The present state of foodborne disease in OECD countries

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I. INTRODUCTION

Foodborne disease (FBD) has emerged as an important and growing public health and economic problem in many countries during the last two decades. Frequent outbreaks caused by new pathogens, the use of antibiotics in animal husbandry and the transfer of antibiotic resistance to human, as well as the ongoing concerns about bovine spongiform encephalitis (BSE) are just a few examples. Countries with reporting systems have documented significant increases in the incidence (number of cases) of FBD during the two last decades. The significance of these increases is discussed later. It is estimated that each year FBD causes approximately 76 million illnesses, 325,000 hospitalizations, 5,000 deaths in the USA and 2,366,000 cases, 21,138 hospitalizations, 718 deaths in England and Wales (Adak et al*.,* 2002; Mead et al*.,* 1999). It can be assumed, from the reported number of cases, that the burden of FBD is probably in the same order of magnitude in most countries of the Organisation for Economic Co-operation and Development (OECD).

Contamination of foods may occur through environmental pollution of the air, water and soil, such as the case with toxic metals, polychlorinated biphenyls (PCBs) and dioxins. Other chemical hazards, such as naturally occurring toxicants, may arise at various points during food production, harvest, processing and preparation. The contamination of food by chemical hazards is generally well controlled in OECD countries although such hazards remain a public health concern to many consumers. The safe use of various chemicals such as food additives, pesticides, veterinary drugs and other agro-chemicals is also largely assured in OECD countries by proper regulation, enforcement and monitoring. However, sporadic problems with chemical hazards continue to occur pointing to the need for constant vigilance with regard to both the levels of chemicals in the diet as well as their potential to cause adverse health effects in the population.

II. WHAT WE KNOW

Severity of foodborne disease

Foodborne disease caused by microorganisms

Foodborne disease is a public health problem which comprises a broad group of illnesses. Among them, gastroenteritis is the most frequent clinical syndrome which can be attributed to a wide range of microorganisms, including bacteria, viruses and parasites. Usually, the incubation period is short, from 1-2 days to 7 days. Different degrees in severity are observed, from a mild disease which does not require medical treatment to the more serious illness requiring hospitalization, long-term disability and/or death {hospitalization rates from 0.6% to 29% and case-fatality rates up to 2.5% in the USA (Mead et al*.,* 1999)}. The outcome of exposure to foodborne diarrhoeal pathogens depends on a number of host factors including pre-existing immunity, the ability to elicit an immune response, nutrition, age and non-specific host factors. As a result, the incidence, the severity and the lethality of foodborne diarrhoea is much higher in some particularly vulnerable segments of the population, including children under five years of age, pregnant women, immunocompromised people (patients undergoing organ transplantation or cancer chemotherapy, AIDS...) and the elderly (Gerba et al*.,* 1996). In addition to these well-known predisposing conditions, new ones are regularly identified {liver disease for *V. paraheamoliticus* septicaemia, thalassemia for *Yersina enterocolitica* infections (Hlady et al*.,* 1996; Adamkiewicz et al*.,* 1998)}. Serious complications may result from these illnesses including intestinal as well as systemic manifestations, like haemolytic uremic syndrome (HUS) (kidney failure and neurological disorders) for 10% of *Escherichia coli* O157:H7 infections with bloody diarrhoea, Guillain-Barré syndrome (nerve degeneration, slow recovery and severe residual disability) after C*ampylobacter jejuni* infection, reactive arthritis after

salmonellosis, and chronic toxoplasmic encephalitis (Griffin et al*.,* 1988; Rees et al., 1995; Thomson et al*.,* 1995). Several authors have estimated that chronic sequelae (long-term complications) may occur in 2% to 3% of all FBD (Lindsay, 1997).

While diarrhoea is the most common syndrome following the consumption of a contaminated food, some diseases are more serious. Clinical manifestations of listeriosis include bacteraemia and central nervous system infections, especially in patients with an impairment of T-cell mediated immunity (neonates, the elderly, immunocompromised patients) and abortion in pregnant women, with an overall case-fatality rate of 25%. Foodborne botulism is a result from the potent toxin by *Clostridium botulinum* that causes paralysis of skeletal and respiratory muscles which, when severe, may result in death in 8% of cases. In addition to the consequences of toxoplasmosis on the fetus (birth defects), *Toxoplasma gondii* is also the most frequent cause of lesion in the central nervous system in patients with AIDS. Hepatitis A is an infectious disease for which age is the most important determinant of morbidity and mortality, with severity of illness and its complications increasing with age. The duration of illness varies, but most cases are symptomatic for three weeks. Complications during the acute illness phase are unusual, with fulminant hepatitis and death being uncommon.

Foodborne disease caused by chemicals and toxins

Because the period of time between exposure to chemicals and effect is usually long, it is difficult to attribute disease caused by long-term exposure to chemicals in food to the actual food in question. This is one of the reasons why, in contrast to biological hazards, the protection of public health from chemical hazards has for a long time largely employed the risk assessment paradigm (WHO, 1999b). Essentially the risk assessment paradigm relies on estimates of potential toxicity, most often from animal studies. Exposure to chemicals in food can result in acute and chronic toxic effects ranging from mild and reversible to serious and life threatening. These effects may include cancer, birth defects and damage to the nervous system, the reproductive system and the immune system (WHO, 1996; WHO, 1999a; WHO, 2001b).

Once the hazard characterization of a chemical has been performed, estimates of exposure through the diet and other sources are necessary to assess whether there is a public health concern. Evaluation measures to assess potential harm has been focused on attaining information on the levels of chemicals in food and the diet as a whole, and national and international programmes have been developed to obtain such data (WHO, 2002). However, biomonitoring for certain chemicals may serve as a better or an additional tool in evaluation studies in the future (WHO, 1998). In addition, the use of biomarkers for exposure as well as hazard identification and hazard characterization may improve the accuracy and reliability of risk assessments of chemicals in food (WHO, 2001a).

Present state of foodborne disease in OECD countries

Foodborne disease caused by microorganisms

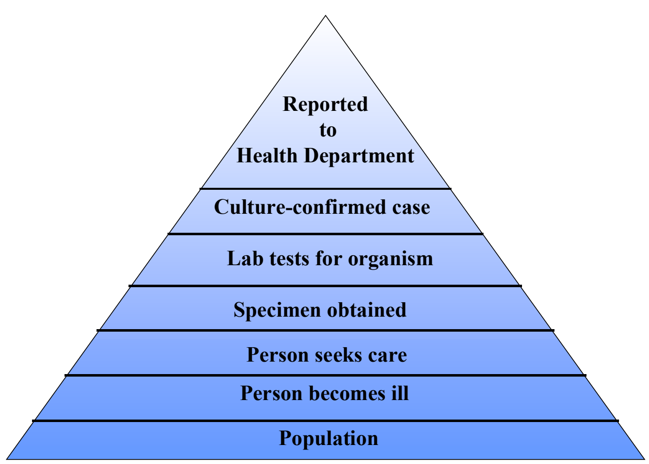
Most of the data presented in this section originate from routine surveillance[1] using a number of health information systems: mandatory notification, outbreak investigations, laboratory-based surveillance systems, sentinel surveillance, and death and hospital diagnose discharge, each of these systems having advantages and drawbacks (Borgdorff and Motarjemi, 1997). Any choice of method depends partly on the objective under consideration. For instance, one method may be very useful in the early detection of outbreaks but may have severe limitations in estimating the size of the burden of FBD. Mandatory

1. Public health surveillance is the ongoing systematic collection, analysis, and interpretation of outcome-specific data for use in the planning, implementation, and evaluation of public health practice (Thacker, 1994).

notification is widely used for FBD, however, it suffers from a number of limitations such as outbreak detection, identification of single cases of severe disease and characterization of long-term trends (Cowden, 2000). Data may vary according to surveillance systems. Although death certificates are an important source of data for determining disease burden, the limitations of mortality statistics may result in substantial biases in epidemiological studies, for example, in a study linking *V. vulnificus* infections surveillance records to death certificates, *V. vulnificus* was not reported on 55% of death certificates (Banatvala et al*.,* 1997). In a capture-recapture study, the sensitivity of three surveillance systems for *Salmonella* outbreaks in France were 10% for the mandatory notification to the National Public Health Network, 15% for the mandatory notification network of the Ministry of Agriculture and 50% for the laboratory-based systems (Gallay et al*.,* 2000). In laboratory-based systems, the reliability of data is highly dependent upon methods used for pathogen detection. For example, while *E. coli* 0157:H7 is the most well-known serotype of EHEC to be responsible for HUS, a significant percentage of cases are caused by non *E. coli* 0157:H7 in a number of countries. Difficulties in detecting these non 0157:H7 serotypes may minimize the extent of the public health problem. No comparison between surveillance systems in term of their efficiency can therefore be made in a realistic way, and subsequently, trying to compare countries data according to their surveillance systems is not informative.

Although many diseases are notifiable, compliance is often poor. Surveillance systems are traditionally passive and very exceptionally active[2] which means that underreporting is a major drawback for data analysis and interpretation. Because most people regard diarrhoea as a transient inconvenience rather than a symptom of disease, the vast majority of diarrhoeal episodes do not result in a visit to a physician, even though the person may be incapacitated for several days. In addition, for the system to function, the general practitioner must order a stool culture, the laboratory must identify the etiologic agent and report the positive results to the local or state public health institution in charge of surveillance. Information is lost at each step of this pyramid (Figure 1). Consequently, reporting of sporadic cases[3] is generally more complete for severe conditions like botulism and listeriosis than for mild disease like diarrhoea. Table 1 provides examples of underreporting factors.

Fig. 1 : The burden of illness pyramid (adapted from CDC, http//www.cdc.gov)



2. Active surveillance: surveillance where public health officers seek reports from participants in the surveillance system on a regular basis, rather than waiting for the reports (WHO, 2002b).

3. Sporadic cases: individual cases that are not linked to other known cases of illness. These sporadic cases are usually difficult or impossible to attribute to a particular source, as the possibilities are too numerous.

