Unclassified **AGR/CA/APM(2002)28/FINAL**

Organisation de Coopération et de Développement Economiques
Organisation for Economic Co-operation and Development

10-Sep-2003

English - Or. English

DIRECTORATE FOR FOOD, AGRICULTURE AND FISHERIES
COMMITTEE FOR AGRICULTURE

Working Party on Agricultural Policies and Markets

THE INCIDENCE AND COSTS OF FOODBORNE DISEASE

Contact person: Wayne Jones (E-mail: Wayne.Jones@oecd.org)
Tel: 33-1-45-24-78-74
Fax:33-1-44-30-61-19

JT00148822

AGR/CA/APM(2002)28/FINAL
Unclassified

English - Or. English

NOTE BY THE SECRETARIAT

A report on the incidence of foodborne disease [AGR/CA/APM(2002)28], prepared for the OECD by the World Health Organisation, was discussed at the 3-4 October 2002 meeting of the Working Party on Agricultural Policies and Markets. A complementary report on the costs of foodborne disease [AGR/CA/APM(2003)12], prepared by the University of Reading consultant Richard TiffIn, was discussed at the 31 March – 2 April APM meeting. It was agreed to revise and combine the two documents for submission to the APM for declassification under the written procedure as a consultant's report under the responsibility of the Secretary-General.

This document is now declassified under the responsibility of the Secretary-General.

TABLE OF CONTENTS

THE INCIDENCE AND COSTS OF FOODBORNE DISEASE

PART I: THE INCIDENCE OF FOODBORNE DISEASE

I. INTRODUCTION

1. 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 hospitalisations, 5 000 deaths in the US and 2 366 000 cases, 21 138 hospitalisations, 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 OECD countries.

2. 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

FBD caused by micro-organisms

3. 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 micro-organisms, 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 hospitalisation, long term disability and/or death (hospitalisation rates from 0.6% to 29% and case-fatality rates up to 2.5% in the US) (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* septiceamia, 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 neurologic 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)

4. While diarrhoea is the most common syndrome following the consumption of a contaminated food, some diseases are more serious. Clinical manifestations of listeriosis include bacteriemia 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 foetus (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.

FBD caused by chemicals and toxins

5. 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)

6. Once the hazard characterisation 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 characterisation may improve the accuracy and reliability of risk assessments of chemicals in food (WHO, 2001a).

Present state of foodborne disease in OECD countries

FBD caused by micro-organisms

7. 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 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 dependant 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.

8. 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.

---

[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).

[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.

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

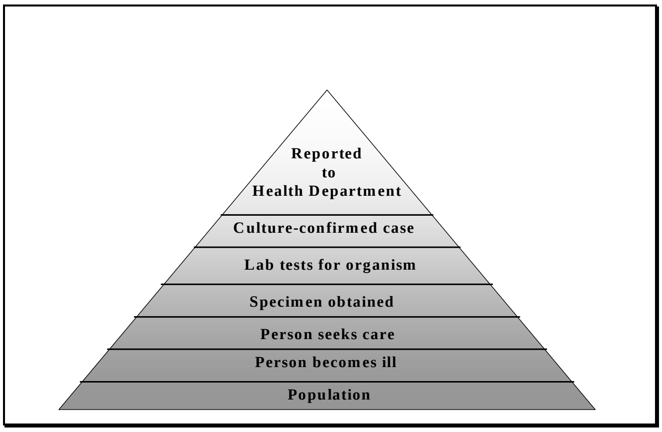


Table 1. Examples of underreporting factors (outbreaks and sporadic cases

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

References: Meal *et al.*, 1999; Michel *et al.*, 2000 ; Goulet *et al.*, 2001.

1. No information
2. Wheeler *et al.*, 1999
3. Adak *et al.*, 2002.
4. *E. coli* 0157 only.

9. 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 micro-organisms 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).

10. 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.

11. Tables 3 and 4 summarise 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 formalised 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 is 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 Witt *et al.*. 2000; Hedlund *et al.,* 2000).

