | 2,980 |
| Spain | 2,070 | 27 | 1,520 | 18 | 125 | 280 | 760 | 4,760 |
| Sweden | 2,230 | 41 | 470 | | 230 | 155 | 1,475 | 4,565 |

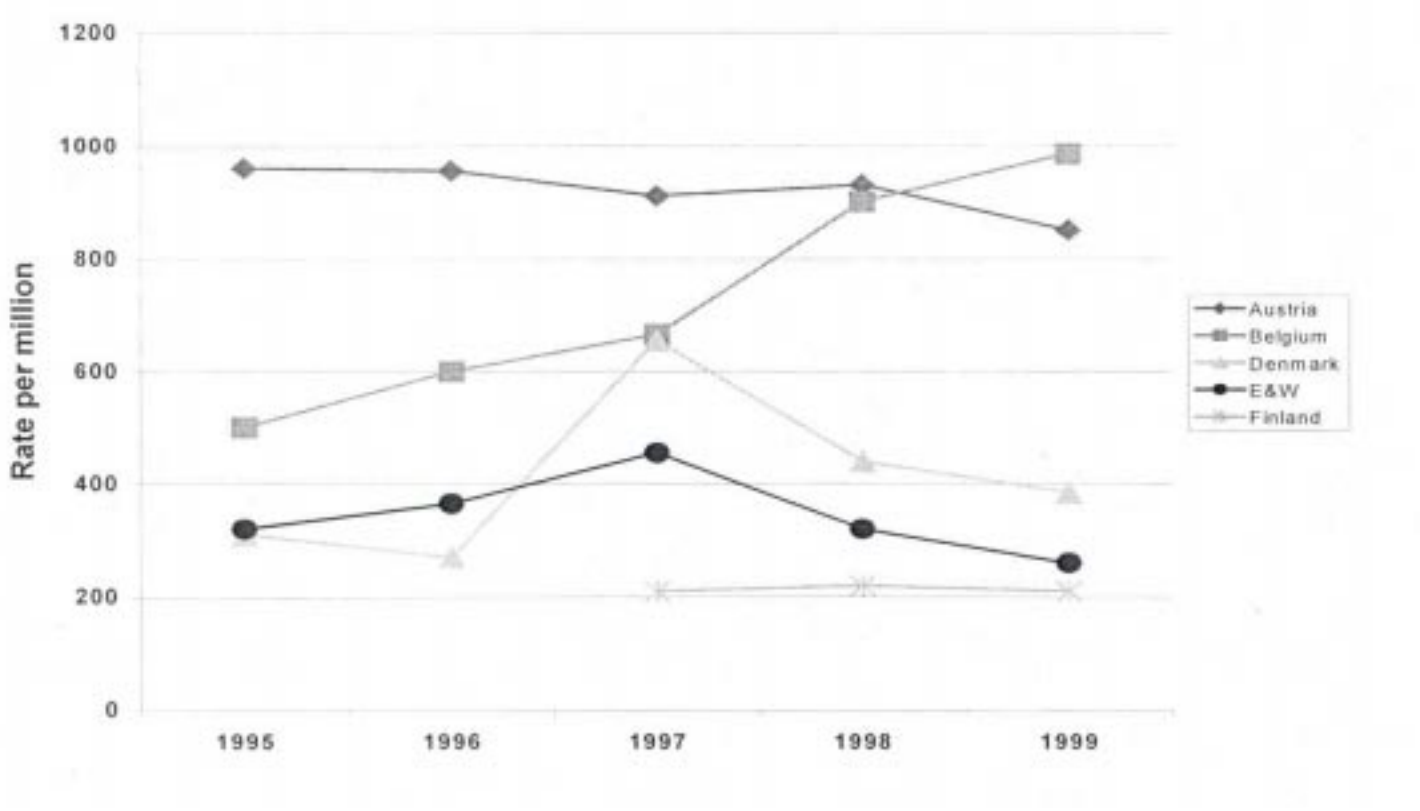
Table 5. Trends in *S. enteritidis* reports

| | | 1995 | 1996 | 1997 | 1998 | 1999* |
|---|---|---|---|---|---|---|
| **Austria** | Number | 7,761 | 7,820 | 7,361 | 7,524 | 6,904 |
| | Rate | 960 | 955 | 910 | 930 | 850 |
| **Belgium** | Number | 5,098 | 6,111 | 6,803 | 9,178 | 10,036 |
| | Rate | 500 | 600 | 670 | 900 | 985 |
| **Denmark** | Number | 1,633 | 1,437 | 3,477 | 2,341 | 2,033 |
| | Rate | 310 | 270 | 655 | 440 | 385 |
| **England & Wales** | Number | 16,290 | 18,541 | 23,227 | 16,371 | 13,192 |
| | Rate | 320 | 365 | 455 | 320 | 260 |
| **Finland** | Number | 0 | 0 | 1,090 | 1,138 | 1,081 |
| | Rate | | | 210 | 220 | 210 |
| **Luxembourg** | Number | 0 | 0 | 185 | 175 | 211 |
| | Rate | | | 465 | 440 | 530 |
| **Scotland** | Number | 1,748 | 2,057 | 2,319 | 1,352 | 1,169 |
| | Rate | 350 | 410 | 465 | 270 | 235 |
| **Sweden** | Number | 1,727 | 2,274 | 2,532 | 2,395 | 2,554 |
| | Rate | 195 | 260 | 290 | 270 | 290 |