Table 1: Examples of underreporting factors (outbreaks and sporadic cases)

PATHOGEN / DISEASE	COUNTRIES			
	USA	Canada	UK	France
Diarrhoea	-[a]	-	136[1]	-
BACTERIA				
Aeromonas	-	-	1011.9[2]	-
Bacillus	38 (*B. cereus*)	-	237[2] (*B. spp*)	-
Brucella	14	-	-	-
Campylobacter spp.	38	-	7.6[1]/10.3[2]	-
Clostridium botulinum	2	-	-	-
Clostridium perfringens	38	-	342[2]	-
Listeria monocytogenes	2	-	2[2]	1.1
Salmonella non-typhoidal	38		3.2[1]/3.9[2]	-
Salmonella typhi	2	-	2[2]	-
Shigella	20	-	3.4[2]	-
Staphylococcus aureus	38	-	237[2]	-
Vibrio cholerae	2	-	2[2]	-
Vibrio vulnificus	2	-	-	-
VTEC[b]	20	4-8	2[2]	-
Yersinia enterocolitica	38	-	1,2543[2]	-
PARASITES		-		
Cryptosporidium parvum	45	-	7.4[2]	-
Cryptosporidium cayatenensis	38	-	26.9[2]	-
Cyclospora	-	-	38[2]	-
Giardia	20	-	4.6[2] (*G. duodenalis*)	-
Trichinella spiralis	2	-	-	-
VIRUSES				
Astrovirus	-	-	721.3[2]	
Norovirus	-	-	1,562[1]/275.5[2]	-
Rotavirus	-	-	35[1]/21.5[2]	-
Hepatitis A virus	3	-	-	
References	Mead et al., 1999	Michel et al., 2000	[1]Wheeler et al., 1999 [2]Adak et al., 2002	Goulet et al., 2001

[a]: no information; [b]: *E. coli* O157 only.

In addition to being an important focus for public health intervention, outbreaks[4] and their investigation are unique events which allow the collection of important data. Such data can add to the knowledge of the natural history of different pathogens, the vehicles of illness, and the common or novel errors that contribute to outbreaks. They are a fundamental source of information to design food safety policies, sometimes the only one when little investigation of sporadic cases is performed. Finally, outbreaks involving less commonly identified microorganisms or with longer incubation periods are less likely to be confirmed, whereas pathogens that usually cause mild illness will be underrepresented. Outbreak reports are frequently deficient because of late notification, unavailability of clinical specimens and/or food samples, unsuitability of laboratories or methods to detect and identify the pathogen, insufficient resources and trained staff to conduct investigations, lack of cooperation between the different disciplines, or failure of investigators to write the final report (Guzewich et al., 1997).

4. Foodborne outbreak: a foodborne outbreak is defined by the occurrence of a similar illness among two or more people which an investigation linked to consumption of a common meal or food items, except for botulism (one case is an outbreak).

Because routine surveillance systems vary widely between diseases and between countries, the collected information presented here does not allow numerical comparison of data on foodborne disease between countries and diseases. A higher number of reported cases can be the result of a well performing surveillance system and not necessarily that people are more often sick from contaminated food. In addition, the reported number of cases for a country can include cases acquired domestically as well as acquired abroad after travel. Finally, no geographical spread of FBD can be inferred from these data, except when differences in food consumption are well known.

Tables 1 and 2 of the annex summarize reported annual incidence of diseases caused by foodborne pathogens (outbreak and sporadic cases) for a specific year selected between 1998 and 2001 in OECD countries (collected through bibliographic databases, internet and by personal communications). This data has been compiled through a limited-time search of data from open literature. It does not represent a formalized enquiry to the relevant authorities in countries affected. Therefore it is plausible that national data not readily available through open international sources has not been included in the tables. A higher number of cases are reported for bacterial agents than parasitic or viral agents. It cannot be assessed whether this reflects the true proportion of cases, higher public health priority, increased interest from epidemiologists and microbiologists, or the present state of laboratory ability to detect and investigate pathogens. However, the incidence of viral diseases seems to be underestimated since a number of specific studies indicate a very substantial portion of FBD in many OECD countries are of viral etiology (causes) (De Wit et al., 2000; Hedlund et al*.,* 2000).

Briefly, data from tables 1 and 2 of the annex indicate that non-typhoidal salmonellosis is the only FBD reported in all countries, with an annual reported incidence rate ranging from 6.2 to 137 cases per 100,000 population with the exception of three countries with much higher values. Campylobacteriosis, when under routine surveillance, appears to be one of the most frequent bacterial FBD in many countries, with reported annual incidence rates up to 95 cases per 100,000 population. For other bacterial FBD, reported annual incidence rates are lower: between 0.2 and 19.9 cases per 100,000 population for shigellosis, 0.01 and 14 cases per 100,000 population for yersiniosis, between 0.03 and 10.4 cases per 100,000 population for VTEC *E. coli* infections, between 0.01 and 0.5 case per 100,000 population for listeriosis, between 0.01 and 1.6 cases per 100,000 population for botulism. Despite the incidence of brucellosis being very low in a number of countries (less than 0.5 cases per 100,000 population), the disease is still endemic in some Mediterranean and Eastern countries of Europe (FAO/WHO, 2002c). For various reasons, most viral and parasitic FBD are inconstantly recorded, except hepatitis A whose annual incidence rates vary from 1.2 to 22.3 cases per 100,000 population.

It should be noted that aggregating data at the national level may not reflect the exact situation. For example, in the USA, data from FoodNet is recorded by State and accordingly indicate variations in incidence of these diseases as well as variations in *Salmonella* serotypes (FoodNet, 2000). Similarly, while the incidence rate of brucellosis is very low in the USA, a higher incidence in California was the starting point of further investigation which demonstrated that during the last decade brucellosis has dramatically changed from being an occupational illness of adult men exposed to livestock or contaminated carcasses in packing and rendering plants to a foodborne illness with a high proportion of Hispanics who were more likely to report being infected by consumption of milk and cheese in Mexico (Chomel et al*.,* 1994).

Surveillance data on most FBD usually include both sporadic and outbreak cases, except for illness caused by *Staphylococcus aureus*, *Clostrium perfringens* and *Bacillus cereus* (only outbreaks are reported due to the nature of the disease). FBD outbreaks can be geographically limited (point-source outbreaks[5]) involving a rather small number of cases or spread over a large geographical area, even internationally, with sometimes a huge number of cases. Some bacterial pathogens generate high numbers of outbreaks,

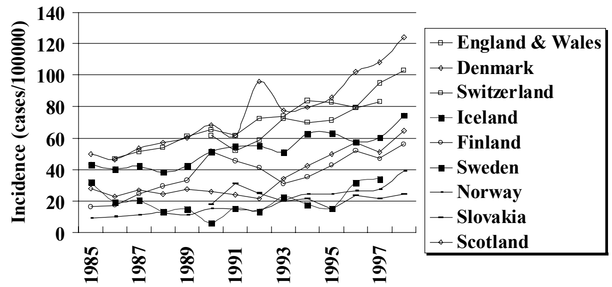
5. Point source outbreak: a localized increase in the incidence of a disease linked to a family or community event (WHO, in press).

like non-typhoidal *Salmonella*. In 1995, 757 salmonellosis outbreaks were estimated in France, a figure which could be as high as 2,000 in reality (Gallay et al., 2000). In the USA, although the incidence of typhoid fever has been very low since the 1940s, *Salmonella* typhi continues to cause outbreaks: 60 outbreaks were reported from 1960 to 1999; of the 36 outbreaks in which the transmission route was identified, 26 (72%) were foodborne, 6 (17 %) were attributed to contaminated water and ice and 4 (11 %) were attributed to either food or water (Olsen et al., 2003). In contrast, *Campylobacter* is the most commonly recognized bacterial cause of gastrointestinal infections in a number of countries but there are few reported outbreaks of campylobacteriosis. For example, among the 2,374 outbreaks reported in UK between 1995 and 1999, *Campylobacter* accounted for only 2% (Frost et al., 2002). Similarly, while outbreaks caused by *V. parahaemolyticus* are frequent, they are rare for *V. vulnificus* (EC, 2001a). Regarding viruses, a recent compilation of data from ten surveillance systems in Europe found Norovirus (Norwalk and Norwalk-like viruses) to be responsible for more than 85% of all non-bacterial outbreaks of gastroenteritis reported from 1995 to 2000 (Lopman et al., 2003). Norovirus was the etiologic agent of 284 outbreaks in the USA between 1997-2000 and in 455 outbreaks in Sweden between 1994-1998 (Fankhauser et al., 2002; Heldlund et al., 2000). In Minnesota Norovirus is the leading cause of outbreaks with 85 outbreaks occurring between 1990-1998, followed by *C. perfringens* with 22 outbreaks and *Salmonella* with 21 outbreaks (Deneen et al., 2000). Similarly, most nonbacterial gastroenteritis outbreaks in paediatric cases in Japan are caused by Norovirus (Inouye et al., 2000).

Seasonal variations in FBD are also observed; a peak in bacterial disease incidence occurs during summer probably because time/temperature abuse allows bacterial pathogens to grow in food (Anonymous, 2001c, 2001; Gerber et al., 2002; Lee et al., 2001). In addition, a nationwide case-control study on acute diarrhoea in summer in France demonstrated that living away from the main residence and returning from a country at high risk were the two major risk factors (Yazdanpanah et al., 2000). For *V. parahaemolyticus* and *V. vulnificus* infections, data suggests that water temperature is an important factor in the epidemiology of the disease (Daniels et al., 2000; Obata and Mozumi, 2001; Shapiro et al., 1998). In contrast, a weaker seasonality was observed for foodborne outbreaks caused by Norovirus in England and Wales, 1992-2000 (Lopman et al., 2003).

Data from a number of countries indicates that the incidence of FBD of known etiology has considerably increased during the past two decades. This is probably mainly a result of the increased reported number of cases caused by *Campylobacter* and *Salmonella*, especially because of *S.* Enteritis pandemic (Rodrigue et al., 1990). In Europe for example, a tremendous increase in the number of cases of non-typhoidal salmonellosis was observed, with a peak being reached in 1992 for a number of countries. Similarly, reports on campylobacteriosis have been continuously increasing in this region since 1985 and this disease is currently the most commonly reported gastroenteritis in many countries. It is often argued that it is unclear whether improvement in diagnosis and surveillance systems could explain part of this rise for campylobacteriosis (FAO/WHO, 2002c). However, a study in New Zealand demonstrated that changes in laboratory techniques were insufficient to account for a marked increase in *Campylobacter* isolations. On the basis of data provided by 12 laboratories, the number of specimens that grew *Campylobacter* increased by 49% between 1992 and 1993 (McNicolas et al., 1995). (Figure 2).