12. Briefly, data from Tables 3 and 4 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 case 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 case and 0.5 case per 100 000 population for listeriosis, between 0.01 case and 1.6 cases per 100 000 population for botulism. Despite the incidence of brucellosis is 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.

---

[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).

13. It should be noted that aggregating data at the national level may not reflect the exact situation. For example, in the US, data from FoodNet indicate variations in incidence of these diseases as well as variations in *Salmonella* serotypes according the States (FoodNet, 2000). Similarly, while the incidence rate of brucellosis is very low in the US, 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).

14. 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 (see paragraphs 20-29). Some bacterial pathogens generate high numbers of outbreaks, like non-typhoidal *Salmonella*. In 1995, 757 salmonellosis outbreaks were estimated in France, a figure which could be as high as 2000 in reality (Gallay *et al.,* 2000). In the US, 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 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 recognised bacterial cause of gastro-intestinal 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. paraheamolyticus* 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 were the etiologic agent of 284 outbreaks in the US 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 pediatric cases in Japan are caused by Norovirus (Inouye *et al.,* 2000).

15. 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 nation-wide 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. paraheamolyticus* 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)

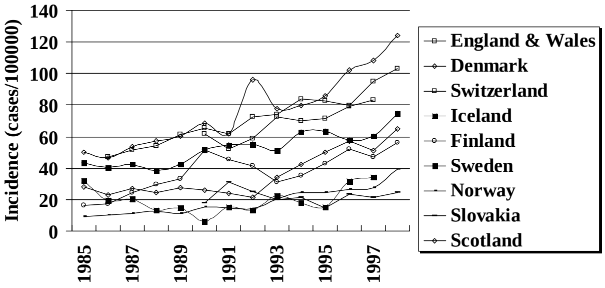
16. Data from a number of countries indicates that the incidence of FBD of known etiology has considerably increased during the past two decades. This is probably mainly a result of the increased reported number of cases caused by *Campylobacter* and *Salmonella*, especially because of *S.* Enteritis pandemic (Rodrigue *et al.*, 1990). In Europe for example, a tremendous increase in the number of cases of nontyphoidal salmonellosis was observed, with a peak being reached in 1992 for a number of countries.

---

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

Similarly, reports on campylobacteriosis have been continuously increasing in this region since 1985 and this disease is currently is 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). (See Figure 2.)

Figure 2. Annual incidences of campylobateriosis in European countries



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

17. 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 5[6], 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 4[6] and: 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 7[6]. From the available data, time/temperature abuse appears to be the most frequent contributing factor in many OECD countries.

---

[6] This data has been compiled through a limited-time search of data from open literature. It does not represent a formalised 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.

FBD caused by chemicals and toxins

18. 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).

19. 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 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).

20. 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% (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 sensitisation (WHO/UNEP, 1990).

21. 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).

22. Accidental or intentional adulteration of food by toxic substances has resulted in serious public health incidents in both developing and industrialised 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

23. The last two decades have been characterised 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

24. 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:

25. *New feeding practices*: 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, 2002c).

26. *Change in animal husbandry*: Modern intensive animal husbandry practices introduced to maximise 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 US, in 1969 470 832 layer-hen farms with an average of 632 hens per farm produced 67 billion 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).

27. *Changes in agronomic process:* The use of manure rather than chemical fertilisers, 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).

28. *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.

29. *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).

30. *Increase in susceptible populations:* Advances in medical treatment have resulted in an increasing number of the elderly and immunocompromised people. In many industrialised 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 industrialised countries is at higher risk of FBD as a result of some sort of immune-suppression (Gerba *et al.,* 1996).

31. *Increase in travel*: Globalisation 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 US are attributed to international travel, (Anonymous, 2001c, Schlosser and Cervantes, 1998; Steinberg *et al.*, 2001).

32. *Change in lifestyle and consumer demands:* Previously unrecognised microbial hazards have emerged as a result in 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

33. New pathogens have been recognised 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 serotype 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 1990s - 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 *Salmonellla* Newport strain (resistant to nine antimicrobials, including some of the most important new antimicrobials) emerged in the US in 1999 and now seems to have spread to many parts of the US (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.

34. 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 realise that these foodborne diseases require new food control strategies.