Rate per million population

*1999 reports include an estimate for October to December

Figure 1. Incidence of *S. enteritidis* in selected countries 1995-99*



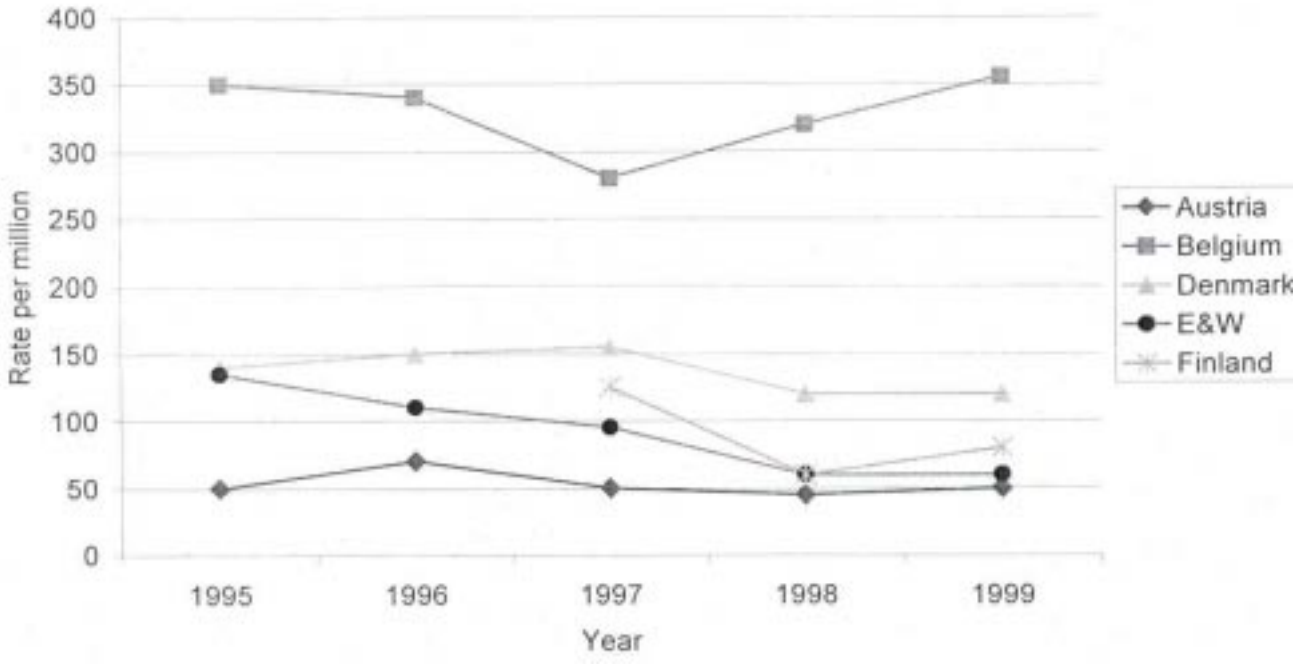

Sweden not shown due to high levels of travel association, for clarity, Scotland is not shown due to similar profile to England and Wales, and Luxembourg not shown due to low actual numbers.

Table 6. Trends *in S. typhimurium* reports

| | | 1995 | 1996 | 1997 | 1998 | 1999* |
|---|---|---|---|---|---|---|
| **Austria** | Number | 407 | 559 | 392 | 380 | 425 |
| | Rate | 50 | 70 | 50 | 45 | 50 |
| **Belgium** | Number | 3,577 | 3,476 | 2,865 | 3,285 | 3,606 |
| | Rate | 350 | 340 | 280 | 320 | 355 |
| **Denmark** | Number | 742 | 791 | 824 | 630 | 638 |
| | Rate | 140 | 150 | 155 | 120 | 120 |
| **England & Wales** | Number | 6,769 | 5,641 | 4,862 | 3,030 | 3048 |
| | Rate | 130 | 110 | 95 | 60 | 60 |
| **Finland** | Number | 0 | 0 | 652 | 325 | 419 |
| | Rate | 0 | 0 | 125 | 60 | 80 |
| **Luxembourg** | Number | 0 | 0 | 72 | 70 | 52 |
| | Rate | 0 | 0 | 180 | 170 | 130 |
| **Scotland** | Number | 714 | 744 | 555 | 375 | 288 |
| | Rate | 145 | 150 | 110 | 75 | 60 |
| **Sweden** | Number | 506 | 455 | 473 | 450 | 611 |
| | Rate | 60 | 50 | 55 | 50 | 70 |

Rate per million population

Figure 2. Incidence of *S. typhimurium* in selected countries 1995-99*




*1999 reports include an estimate for October to December

Sweden not shown due to high levels of travel association, for clarity, Scotland is not shown due to similar profile to England and Wales, and Luxembourg not shown due to low actual numbers.