Fig 2: Annual incidences of campylobateriosis in European countries



Source: WHO Surveillance Programme for Control of Foodborne Infections and Intoxications in Europe

Foods most frequently involved in outbreaks in OECD countries are meat and meat products, poultry, eggs and egg products, with the likely implication of these foods being associated with *Salmonella* and *Campylobacter* (Table 3[6] of the annex, Michino and Otsuki, 2000). Case-control studies confirmed the same food sources for sporadic cases: raw and undercooked eggs, egg containing food and poultry for salmonellosis (Cowden et al*.,* 1989; Delarocque-Astagneau et al*.,* 1998; Hedberg et al*.,* 1993; Kapperud et al*.,* 1998; Schmid et al., 1996), poultry for campylobacteriosis (Effler et al*.,* 2001; Kapperud et al*.,* 1992; Studahl and Andersson, 2000) and raw oyster for *Vibrio* illness (Desenclos et al*.,* 1991). Reflecting food habits and way of life, places where the implicated outbreak vehicle is prepared or eaten vary between OECD countries, with a predominance of home or outside of home settings (Table 2[6] of the annex: Daniels et al*.,* 2002; Fankhauser et al*.,* 2002; Lee et al*.,* 2001; Levine et al*.,* 1991; Przybylska, 2001; Ryan et al*.,* 1997). Eating food outside the home or food prepared by commercial food establishments were also found to be risk factors for sporadic cases of salmonellosis and campylobacteriosis in some countries (Cowden et al*.,* 1989; Effler et al*.,* 2001). Three main groups of factors can contribute to outbreaks (related to contamination, to survival of microorganisms and related to microbial growth). Data on these factors in OECD countries are shown in Table 5[6] of the annex. From the available data, time/temperature abuse appears to be the most frequent contributing factor in many OECD countries.

Foodborne disease caused by chemicals and toxins

A significant portion of human cancers may relate to dietary factors, including both exogenous and endogenous mutagens. Of exogenous factors, certain metals and certain pesticides (both naturally produced or manufactured by the chemical industry), N-nitroso compounds, heterocyclic amines, and polycyclic aromatic hydrocarbons are all probable human carcinogens (Ferguson, 1999).

Similarly, a large number of pregnancies result in prenatal or postnatal death or an otherwise less than healthy baby (ICBD, 1991; CDC, 1995; Holmes, 1997; March of Dimes, 1999). Exposure to toxic chemicals, both manufactured and natural, cause about 3% of all developmental defects, such as neural tube and heart deformities, and at least 25% might be the result of a combination of genetic and

6. This data has been compiled through a limited-time search of data from open literature. It does not represent a formalized enquiry to the relevant authorities in countries affected. Therefore it is plausible that national data not readily available through open international sources has not been included in the tables.

environmental factors. These estimates might be higher if complete data were available on the developmental toxicity of the many untested chemicals that are currently being used (NAC, 2000).

In a recent study of EU countries, the number of samples for which residues of pesticides in food exceeded the corresponding maximum residue limits was 4.3 percent (EC, 1999). While this increasing trend in the number of violative samples is worrisome, the more significant public health concern is the high levels of certain pesticides, which may produce acute adverse health effects. In particular, developmental and reproductive effects are of concern because these can be caused by single exposures to high levels of pesticides. Long-term, low-dose exposure to organophosphorus compounds lowers the threshold for acute poisoning from such insecticides. Documented effects in humans of pesticides include male sterility, neuro-behavioural disorders, proliferative lung disease and allergenic sensitization (WHO/UNEP, 1990).

Because diets in OECD countries contain relatively large amounts of processed foods, contaminants that appear in such foods pose particular risk to these populations. Polycyclic aromatic hydrocarbons, many of which are known human carcinogens, have been found in smoked foods, grilled meats and heat-recovered oils. More recently, the presence of the suspected human carcinogen acrylamide was discovered in a wide range of processed food products (FAO/WHO, 2002b). Further the collection of further information on the nature and extent of the risk posed by acrylamide is coordinated internationally by WHO in collaboration with FAO and the Joint FDA/UMD Institute for Food Safety and Applied Nutrition (FAO/WHO/JIFSAN Infonet, 2002).

Accidental or intentional adulteration of food by toxic substances has resulted in serious public health incidents in both developing and industrialized countries. For example, in Spain in 1981-82, adulterated cooking oil killed some 600 people and disabled another 20,000, many permanently with neurotoxic disorders. In this case, the agent responsible was never identified in spite of intensive investigations (WHO, 1992).

Increase in reported foodborne disease incidences

The last two decades have been characterized by a number of developments which can help to explain the increase in the reported number of cases in a number of countries. It should be noted that for some pathogens (notably some *Salmonella* serovars) action taken at the national level, mainly at the production level, has resulted in a recent decrease in the incidence of disease from these pathogens in some countries.

New conditions for the emergence of pathogens

While no good overview of the relative importance of these factors exists, a number of factors can be suggested to explain the emergence of new foodborne pathogens as well as the re-emergence of well known pathogens over the last two decades:

<u>New feeding practices</u>: While the initial cause of the emergence of BSE remains unknown, the ultimate driving force of the epidemic has been identified. The establishment of BSE in its new bovine host and subsequent epidemic spread has been clearly linked to the use of meat and bone meal from cattle and other ruminant carcasses in the preparation of cattle feed. From the initial cases detected in 1986, the epidemic spread to infect over 178,000 head of cattle in over 35,000 herds in UK. In 1996, another new disease, variant Creutzfeldt-Jakob disease, was detected in humans and linked to the BSE epidemic in cattle. Consumption of contaminated meat products from cattle is presumed to be the cause (WHO, 2002 c).

<u>Change in animal husbandry</u>: Modern intensive animal husbandry practices introduced to maximize production seem to have led to the emergence and increased prevalence of *Salmonella* serovars and/or *Campylobacter* in herds of all the most important production animals (poultry, cattle, pig). For example, in

the USA, in 1969 470,832 layer-hen farms with an average of 632 hens per farm produced 67 billions eggs per year; by 1992, the number of farms dropped by 85 % to 70,623, the number of hens per farm increased by 470 % to 2,985 and annual production rose to 70 billion eggs (Sobel et al*.,* 2002). In addition, the conditions and stress associated with transporting animals to slaughter and dietary changes prior to slaughter can increase carriage rates and shedding (WHO, 2001).

Changes in agronomic process: The use of manure rather than chemical fertilizers, as well as the use of untreated sewage or irrigation water containing pathogens undoubtedly contributes to the increased risk associated with fresh fruit and vegetables, especially in countries where an important increase in consumption of such products occurred in recent years (Beuchat and Ryu, 1997). The major *E.coli* O157:H7 outbreak (more than 9,000 cases) in Japan in 1996 as well as recent observation of *Cyclospora* infection outbreaks in North America and Germany are typical examples (Bern et al*.,* 1999; Döller et al*.,* 2002; Hideshi et al*.,* 1999).

Increase in international trade: This has three main consequences : i) the rapid transfer of microorganisms from one country to another, ii) the time between processing and consumption of food is increasing, leading to increased opportunity for contamination and time/temperature abuse of the products and hence the risk of foodborne illness, and iii) the population is more likely to be exposed to a higher number of different strains/types of foodborne pathogens.

Changes in food technology: Advances in processing, preservation, packaging, shipping and storage technologies on a global scale have enabled the food industry to supply a greater variety of foods, especially ready-to-eat foods. The increased use of refrigeration to prolong shelf-life has contributed to the emergence of *Listeria monocytogenes* (Rocourt and Cossart, 1997).

Increase in susceptible populations: Advances in medical treatment have resulted in an increasing number of the elderly and immunocompromised people. In many industrialized countries, the absolute number of the elderly is rapidly increasing. Studies of foodborne outbreaks in nursing homes illustrate the potential severity of FBD in institutions for the elderly, with a higher case-fatality rate than for outbreaks occurring in other settings (Levine et al*.,* 1991; Mishu et al*.,* 1994). Similarly, the population of patients with AIDS is rapidly increasing. These patients show a clear increase in susceptibility to *Salmonella* (relative risk of infection increased by 20-100) and to *Campylobacter* (35-fold increase in relative risk), as well as an increased risk of more severe clinical manifestations (Morris and Potter, 1997). While *Toxoplasma gondii* was before primarily of concern because of congenital infections, it is now a leading cause of cranial lesions in persons with AIDS (Garly et al., 1997). It is estimated that around 20% of the population of industrialized countries are at higher risk of FBD as a result of some sort of immune-suppression(Gerba et al*.,* 1996).

Increase in travel: Globalization of FBD results also from increased travel. Five million international arrivals were reported worldwide in 1950 and this number is expected to increase to 937 million by 2010. As a result, a person can be exposed to a foodborne illness in one country and expose others to the infection in a location thousands of miles from the original source of infection. Depending on their destination, travellers are estimated to run a 20% to 50% risk of contracting foodborne disease (Käferstein et al*.,* 1997). For example, 90% of salmonellosis in Sweden, 71% of typhoid fever cases in France, 61% of cholera cases in the USA are attributed to international travel (Anonymous, 2001c; Schlosser and Cervantes, 1998; Steinberg et al., 2001).

Change in lifestyle and consumer demands: Previously unrecognized microbial hazards have emerged as a result of changes in food consumption, like the increasing consumption of fresh fruit and vegetables in a number of countries. While dining in restaurants and salad bars was relatively rare 50 years ago, they are today a major source of food consumption in a number of OECD countries. As a result, an increasing number of outbreaks are associated with food prepared outside the home (Table 4). In addition, the recent

interest of consumers in foreign cooking can be an unexpected source of FBD in a geographical area {like an outbreak of ciguatera in France (Vaillant et al*.,* 2001)}.