35. These characteristics, associated with changes in food production and distribution have generated a new outbreak scenario. Traditional outbreaks were characterised by an acute and locally limited number of cases, with a high inoculum dose and a high attack rate sometimes because of a food-handler 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 food-handling 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 US 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

36. These new pathogens prompted several new surveillance approaches to provide more information. In the US, 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 US 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 US 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 nation wide database. When a cluster is flagged, a detailed epidemiological investigation can often determine the source (Swaminathan *et al.,* 2001).

37. Concurrently 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 characterise microorganisms which introduced new means for laboratory-based surveillance system (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).38. Because of changes in reporting systems during the last two decades, data should be analysed 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 unmeasurable extent, related to surveillance and laboratory testing improvement.

Success in foodborne disease reduction

FBD caused by microorganisms

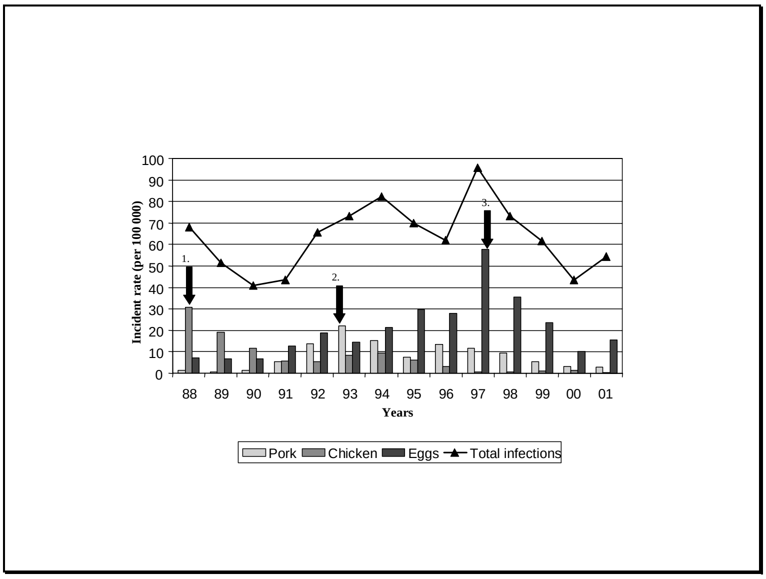
38. Success in FBD incidence declining have been mainly the result of a limited number of interventions, especially at the production level, for a limited number of pathogens in a limited number of countries.

39. Sanitation and the decrease of typhoid fever, milk pasteurisation 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 has been associated with a clear reduction in incidence of FBD.

40. To control *Salmonella* in poultry, a compulsory programme was implemented in Sweden by control and quarantine of grand-parent 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).

41. 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 (see paragraph 50).

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



42. 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).

43. 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 US (Tappero *et al.,* 1995).

44. 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).

FBD caused by chemicals and toxins

45. 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

FBD caused by Microorganisms

46. One of the main goals of FBD surveillance systems is to interpret trends, which means that exhaustive numbers of cases is 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 more broad public health policies. Assessing a disease burden requires additional epidemiological studies, first to determine the real number of cases.

47. 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 under-reporting factors determined for one country could be used in other countries is questionable (Lake *et al.*, 2000).

48. While estimating the total number of cases is a prerequisite, more information is needed on the social impact of the disease like hospitalisation 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).

49. 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 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 (Havalaar *et al.*, 2003). More studies of a similar nature are needed for a better picture of the FBD burden in OECD countries.

FBD caused by chemicals and toxins

50. 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 characterised 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

51. Raw data from surveillance do not allow to estimate 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.

52. The percentage of cases transmitted by food was recently estimated in the US 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 US, more than 13 million foodborne cases were estimated, with 9 280 000 (67%) of viral etiology (including 9 200 000 bases of Norwalk-like virus infection cases), 4 170 000 (30%) of bacterial etiology (1 960 000 campyloacteriosis 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	
	US[1]	England and Wales[2]
BACTERIA		
Aeromonas	ND[3]	0
Bacillus	100 (*B. cereus*)	100 (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.

53. 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).