Unusual features of new pathogens

New pathogens have been recognized as predominantly foodborne in the last two decades, either newly described pathogens or newly associated with foodborne transmission: *Salmonella* Enteritidis, *Campylobacter*, VTEC *E. coli*, *Listeria monocytogenes*, Noroviruses, *Vibrio cholerae* O1*, V. paraheamoliticus, V. vulnificus*, *Yersinia enterocolitica, Cyclospora* and prions. Salmonellosis caused by the serotypes Enteritidis and campylobacteriosis are the two most frequent diseases in many OECD countries. Listeriosis, VTEC *E. coli* infections and the new variant Creuzfeld-Jacob disease are very severe illnesses. In addition, antimicrobial resistant strains, like quinolone-resistant *Campylobacter or S.* Typhimurium DT104 - a strain resistant to five antibiotics. *S.* Typhimurium DT104 has shown a rapid national and international spread in the 1990's - probably largely because of the widespread use of antibiotics in the animal reservoir (Aarestrup et al*.,* 1998; Smith et al*.,* 1999). A new, highly multi-resistant *Salmonella* Newport strain (resistant to nine antimicrobials, including some of the most important new antimicrobials) emerged in the USA in 1999 and now seems to have spread to many parts of the USA (Angulo, 2002); in some ways the spread of this strain seems to mimic the earlier spread of DT104. It is likely that new foodborne pathogens will regularly emerge in the future given the high percentage of cases of undetermined etiology.

Most of these new pathogens have an animal reservoir but they do not often cause illness in the infected animal (chicken and *S.* Enteritidis, calf and *E. coli* O157:H7*, V. vulnificus* and Norwalk viruses and oysters, *Listeria monocytogenes* and various animals produced for food). Therefore, these new foodborne hazards often escape traditional food inspection systems, often relying on the presence of visual signs of disease. It is thus important to realize that these foodborne diseases require new food control strategies.

These characteristics, associated with changes in food production and distribution have generated a new outbreak scenario. Traditional outbreaks were characterized by an acute and locally limited number of cases, with a high inoculum dose and a high attack rate sometimes because of a foodhandler error in a small kitchen shortly before consumption, often after a social event. In contrast, new outbreaks are often spreading over a wide geographic area involving different parts of a country or even internationally with a potentially high number of patients involved. The originating event can be a low-level contamination of a widely distributed food, often industrially processed. In these cases food contamination is not the result of a terminal foodhandling error but the consequence of an event in the early stages of the food-chain. Investigation and prevention of such outbreaks can have serious implications for the food industry (Tauxe, 1997; 2001). The ice cream associated salmonellosis outbreak of the USA in 1994 which involved more than 224,000 patients or the extensive outbreak of staphylococcus intoxication in Japan which affected 13,420 people are typical examples of this new kind of outbreak (Hennessy et al*.,* 1996; Asao et al*.,* 2003).

Modification of surveillance systems and additional epidemiological studies

These new pathogens prompted several new surveillance approaches to provide more information. In the USA, FoodNet is a network of nine sentinel sites conducting active surveillance for a number of foodborne pathogens. It measures the burden of illness, determines the source of infections through large case-control studies of sporadic cases and evaluates the impact of control measures on these infections (Tauxe, 2001). FoodNet also conducts studies of the population at large on diarrhoeal disease. In the UK and in the Netherlands, studies aiming at assessing the true incidence of diarrhoeal disease have been undertaken (De Witt, 2000a and 2001a,b; Wheeler et al., 1999). Enter-Net was created in 1994 as a European Union initiative. It is an international network for the surveillance of human intestinal infections, which monitors salmonellosis and VTEC *E. coli* infections, including antimicrobial resistance (Fisher, 1999). In Denmark a national system to monitor the developments in antimicrobial resistance (DANMAP) was initiated in

1995, and such systems are now being initiated in other European countries (Aasrestrup et al*.,* 1998). Similarly the National Antimicrobial Resistance Monitoring System (NARMS) in the U.S. monitors antimicrobial resistance by testing a representative sample of isolates of major foodborne pathogens. It has provided early warning for the appearance of *Salmonella* strains resistant to drugs critical in human infection treatment (Tauxe, 2001). The capacity of surveillance to detect widespread outbreaks in the USA has been dramatically improved in recent years with PulseNet, a national molecular subtyping network of foodborne pathogens. PulseNet is able to compare online results of different laboratories with each other and with a nationwide database. When a cluster is flagged, a detailed epidemiological investigation can often determine the source (Swaminathan et al*.,* 2001).

Concurrent to these initiatives, traditional surveillance systems were strengthened in a number of countries by various means (Anonymous, 2001c, 2001; Hutwagner et al., 1997; Scuderi and Gabriella, 2000). While 164 outbreaks were notified in France in 1987, this number had doubled in 1989, partly because of efforts to strengthen this notification (Hubert et al*.,* 1990). Similarly, the increase in foodborne outbreaks observed after 1992 in the UK might have been due in part to improved notification by general practitioners (Wall et al*.,* 1996). The same period of time, was characterized by the application of molecular methods to detect and characterize microorganisms which introduced new means for laboratory-based surveillance systems (Swaminathan and Matar, 1993). This can be illustrated with the introduction of PCR (Polymerase Chain Reaction)-based methods and Norovirus. The primary reason for the under-appreciation of the disease burden has been the difficulty in developing and applying sensitive and easy to perform diagnostic assays (the virus cannot be cultivated from clinical samples, no animal models are available to study the virus, the primary diagnostic methods until recently were electron microscopy and serological assays) (Bresee et al*.,* 2002). Because of changes in reporting systems during the last two decades, data should be analyzed and interpreted very carefully regarding incidence trends. However, a clear increase in the incidence of a number of FBD in some OECD countries has been observed during the two last decades, even if this increase is, in some countries, and to some extent, not measurable, related to surveillance and laboratory testing improvement.

Success in the reduction of foodborne disease caused by microorganisms

Success in FBD incidences declining have been mainly the result of a limited number of interventions, especially at the production level, for a limited number of pathogens in a limited number of countries. Sanitation and the decrease of typhoid fever, milk pasteurization and the decrease in tuberculosis, canning and the decrease in botulism, and herd vaccination and the decrease in brucellosis illustrate very well the impact of appropriate prevention measure implementation on public health (Lyndt et al.; Tauxe, 1997). While these measures were able to drastically reduce the incidence of a specific disease, the complex interactions between new pathogens and the food-chain suggest that future successful reduction strategies will often need to be much more sophisticated. In spite of these new difficulties, a number of recent initiatives have been associated with a clear reduction in incidence of FBD.

To control *Salmonella* in poultry, a compulsory programme was implemented in Sweden of control and quarantine of grandparent stock and pre-slaughter control of broilers. Control in relation to parent stock, hatcheries and layers continues to be voluntary, but mandatory testing of layers during production and before slaughter has been required since 1994 (Mulder and Schlundt, 1999). As a result, the incidence of domestic cases is very low, 5 cases per 100,000 in 1998, i.e. 10% of the reported cases (Anonymous, 2001c). Similarly, a sharp decrease in the number of salmonellosis cases was recently reported in England and Wales following the introduction of a vaccination programme against *Salmonella* Enteritidis in chicken by the British poultry industry (Adak et al*.,* 2002).

In the period 1988 to 2000 Danish authorities initiated a series of action plans to control human salmonellosis through initiatives primarily at farm level. Following peaks of human salmonellosis caused by serotypes related to pigs (1988), chicken (1993) and eggs (1997) such action plans were successful in

reducing salmonella prevalence at the farm level and the resulting human disease burden (Figure 3) (H.C. Wegener, personal communication and Hald and Wegener, 1999). It is interesting to note that measurement of success in these cases was only possible through centrally managed typing regimes (primarily phage typing) of strains from the whole food-chain and human isolates, enabling a 'pathogen-account' system attributing fraction of human disease to foods.

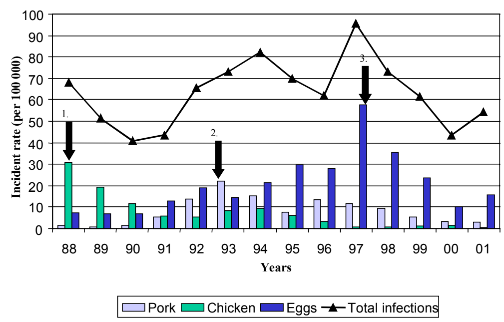


Fig 3: Salmonellosis in Denmark 1988-2001 (H.C. Wegener, personal communication)

Following an increase in the incidence of campylobacteriosis in Iceland, interventions consisting of an educational programme for farmers, an extensive surveillance programme for *Campylobacter* in poultry, freezing all *Campylobacter*-positive flocks before they go to retail and extensive consumer education were implemented in 2000. Very preliminary data indicate a decrease in the incidence of human cases (FAO/WHO, 2002a; Stern et al*.,* 2003).

A sharp decrease in the incidence of listeriosis was observed in France between 1992 and 1996 following a number of measures. Interestingly, the reduction was higher for previously healthy adults and pregnant women than for immunocompromised adults. Food monitoring of ready-to-eat products indicated that an important decrease in heavily contaminated products occurred during the same period (Goulet et al*.,* 2001). These data support dose-response relationships recently established for *Listeria* (FAO/WHO, 2000; 2001a). A similar decrease in listeriosis incidence was observed in the USA (Tappero et al*.,* 1995).

In Belgium a study identified eating raw or undercooked pork as major risk factors for yersiniosis. This was followed by a campaign in the media dissuading people to eat such products and by some measures to prevent contamination during the slaughtering process. The number of cases decreased from around 1,500 cases in 1986 to around 700 cases in 1996 (Verhaegen et al*.,* 1998).

Foodborne disease caused by chemicals and toxins

The use and presence of chemicals in OECD countries has been largely controlled because of effective pre-market review procedures and post-market enforcement and monitoring programmes. In the case of contaminants and naturally occurring toxicants, regulatory and voluntary programmes have reduced levels of targeted chemicals in a number of countries. For example, exposure of lead through food and the environment have shown dramatic reductions in Japan, Mexico, New Zealand, UK and USA (Watanabe, 1996; Rothenberg et al*.,* 2000; Wang et al*.,* 1997; Grosse et al*.,* 2002).

III. WHAT WE DO NOT KNOW

The extent of the foodborne disease burden

Foodborne disease caused by microorganisms

One of the main goals of FBD surveillance systems is to interpret trends, which means that exhaustive numbers of cases are not necessary and not collected. While data obtained through these surveillance systems can provide sufficient information to monitor long-term trends and identify unusual short-term trends, estimates of the burden of these diseases become necessary to design broader public health policies. Assessing a disease burden requires additional epidemiological studies, the first priority being to determine the real number of cases.

In a study done in the UK in 1994-5, one case of intestinal disease was reported for every 1.4 laboratory identifications, 6.2 stools sent for laboratory investigations, 23 cases presenting to general practice and 136 community cases (Wheeler et al., 1999). The ratio of cases in the community to cases reaching national surveillance differs between pathogens (for example, the underreporting factor is 3.2 for salmonellosis and 1,562 for infection by small round structured virus in England) and between countries (for example, salmonellosis underreporting has been estimated to 3.2 in England and to 38 in USA) (Mead et al., 1999; Wheeler et al., 1999). The limitations of the data gathered through these surveillance systems are clear. For this reason, except particular studies based on representative populations outside the health care system (Herikstad et al., 2002, Mead et al., 1999, Wheeler et al., 1999, De Wit et al., 2001a, b) or studies designed for specific diseases (Evengard, et al., 2001), data from both developed and developing countries on the extent of FBD and related deaths are very incomplete and understate the extent of the problem. Whether underreporting factors determined for one country could be used in other countries is questionable (Lake et al., 2000).

While estimating the total number of cases is a prerequisite, more information is needed on the social impact of the disease like hospitalization duration and rate, short- and long-term complications (CAST, 1994) and case-fatality rate. Little information has been collected (Adak et al., 2002; Food Standards Agency, 2000; Mead et al., 1999; De Wit et al., 2000a). Estimating the burden of a disease implies to integrate the different health effects of these illnesses such as short- and long-term complication and their impact on daily life and mortality. A public health indicator which combines the effects of morbidity and mortality is the "disability adjusted life years" (DALYs) as previously demonstrated in the WHO Global Burden of Disease study (Murray and Lopez, 1997a, 1997b). The DALY methodology requires the availability of high quality data for all relevant inputs. These data are currently available to only a limited extent. Using this method, the mean burden of campylobacteriosis in the Dutch population in 1990-1995 was estimated as 1,400 DALY per year. The mean determinants were acute gastroenteritis (440 DALY), gastroenteritis related mortality (310 DALY) and residual symptoms of Guillain-Barré syndrome (340 DALY) (Havelaar et al., 2000). A similar study done for *E. coli* O157:H7 indicated that the mean disease burden in the Netherlands was estimated at 116 DALY per year. The disease burden is also highly variable. Mortality due to HUS (58 DALY), to ESRD (end stage renal disease) (21 DALY) and dialysis due to ESRD (21 DALY) constitute the main determinants of disease burden (Havelaar et al., 2003). More studies of a similar nature are needed for a better picture of the FBD burden in OECD countries.

Foodborne disease caused by chemicals and toxins

More than 10 million chemical compounds are known to science and around 100,000 are in common use around the world. Only a small proportion of these chemicals have been fully characterized in terms of the potential toxicities to animals and humans, particularly in relation to their long-term effects. Furthermore,

prevention and control of adverse health effects due to chemicals in food are highly dependent on adequate and reliable data on levels of these chemicals in food and the total diet (Baht and Moy, 1997). In addition, new contaminants continue to be discovered. For example, acrylamide, a neurotoxin and probable human carcinogen, has recently been identified in a range of foods at relatively high levels (FAO/WHO, 2002).

Disease attributable to specific food commodities

Raw data from surveillance do not allow estimation of the percentage of cases which are foodborne and, more specifically, the number of cases which can be attributed to specific food commodities. This information is crucial for food safety risk management because of additional transmission routes for most foodborne pathogens (waterborne, animal contact, farm environment...) and because of specific pathogen-food commodity associations. However, very limited data are available.

The percentage of cases transmitted by food was recently estimated in the USA and the UK using mainly epidemiological data (Adak et al*.,* 2002; Mead et al*.,* 1999). Percentages of cases transmitted by food vary greatly according to pathogens (Table 2). In the USA, more than 13 million foodborne cases were estimated, with 9,280,000 (67%) of viral etiology (including 9,200,000 cases of Norwalk-like virus infection cases), 4,170,000 (30%) of bacterial etiology (1,960,000 campylobacteriosis cases and 1,340,000 non-typhoidal salmonellosis cases) and 350,000 (3%) of parasitic etiology. This demonstrates that three diseases - Norovirus infections, campylobacteriosis and salmonellosis - account for 70% of cases of known etiology transmitted by food. In contrast, salmonellosis, listeriosis and toxoplasmosis account for 30% of deaths caused by microorganisms. In England and Wales, six pathogens are responsible for 93% of cases of known etiology: non-typhoidal *Salmonella, Campylobacter, Yersinia, C. perfringens*, non-VTEC *E. coli* and Norovirus (Adak et al*.,* 2002).

Table 2: Percentages of foodborne transmission according to pathogens

PATHOGENS	PERCENTAGE OF FOODBORNE TRANSMISSION	
	U.S.[1]	England and Wales[2]
BACTERIA		
Aeromonas	ND[3]	0
Bacillus	100 (*B. cereus*)	100 (*B. spp.*)
Brucella	50	ND
Campylobacter	80	79.7
C. perfringens	100	94.4
VTEC O157 and non-O157	85	63
Other *E. coli*	30-70[4]	8.2
Listeria monocytogenes	99	99
Salmonella non-typhoidal	95	91.6
Salmonella typhi	80	80
Shigella spp	20	8.2
Staphylococcus aureus	100	96
Vibrio cholerae toxinogenic	90	90
Vibrio vulnificus	50	ND
Yersinia enterocolitica	90	90
PARASITES		
Cryptosporidium parvum	10	5.6
Cyclospora cayetanensis	90	90
Giardia	10 (*G. lamblia*)	10 (*G. duodenalis*)
Toxoplasma gondii	50	ND
Trichinella spiralis	100	ND
VIRUSES		
Noroviruses	40	10.7
Rotaviruses	1	2.5
Astroviruses	1	10.7
Hepatitis A virus	5	ND

[1]: Mead et al., 1999; [2]: Adak et al., 2002; [3]: ND: not determined; [4]: 70 for enterotoxigenic and 30 for other diarrheogenic

A unique microbiological approach was used in Denmark to evaluate the percentage of salmonellosis cases associated with the consumption of some specific foods. By comparing human strains and strains isolated from various products using a number of typing methods (serotyping, phage-typing, DNA macro-restriction patterns), the portions of salmonellosis cases attributable to pork, beef, table eggs, broilers, turkeys, ducks, imported pork, imported beef and imported poultry were estimated to 4.8-6.4%, 0.7-1.1%, 28-31%, 0.8-1.3%, 1.8-2.1%, 0.4-0.8%, 3.5-4.8%, 0.5-0.9% and 5.9-8.4% respectively (Anonymous, 2002b, 2002).

Foodborne disease of unknown etiology

Data from Table 2 indicates that a substantial percentage of cases are of unknown etiology. The concept of unknown etiology is supported by well-documented foodborne outbreaks of distinctive illness for which the causative agent remains unknown, the large number of outbreaks for which no pathogens is identified and by the large number of new foodborne pathogens identified in recent years (Mead et al., 1999). In the USA these unknown agents account for approximately 78-81% of foodborne illnesses (183,000,000 cases annually), for 50% hospitalizations and 64% of deaths as determined by subtracting the number of cases

accounted for known pathogens from the total number of acute gastrointestinal illnesses and applying to these figures to the previously estimated percentages of foodborne transmission (Mead et al., 1999; Mounts et al., 1999). A similar percentage, 74%, was determined for data of England and Wales (Adak et al., 2002).

Outbreaks may be classified as undetermined etiology for two main reasons: 1) because an appropriate specimen for testing was not collected or 2) because the specimen for testing was negative for all pathogens tested for in the laboratory. In this last case, a result can be negative because many pathogens are not routinely tested for in clinical laboratories or because of an unknown pathogen. In a study done in the UK in 1994-1995, 2,264 stools samples were tested for 18 bacteria, 2 protozoa and 6 viruses, no pathogens were detected in 45% of samples (Tompkins et al., 1999). A recent study was undertaken in the USA to classify foodborne outbreaks of undetermined etiology by comparing them to pathogen specific clinico-epidemiologic profiles of laboratory-confirmed outbreaks (profiles based on pathogen specific disease characteristics such as incubation period, duration and symptoms). Using this method, 12% of outbreaks remained unclassified. Such profiling could help classify outbreaks, guide investigations and direct laboratory testing to detect more often known pathogens as well as new and emerging foodborne pathogens (Hall et al., 2001).

IV. CONCLUDING REMARKS

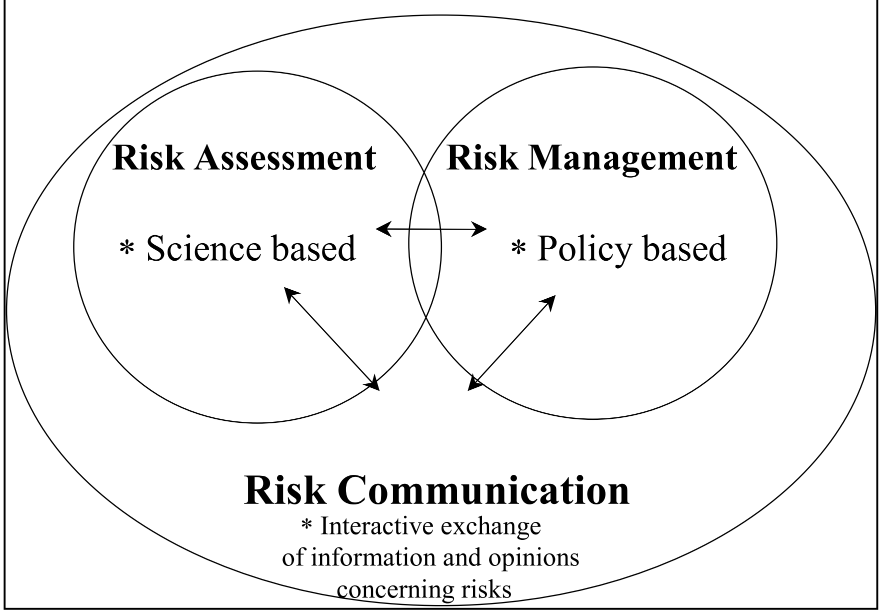
The primary goal of collecting data on FBD is for public health action. A considerable amount of information on causative agents, disease characteristics, vehicles of transmission, and mishandling errors is collected by public health authorities in all OECD countries which have been often successfully used to decrease the incidence. However, the burden of foodborne disease is still very high and certainly needs to be reduced significantly. FBD are preventable diseases but, very rare diseases excepted (typhoid fever, hepatitis A, rotavirus infection), effective vaccines are not available despite substantial research. The challenge is therefore to use a multidisciplinary approach to identify the best mitigation strategies (including consumer information and education) along the food-chain to prevent these diseases, especially at the primary production level, and then implement appropriate prevention programmes. The most appropriate method to achieve this goal is the use of the risk analysis process which links pathogens in food to the public health problem. There is therefore a strong need to collect more data on FBD, to develop research on foodborne hazards and use this information to lower the risk using the modern framework of risk analysis.