FBD of unknown etiology

54. 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 US, these unknown agents account for approximately 78-81% of foodborne illnesses (183 000 000 cases annually), for 50% hospitalisations 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).

55. 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 US 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

56. 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

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

- risk assessment which is a scientific process aiming at estimating the risk using four steps : hazard identification, exposure assessment, hazard characterisation (a dose-response in a quantitative approach) and risk characterisation (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.

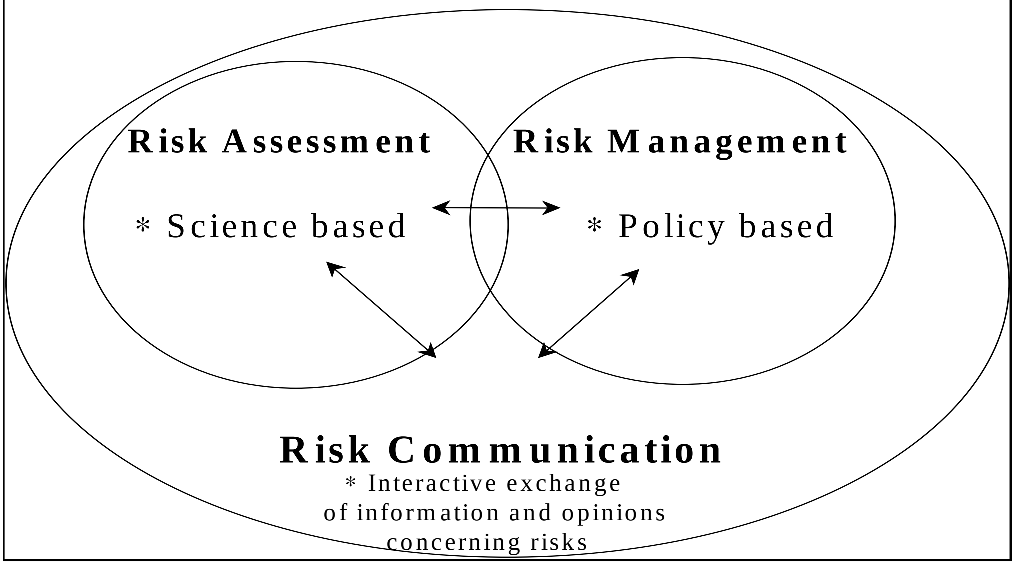


Fig. 4 : the risk analysis process

58. As described in Table 8, 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).

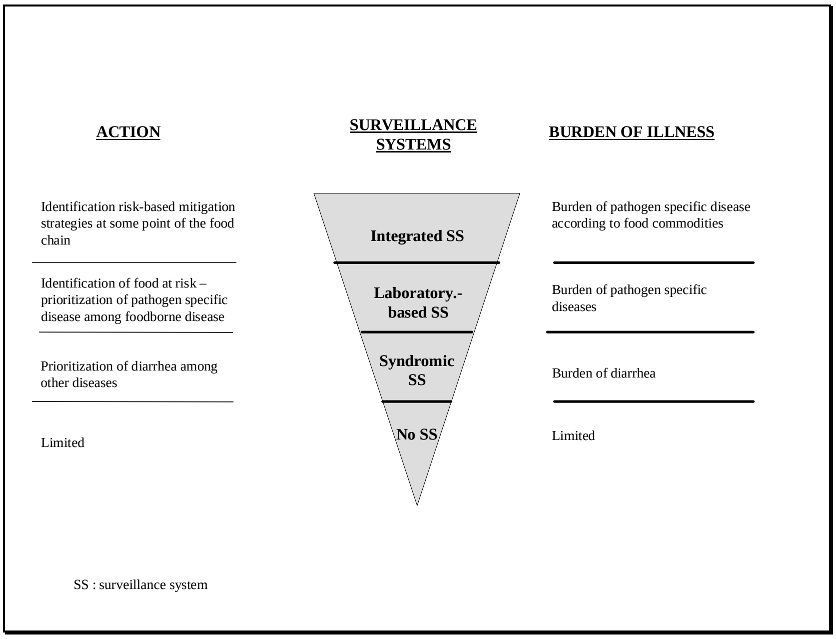
59. Much progress has been made in protecting the consumer from chemical hazards. However, with the incorporation of risk analysis principles into the development of international standards, it is becoming increasingly clear that risks must be characterised more precisely and transparently than has been 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.

Strengthening foodborne disease surveillance and epidemiological investigations

60. A WHO consultation held in 2002 categorised FBD surveillance systems according to their capacity to generate information (WHO, 2002 b). Figure 5 summarises the relation between increasing

degree 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 a interdisciplinary team, is the most appropriate one for a comprehensive approach, as demonstrated by the Danish experience regarding salmonellosis and food of animal origin (see paragraph 41).

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



61. There is also a strong need to standardise 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

62. 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 characterise 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

---

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

63. *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 recognised 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 characterisation 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 harmonisation under agreements of the World Trade Organization.

The economic costs of FBD

64. 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 publicised 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.

65. A second phase of this study will examine the economics of foodborne disease. Available estimates suggest medical costs and productivity losses are very high. A recent USDA study (Buzby *et al.*, 1996) of six bacterial pathogens, for example, estimated the costs of human illness attributed to six foodborne bacteria at USD 9.3-12.9 billion annually. Based on existing literature, this second phase, to be completed by the end of the year, will:

- identify the various economic costs associated with foodborne diseases (*e.g.* productivity losses, medical costs, prevention of premature death) and briefly describe the methods of estimation;

- collect, assemble and interpret available country estimates of the economic costs of foodborne disease for the OECD area;

- offer observations on the importance, availability and quality of economic information on foodborne diseases and possible areas of future work;

- compare economic cost estimates using different valuation methods (DALYs, cost of illness and willingness to pay); and

- investigate economic incentives for food safety, including regulatory interventions (such as HACCP, Food Safety Objectives...), control procedures to minimize cross-contamination, microbial growth, equipment to kill pathogen.

ANNEX 1: TABLES

Annex 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* , yphi	*Salmonella*, nontyphoidal	*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)**	-	-	-
Mexico	-	-	-	-	-	-	-	-	-	-	-	-	-	-
United States1999	**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)**	-	-	-
Europe														

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* , yphi	*Salmonella*, nontyphoidal	*Shigella* spp.	*Staphylococcus aureus*	*Vibrio*, (excluding *cholerae* and *vulnificus*)	*Yersinia enterocolitica*
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)**
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)**	-	-

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* , yphi	*Salmonella*, nontyphoidal	*Shigella* spp.	*Staphylococcus aureus*	*Vibrio*, (excluding *cholerae* and *vulnificus*)	*Yersinia enterocolitica*
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)**	-	-	-
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.

Annex Table 2. Annual incidence (sporadic cases and outbreaks) of lab confirmed 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)	-	-	-	-	-

	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**
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.

Annex 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
Confectionary (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.

Annex 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

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

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

Annex Table 6. 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	+	+	+	+	+	+	-	+	+

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

REFERENCES

Aarestrup FM, Bager F, Jensen NE, Madsen M, Meyling A, Wegener HC. “Resistance to antimicrobial agents used for animal therapy in pathogenic-, zoonotic- and indicator bacteria isolated from different food animals in Denmark: a baseline study for the Danish Integrated Antimicrobiol Resistance Monitoring (DANMAP).” *Acta Pathologica Microbiologica et Immunologica Scandinavia 1998*; 106:745-70.

Adamkiewitz TV, Berkovitch M, Krishnan C, Polsinelli C, Kermack D, Olivieri F. Infection due to *Yersinia enterocolitica* in a series of patients with B-thalassemia: incidence and predisposing factors. Clinical Infectious Diseases 1998; 27:1362-6.

Angulo F. *Emergence of highly multidrug resistant Salmonella Newport infections.* Proceedings of a Euroconference: Risk Management Strategies - Monitoring and Surveillance, Dublin, September 7-9 2002.

Anonymous (1). 2002a; http://www.fsai.ie, Food Safety Authority of Ireland.