Strengthening surveillance data for microbiological risk analysis

To deal with the complexity of interactions between various human populations, pathogens and food on the one hand and to minimize the impact on public health and food economy on the other hand, the Codex Alimentarius, WHO and the Food and Agriculture Organization of the United Nations (FAO) have promoted risk analysis. Briefly, risk analysis is a process consisting in three steps (Figure 4):

- risk assessment which is a scientific process aiming at estimating the risk using four steps: hazard identification, exposure assessment, hazard characterization (a dose-response in a quantitative approach) and risk characterization (probability of disease occurrence),
- risk management which is the process of selecting, implementing and reviewing food safety policies, and especially outline and decide upon options to control the risk
- and risk communication which is an interactive exchange of information on hazards and risk between all interested parties.

Fig. 4 : the risk analysis process



As described in Table 3, data on FBD, and more especially those generated by surveillance systems, are key elements in the three parts of risk analysis. However, the experience collected at the international and national level (FAO/WHO 2000, 2001a and b; Schlundt, 2000) indicate that, due to the present characteristics of data routinely collected by surveillance it is often very difficult to use this data directly in risk assessment (Powell et al*.,* 2001). More generally a WHO consultation held in November 2000 stressed the need for more epidemiological data on FBD in formats relevant to the risk analysis and risk assessment processes (WHO, in press a).

Much progress has been made in protecting the consumer from chemical hazards. However, with the incorporation of risk analysis principles into the development of international standards, it is becoming increasingly clear that risks must be characterized more precisely and transparently than has been done in the past. In addition to long-term risks, it is becoming increasingly evident that the short-term consumption of certain substances may pose acute risks. Examples are organophosphorus pesticides and pharmacologically active veterinary drugs. Methods for evaluating these risks have been under development during the last few years, but more work needs to be done in this area.

Table 3: Interrelations between surveillance / epidemiological studies and the risk analysis process for microbiological hazards

INFORMATION ON FOODBORNE DISEASE	RISK ANALYSIS								
	Risk Profile	Risk Assessment				Risk Management			Risk Communication
		HI	HC	EA	RC	OA	I	M&R	
Incidence of cases	+	+	+	-	+	+	-	+	+
Severity of disease	+	+	+	-	+	+	-	+	+
Outbreak detection and investigation	+	+	+	-	+	+	-	+	+
Geographic distribution and spread	+	+	+	+	+	+	-	+	+
Identification of populations at higher risk	+	+	+	+	+	+	-	+	+
Trends of diseases	+	+	-	+	+	+	-	+	+
Identification of hazardous foods and handling practices	+	+	-	+	+	+	-	+	+
Percentage of cases transmitted by food and percentage of cases attributable to specific food commodities	+	+	-	-	+	+	-	-	+
Monitoring in changes in pathogens	+	+	-	-	-	-	-	-	+
Detection of emerging pathogens	+	-	-	-	-	-	-	-	+
Evaluation of prevention strategies	+	-	-	-	-	-	-	+	+
Estimation of burden	+	+	-	-	+	+	-	+	+
Understanding the natural history of the disease	+	+	+	-	+	-	-	+	+
Identification of research needs	+	+	+	+	+	+	-	+	+

HI: hazard identification, HC: hazard characterization, EA: exposure assessment, RC: risk characterization, OA: option assessment, I: implementation, M&R: monitoring and review.

Strengthening foodborne disease surveillance and epidemiological investigations

A WHO consultation held in 2002 categorized FBD surveillance systems according to their capacity to generate information (WHO, 2002 b). Figure 5 summarizes the relation between increasing degrees of maturation of surveillance systems and the associated action in public health. Briefly, syndromic surveillance systems[7], laboratory-based surveillance systems and integrated food-chain surveillance systems are the collection, analysis and interpretation of respectively: syndromic data (e.g. diarrhoea, food poisoning) from at least selected sites, of laboratory data from at least selected sites and of data from animals, food and humans (WHO, in press b). By combining a permanent analysis and interpretation of data from the food-chain and from FBD, it is obvious that the integrated system, which requires an interdisciplinary team, is the most appropriate one for a comprehensive approach, as demonstrated by the Danish experience regarding salmonellosis and food of animal origin.

7. Syndromic surveillance: surveillance that captures a set of symptoms rather than a specific disease.

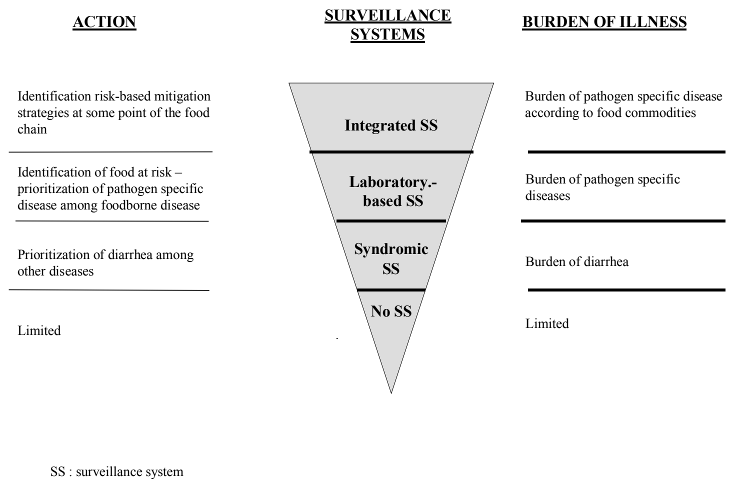


Fig. 5 : Relations between surveillance systems, burden of illness and prevention strategies.

There is also a strong need to standardize surveillance data collection and analysis as well as microbiological methods (especially detection, identification and typing of microorganisms) for laboratory-based surveillance systems. And, as mentioned earlier, additional epidemiological studies are necessary to estimate the FBD burden and to estimate the percentage of cases transmitted by food and especially by specific food commodities.

Stimulating research

Microorganisms: More research is required to decipher the complex relations between pathogens, their host and their food environment. The recent development of the genomics and the proteomics are very promising tools to improve current knowledge on microorganisms virulence factors and to use this new information to design more informative typing systems, able to characterize strains according to their ability to generate disease (DNA chips). Increased understanding about the ecology of pathogens in the food-chain, using new molecular methods, is needed to enable identification of routes of contamination and of ways to reduce this contamination. Sophisticated approaches have to been designed and used to investigate the multifaceted interactions between pathogens and hosts, especially in the field of disease pathogenesis and immunity. Finally, clinicians, epidemiologists, veterinarians, microbiologists and food scientists must collaborate even more closely to unravel the substantial amount of FBD of unknown etiology.

Chemicals and toxins: The nature of the adverse health effects posed by chemicals is of growing concern. The ability of certain chemicals to cause endocrine disruption in environmentally exposed animals is well documented and the potential health effects in humans could have serious implications. Developmental neurotoxicity has not been evaluated for many chemicals and it is recognized that immunotoxicity may occur at levels previously thought to produce no adverse effects. Two approaches that show promise include biomarkers of response at the cellular level (WHO, 2001a) and toxicogenomics which uses interactions at the molecular level (Iannaccone, 2001). Research into the potential adverse health effects of chemicals should include refinements of our knowledge about both hazard characterization and exposure assessment in order to provide the latest scientific assessments of the risks posed by these hazards. This

also serves to provide the basis for international harmonization under agreements of the World Trade Organization.

In conclusion. In spite of some very successful efforts, the burden of FBD remains high. FBD has been brought to the attention of consumers and policy-makers during the two last decades because of some highly publicized outbreaks caused by microorganisms and chemicals, and some of these incidents have been especially detrimental for the food industry. There is a need to strengthen the work already undertaken and to improve interdisciplinary approaches so that a better understanding of public health issues, including their economic consequences, will allow policy makers to design appropriate prevention strategies to lower the risk.

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ANNEX 1: TABLES

Table 1: Annual incidence (reported sporadic cases and outbreaks) of disease caused by foodborne bacterial agents in OECD countries, 1998-2001

Regions/ Countries[3]	**Bacterial Agents**[1,2]													
	Bacillus cereus	*Brucella* spp.	*Campylobacter* spp.	*Clostridium botulinum*	*Clostridium perfringens*	*Escherichia coli* VTEC[4]	*Escherichia coli* Non-VTEC	*Listeria monocytogenes*	*Salmonella*, Typhi	*Salmonella*, non-typhoidal	*Shigella* spp.	*Staphylococcus aureus*	*Vibrio*, (excluding *cholerae* and *vulnificus*)	*Yersinia enterocolitica*
Americas														
Canada 1999	-	-	**11,500 (37.7)**	-	-	**1,490 (4.9)**	-	**59 (0.3)**	**71 (0.2)**	**5,611 (18.4)**	**1,084 (3.6)**	-	-	-
United States 1999	**194 (0.1)** 7 (194) *27,360*	**82 (0.03)** *1,554*	**-(13.8)**[6] 5 (85) *2,453,926*	**23 (< 0.01)** 1 (3) *58*	**1,213 (0.4)** 24 (1,213) *248,520*	**- (1.6)** [6] 38 (1,897) *73,480*	**69 (0.03)** 2 (69) *36,740*	**-(0.3)** [6] 5 (28) *2,518*	**346 (0.1)** 1 (16) *824*	**-(15.1)** [6] 119 *(3,378)* *1,412,498*	**17,521 (6.4)** 14 (221) *448,240*	**346 (0.1)** 18 (346) *185,060*	**14 (<0.01)** 3 (14) *7,880*	**-(0.4)** [6] 1 (32) *96,368*
Asia														
Japan 2001	-	**444 (0.4)**	**1,880 (1.5)**	-	**1,656 (1.3)**	**378 (0.3)**	**2,293 (1.8)**	-	-	**4,949 (3.9)**	**19 (0.02)**	**1,039 (0.8)**	**3,065 (2.4)**	**4 (<0.01)**
Korea 2001	-	-	-	-	-	-	-	-	13 (561)		-	10 (363)	13 (254)	-
Europe														
Austria 1998	-	**1 (<0.01)**	**2,454 (30.3)**	-	-	**17 (0.2)**	-	-	**12 (0.3)**	**7,236 (89.3)** 870	**167 (2.1)**	**16 (0.2)**	-	**94 (1.2)**
Belgium 2000	-	-	**7,473 (73.0)**	-	-	**47 (0.5)**	-	**48 (0.5)**	**16 (0.2)**	**14,001 (137.0)**	**208 (2.0)**	-	-	**507 (5.0)**
Czech Republic 1998	-	-	-	**6 (0.1)**	-	**126 (1.2)**	-	**13 (0.1)**	**3 (<0.01)**	**49,045 (476.2)**	**511 (4.9)**	-	-	-
Denmark 2001	-	**18 (0.3)**	**4,620 (86.4)**	-	-	**92 (1.7)**	-	**38 (0.7)**	**17 (0.3)**	**2,918 (54.5)**	**148 (2.8)**	-	-	**286 (5.3)**
Finland 2001	-	**1 (<0.01)**	**3,969 (76.4)**	-	-	**18 (0.3)**	**13 (0.3)**	**28 (0.5)**	**245 (4.7)**	**2,731 (52.6)**	**223 (4.3)**	-	-	**728 (14.0)**

Regions/ Countries[3]	Bacterial Agents[1,2]													
	Bacillus cereus	*Brucella* spp.	*Campylobacter* spp.	*Clostridium botulinum*	*Clostridium perfringens*	*Escherichia coli* VTEC[4]	*Escherichia coli* Non-VTEC	*Listeria monocytogenes*	*Salmonella*, Typhi	*Salmonella*, non-typhoidal	*Shigella* spp.	*Staphylococcus aureus*	*Vibrio*, (excluding *cholerae* and *vulnificus*)	*Yersinia enterocolitica*
Europe (continued)														
France **1998**/1999	155 (2,214)	-	-	**28 (0.05)**	15 (224)	**98[5] (0.9)**	-	**270 (0.5)**	-	**13,668 (23.1)** 297 (3,159)	**941 (1.6)**	22 (235)	-	-
Germany 1998	-	**18 (0.02)**	**60 (0.1)** 4 (60)	**21 (0.02)**	-	-	-	**31 (0.04)**	-	**97,505 (118.6)** 108 (1,838)	**1,607 (2.0)**	**94 (0.1)** 2 (94)	-	-
Greece 1998	-	**440 (4.2)**	**136 (1.3)**	-	-	-	-	**1 (<0.01)**	-	**922 (8.8)**	**92 (0.9)** 1	-	-	**10 (0.1)**
Hungary 1998	**177 (1.8)** 5 (177)	-	**207 (2.0)** 13 (173)	**19 (0.2)** 4 (13)	**83 (0.8)** 1 (83)	-	**13 (0.1)** 1 (13)	-	-	**18,107 (179.3)** 269 (2,319)	**645 (6.4)** 6 (63)	**1 (<0.01)**	-	-
Iceland 2001	-	-	**214 (79.9)**	-	**14 (4.9)**	-	**1 (<0.01)**	-	-	**166 (58.0)**	-	**12 (4.2)**	-	-
Ireland 2000	-	**15 (0.4)**	**2,085 (57.5)**	-	**9 (0.2)** 1 (9)	-	**35 (1.0)** 4 (21)	-	-	**640 (17.6)** 6 (133)	**71 (2.0)** 1 (41)	**7 (0.2)** 1 (7)	-	-
Italy 1998	1	**1,461 (2.6)**	-	**33 (0.1)** 5	-	-	-	**45 (0.1)** 1	2	**14,358 (25.1)** 177	-	4	-	-
Luxembourg 1998	-	-	-	-	-	-	-	-	**49 (12.6)**		-	-	-	-
Netherlands 2001	-	**3 (0.02)**	*100,000*	-	-	**43 (0.3)**	-	-	**17 (0.1)**	**4,384 (30.6)**	-	-	-	*180*
Norway 2001	-	**2 (<0.01)**	**2,889 (64.2)** 2 (18)	-	-	-	**15 (0.3)**	**18 (0.4)**	**18 (0.4)**	**1,899 (42.0)** 8 (338)	**189 (4.2)**	-	-	**123 (2.8)**
Poland 1998	-	-	-	**93 (0.2)**	-	-	-	-	-	**26,675 (69.0)**	-	**375 (1.0)**	-	-
Portugal 1998	**3 (0.03)**	**817 (7.9)**	-	**17 (0.2)**	**1 (0.01)**	**1 (0.01)**	-	-	-	**643 (6.2)**	**10 (0.1)**	**9 (0.09)**	-	-
Slovak Republic 1998	-	-	**1,304 (26.1)**	**5 (0.1)**	-	**521 (10.4)**		-	**1 (0.02)**	**21,471 (398.3)** 82 (3,237)	**1,075 (19.9)**	-	-	-

Regions/ Countries[3]	Bacterial Agents[1,2]													
	Bacillus cereus	*Brucella* spp.	*Campylobacter* spp.	*Clostridium botulinum*	*Clostridium perfringens*	*Escherichia coli* VTEC[4]	*Escherichia coli* Non-VTEC	*Listeria monocytogenes*	*Salmonella*, Typhi	*Salmonella*, non-typhoidal	*Shigella* spp.	*Staphylococcus aureus*	*Vibrio*, (excluding *cholerae* and *vulnificus*)	*Yersinia enterocolitica*
Europe (continued)														
Spain 1998	4	**1,545 (3.9)** 10	**4,389 (11.1)** 1	**13 (0.03)** 9	22	12		**16 (0.04)**	**316 (0.8)** 3	**6,653 (16.8)** 551	**170 (0.4)** 3	36	2	**425 (1.1)**
Sweden 2001	-	-	**8,577 (96.3)**	-	-	**95 (1.1)**	-	**67 (0.8)**	**10 (0.1)**	**4,711 (52.9)**	**540 (6.1)**	**429 (4.8)**	-	**579 (6.5)**
Switzerland 1998	-	-	**5,455 (76.5)**	-	-	-	-	-	**3,004 (42.1)**		**499 (7.0)**	-	-	**51 (0.7)**
Turkey 1998	-	**12,330 (19.6)**	-	**120 (0.2)**	-	-	-	-	**30,269 (48.1)**	-	**1,457 (2.3)**	-	-	-
United Kingdom 2000 England & Wales	-	-	**55,887 (95.0)**	-	-	**986 (1.5)**	-	**100 (0.2)**	**14,844 (25.2)**		**966 (1.6)**	-	-	**27 (0.05)**
Oceania														
Australia 2000	-	**27 (0.1)**	**13,595 (107.1)**	**2 (<0.01)**	-	-	**33 (0.2)**	**67 (0.3)**	**58 (0.3)**	**6,151(32.1)** 22 (495)	**487 (3.8)** 3 (172)	-	-	**73 (0.6)**
New Zealand 2001	**21 (0.6)** 6 (21)	-	**10,148 (271.5)** 56 (301)	**59 (1.6)** 15 (59)	**16 (0.4)**	**76 (2.0)** 4 (10)	-	**18 (0.5)**	**26 (0.7)**	**2,417 (64.7)** 37 (214)	**157 (4.2)** 9 (61)	**1,710 (45.8)** 11 (23)	-	**429 (11.5)** 3 (10)

[1] Bold font = incidence (incidence rate per 100,000); regular font = number of outbreaks (total number of cases); italics = estimated total number of cases per year (estimated incidence rate per 100,000).

[2] Cases caused by multiple pathogens are not included due to their very low incidence.

[3] Latest available year of data between 1998-2001 selected for each country.

[4] VTEC – *E. coli* Shiga toxin-producing serogroups other than O157.

[5] Cases of children < 5 only.

[6] Data from FoodNet

[-] No data presently available.

Sources: Anonymous (1), 2002a; Anonymous (1), 2000a; Anonymous (2), 2002c; Anonymous (2), 2000b; Anonymous (3), 2002d; Anonymous (3), 2001a; Anonymous (3), 2000c; Anonymous (4), 2002e; Anonymous (4), 2000d; Anonymous (5), 2002f; Anonymous (5), 2001b; Anonymous (6), 2002g; Anonymous (7), 2001c; Anonymous (8), 2001d; Bouvet, Grimont, 2001; Ducoffre, 2002; Ekdahl, 2001; Goulet et al., 2001; Groseclose et al., 2000; Haeghebaert et al., 2001a; Haeghebaert et al., 2001b; Haeghebaert et al., 2002; Korean Food and Drug Administration, personal correspondence; Lin, 2002; Mead et al., 1999; Sneyd et al., 2002; Thornley et al., 2002.

Table 2: Annual incidence (sporadic cases and outbreaks) of disease caused by foodborne parasites, viruses, and unknown etiology in OECD countries, 1998-2001

	Parasites[1,2]					**Viruses[1,2]**				
Regions/ Countries[3]	*Cryptosporidium parvum*	*Cyclospora cayetanensis*	*Giardia lamblia*	*Toxoplasma gondii*	Trichinella *spiralis*	Astrovirus	Hepatitis A	Norwalk-like viruses	Rotavirus	**Unknown etiology**
Americas										
Canada 1999	-	-	**5,234 (17.2)** 1	-	-	-	**887 (2.9)**	-	-	-
Mexico[4]	-	-	-	-	-	-	-	-	-	-
United States 1999	**3,128 (1.1)** *300,000*	**60 (0.02)** *16,264*	*2,000,000*	*225,000*	**16 (<0.01)** *52*	*3,900,000*	**13,397 (4.9)** *83,391*	*23,000,000)*	*3,900,000*	-
Asia										
Japan 2001	-	-	-	-	-	-	-	**7,358 (5.8)**	-	**2,298 (1.8)**
Korea 2001	-	-	-	-	-	-	-	-	-	39 (3,380)
Europe										
Austria 1998	-	-	-	-	**1 (<0.01)**	-	-	-	-	**11 (0.1)**
Belgium 2000	**659 (6.4)**	**19 (0.1)**	**1,669 (16.0)**	-	-	-	**437 (4.3)**	-	**6,752 (65.9)**	-
Czech Republic 1999	-	-	**276 (2.7)**	**(8.3)**	-	-	**904 (9.0)**	-	-	**2,070 (20.6)**
Denmark 2001	**84 (1.6)**	-	-	**NR[5]**	-	-	**63 (1.2)**	-	-	-

	Parasites[1,2]					Viruses[1,2]				
Regions/ Countries[3]	*Cryptosporidium parvum*	*Cyclospora cayetanensis*	*Giardia lamblia*	*Toxoplasma gondii*	Trichinella *spiralis*	Astrovirus	Hepatitis A	Norwalk-like viruses	Rotavirus	**Unknown etiology**
Finland 1999	-	-	-	-	-	-	-	-	-	-
France 1998/1999	-	-	-	-	-	-	-	-	-	59 (187)
Germany 1998	-	-	-	-	**51 (0.06)**	-	**3,856 (4.7)**	-	2 (29)	26
Greece 1998	-	-	**42 (0.4)**	-	-	-	**261 (2.5)**	-	-	-
Hungary 1998	-	-	-	-	**3 (< 0.01)**	-	-	-	-	35 (707)
Iceland 2001	-	-	**26 (9.0)**	-	-	-	-	-	1 (4)	-
Ireland 2000	-	-	-	-	-	-	**309 (8.5)**	-	**4 (0.1)** 1 (4)	-
Italy 1998	-	-	-	**NR**	**92 (0.2)**	-	**2,962 (5.2)**	-	-	-
Luxembourg 1998	-	-	-	-	-	-	-	-	-	-
Netherlands 2001	-	-	-	-	**2 (0.01)**	-	-	-	-	-
Norway 2001	-	-	**338 (7.5)**	**NR** 0	**0** 0	-	**86 (1.9)**	-	-	-
Poland 1998	-	-	-	-	**33 (0.1)**	-	-	-	-	**3,840 (9.9)**
Portugal 1998	-	-	-	-	-	-	-	-	-	**29 (0.3)**
Slovak Republic 1998	-	-	-	-	**345 (6.9)** 1 (345)	-	-	-	-	-

Regions/ Countries[3]	Parasites[1,2]					Viruses[1,2]				
	Cryptosporidium parvum	*Cyclospora cayetanensis*	*Giardia lamblia*	*Toxoplasma gondii*	Trichinella *spiralis*	Astrovirus	Hepatitis A	Norwalk-like viruses	Rotavirus	**Unknown etiology**
Spain 1998	-	-	-	-	**58 (0.1)** 2	-	10	-	-	245
Sweden 2001	**92 (1.0)**	-	**1,435 (16.1)**	**18 (0.2)**	**0**	-	**169 (1.9)**	-	-	-
Switzerland 1998	-	-	-	-	-	-	-	-	-	-
Turkey 1998	-	-	-	-	-	-	**14,000 (22.3)**	-	-	-
United Kingdom 2000 England & Wales	**5,799 (9.9)**	-	**4,015 (6.8)**	-	-	**234 (0.4)**	**1,024 (1.7)**	-	**16,528 (28.1)**	-
Oceania										
Australia 2000	**1,570 (8.2)**	-	-	-	-	-	**812 (4.2)**	-	-	-
New Zealand 2001	**1,207 (32.3)** 27 (147)	-	**1,603 (42.9)** 18 (75)	-	**2 (0.1)**	-	**61 (1.6)** 3 (11)	**647 (17.3)** 45 (541)	**49 (1.3)** 3 (41)	-

[1] Bold font = incidence (incidence rate per 100,000); regular font = number of outbreaks (number of cases); italics = estimated total number of cases per year (estimated incidence rate per 100,000)
[2] Cases caused by multiple pathogens are not included due to their very low incidence
[3] Latest available year of data between 1998-2001 selected for each country
[4] Data pending
[5] NR = Not Reportable
[-] No data presently available

Sources: See Table 1.

Table 3: Foods implicated in foodborne disease outbreaks caused by microorganisms in OECD countries, 1998-2001[1]

Foods	Czech Republic (1998)	France (1998)	Germany (1998)	Hungary (1998)	Iceland (1998)	Ireland (2000)	Italy (1998)	Japan (2000)	Netherlands (1998)	New Zealand (2001)	Norway (1998)	Poland (1998)	Portugal (1998)	Slovak Republic (1998)	Spain (1998)	Sweden (1998)	Switzerland (1998)	UK (1998)	United States (2000)
Meat and meat products	12	100	9	131	2	2	7	56	38	13	5	56	9	2	-	15	-	17	101
Poultry	2	43	-	-	-	2	-	-	16[2]	17	-	20[2]	2[3]	2	73[3]	4	-	20	81
Eggs and egg products	18	175	19	242	-	1	40	35	-	-	1	19	-	52	363	-	6	14	12
Seafoods	-	57	-	-	-	2	8	200	10	13	1	2	2	-	63	3	-	12	79
Milk and dairy products	2	40	-	5	-	1	5	3	15	2	1	11	1	-	30	6	-	2	12
Produce (fruits and vegetables)	-	-	-	-	-	-	-	22	3	-	-	-	-	-	-	2	-	8	64
Cereals, pasta	-	-	-	11	-	1	-	23	-	2	-	-	-	-	-	1	-	2	13
Confectionery (high sugar)	20	-	-	71	-	-	19	14	-	4	-	155	10	-	48	2	-	13	13
Mixed dishes	-	-	1	-	1	2	-	82	-	49	-	39	-	-	-	15	-	-	183
Multiple foods	-	-	-	-	-	-	-	-	-	5	-	-	7	-	-	12	-	-	157
Other	21	105	-	107	1	2	1	363	90	29	20	55	8	6	91	1	2	19	58
Unknown	72	142	-	22	5	10	-	1094	-	-	-	51	9	20	274	11	5	-	720
Total	147	662	29	589	9	23	80	1892	156	134	28	388	46	82	869	72	13	107	1493

[1] Latest year of data between 1998-2001 selected for countries with available data
[2] Includes poultry and egg products
[3] Includes poultry and meat products
- No data presently available

Sources: Anonymous (1), 2002a; Anonymous (2), 2002c; Anonymous (5), 2002f; Anonymous (7), 2001c; Haeghbaert et al*.,* 2001; Sneyd et al*.*, 2002; Thornley et al*.*, 2002.

Table 4: Foodborne disease outbreaks caused by microorganisms by place where food was eaten, acquired, or prepared in OECD countries, 1998-2001[1]

Place	Denmark (1998)	Finland (1998)	France(1998)	Germany 1998)	Hungary (1998)	Iceland (1998)	Ireland (2000)	Japan (2001)	Netherlands (1998)	New Zealand (2001)	Poland (1998)	Portugal (1998)	Slovak Republic (1998)	Spain (1998)	Sweden (1998)	Switzerland (1998)	UK (1998)	United States (2000)
Private House	22	13	257	15	665	4	6	206	10	138	210	6	23	407	17	6	12	225
Hotel/Restaurant/other eating establishments	39	49	156	5	39	2	17	577	118	148	40	25	22	315	40	6	62	615
Hospital/Residential Institution	2	4	35	-	6	-	6	37	1	24	26	-	4	19	-	1	2	27
Workplace/School/Kindergarten	1	13	137	4	37	1	1	50	-	37	41	4	13	34	1	1	2	84
Catering	-	-	-	1	-	1	-	59	-	7	-	2	14	-	2	-	7	-
Food manufacturing	1	-	-	-	9	-	-	23	-	-	-	-	-	-	-	-	-	-
Retail/mobile retailer	9	-	-	-	5	-	-	5	-	12	-	6	-	37	-	-	13	3
Other	3	12	67	-	8	1	6	32	23	25	95	3	6	87	5	-	22	221
Unknown	-	1	-	4	3	-	-	939	20	43	-	1	-	43	7	-	-	126
Total	77	92	652	29	772	9	36	1928	172	434	412	47	82	942	72	14	120	1301

[1] Latest year of data between 1998-2001 selected for countries with available data

Sources: Anonymous (1), 2002a; Anonymous (2), 2002c; Anonymous (5), 2002f; Anonymous (7), 2001c; Haeghbaert et al., 2001; Sneyd et al., 2002; Thornley et al., 2002

Table 5: Contributing factors of foodborne disease outbreaks caused by microorganisms in OECD countries, 1998-2001[1,2]

Contributing Factors	Denmark (1998)	Finland (1998)	France(1998)	Hungary (1998)	Iceland (1998)	Ireland (1998)	New Zealand (2001)	Slovak Republic (1998)	Spain (1998)	Sweden (1998)	UK (1998)
Factors related to contamination	-	-	-	-	-	-	-	-	-	-	-
Raw foods	-	-	39	120	-	-	3	-	112	-	-
Use of a contaminated ingredient(s)	22	14	-	-	-	-	3	32	-	-	-
Foods obtained from unsafe sources	-	-	-	-	-	-	9	-	-	-	-
Infected person(s)	-	7	2	1	-	5	9	-	-	2	119
Inadequate food handling/food handlers	-	-	-	-	-	-	6	-	131	-	-
Contaminated equipment	-	2	39	3	1	-	-	-	-	-	-
Improper storage	4	12	-	-	-	3	15	19	-	-	324
Cross contamination	14	-	-	-	-	6	29	45	50	-	286
Factors related to survival of microorganisms	-	-	-	-	-	-	-	-	-	-	-
Time/temperature abuse	16	32	55	321	4	6	68	21	261	14	333
Food inadequately preserved	-	-	-	-	-	-	1	-	-	-	-
Factors related to microbial growth	-	-	-	-	-	-	-	-	-	-	-
Food was prepared too far in advance	-	3	36	-	-	6	3	-	110	-	-
Low and intermediate moisture foods had elevated water activity or condensation	-	-	-	-	-	-	4	-	-	-	-
Preparation of too large quantities	-	-	-	-	-	-	-	-	12	-	-
Other	-	-	-	-	-	-	-	-	-	-	-
Inadequate food preparation facilities	-	-	-	3	-	-	3	-	17	-	-
Insufficient hygiene	-	-	-	-	-	-	-	5	69	-	-
Error in processing	-	-	41	-	-	-	-	-	-	-	-
Other	-	11	-	2	-	-	21	20	67	3	100
Unknown	21	35	-	147	4	18	69	27	426	53	-
Total	77	116	212	597	9	44	243	169	1255	72	1162

[1] Latest year of data between 1998-2001 selected for countries with available data
[2] More than one factor identified for some outbreaks

Sources: Anonymous (1), 2002a; Anonymous (7), 2001c; Haeghbaert et al., 2001; Sneyd et al., 2002; Thornley et al., 2002