Anonymous (1). *Annual Report of the National Disease Surveillance Centre, Ireland 2000*. 2000a; National Disease Surveillance Centre (Ireland).

Anonymous. *Annual report on zoonoses in Denmark 2001*. 2002b; www.vetinst.dk, Ministry of Food, Agriculture and Fisheries (Denmark).

Anonymous. Epidat - *Notifications of infectious diseases in the Czech Republic* years 1993-1999. 2000b. http://www.szu.cz/english.html.

Anonymous. *Food Poisoning Report 2002c*. www.mhlw.go.jp, Ministry of Health, Labour, Welfare (Japan).

Anonymous. *Human Annual Report 2000*. National Enteric Pathogens Surveillance Scheme (NEPSS) 2001a; 2/01.

Anonymous. *Infectious Agents Surveillance Report 2000c*; http://idsc.nih.gov-jp.

Anonymous. “Infectious diseases in the Barents and Baltic Sea Regions 2001.” EpiNorth - *Bulletin of the Network for Communicable Disease Control in Northern Europe* 2002d; www.epinorth.org.

Anonymous. *Laboratory Reports*. 2000 d. www.phls.org.uk, Public Health Laboratory Service (UK).

Anonymous. Notifiable Diseases On-Line. 2002e. www.hc-sc.gc.ca, Health Canada.

Anonymous. *National Enteric Surveillance Program. Annual Summary 2000*. 2001b; Health Canada.

Anonymous. *New Zealand Total Diet Survey*, Part 2 Elements. 2000e; Ministry of Public Health, Wellington, New Zealand.

Anonymous. *Outbreak Response and Surveillance Unit*. 2002f. www.cdc.gov, Centers for Disease Control and Prevention.

Anonymous. *Trends and Sources of Zoonotic Agents in animals, Feedingstuffs, Food and Man in Norway 2001. Annual Report*. 2002g. Norwegian Zoonosis Centre.

Anonymous. WHO Surveillance Programme for Control of Foodborne Infections and Intoxications in Europe - 7th Report 1993-1998. 2001c. Federal Institute for Health Protection of Consumers and Veterinary Medicine (BgVV) (Berlin, Germany).

Anonymous. *Zoonoses and zoonotic agents in humans, food, animals and feed in the Netherlands*. 2001d; Inspectorate for Health Protection and Veterinary Public Health in collaboration with the National Institute for Public Health and Environment (RIVM).

Asao T., Kumeda Y, Kawai T., Shibata T, Oda H., Haruki K., Nakazawa H., Kozaki S. “An extensive outbreak of staphylococcal poisoning due to low-fat milk in Japan: estimation of enterotoxin A in the incriminated milk and powdered milk.” *Epidemiology and Infection* 2003; 130 : 33-40.

Baht RV, Moy GG. “Monitoring and assessment of dietary exposure to chemical contaminants.” *World Health Statistics Quarterly 1997*; 50:132-49.

Banatvala N, Hlady WG, Ray BJ, McFarland LM, Thompson S, Tauxe RV. “*Vibrio vulnificus* infection reporting on death certificates: the invisible impact of an often fatal infection.” *Epidemiology and Infection* 1997, 118:221-225.

Bern C, Hernandez B, Lopez MB, Arrowood MJ, Alvarez de Mejia M., De Merida AM, Hightower AW, Venczel L, Herwaldt BL, Klein RE. “Epidemiologic studies of *Cyclospora cayetanensis* in Guatemala.” *Emerging Infectious Diseases* 1999; 5:766-74.

Beuchat LR, Ryu J-H. *Produce handling and processing practices. Emerging Infectious Diseases* 1997; 3:459-65.

Borgdorff MW, Motarjemi Y. *Surveillance of foodborne diseases: what are the options?* 1997; WHO/FSF/97 (Geneva, Switzerland).

Bouvet PJM, Grimont PAD. Données de surveillance 1999 du Centre National de Référence des *Salmonella* et *Shigella*. *Bulletin Epidémiologique Hebdomadaire* 2001; 12.

Bresee JS, Widdowson M-A, Monroe SS, Glass RI. “Foodborne viral gastroenteritis: Challenges and opportunities.” *Clinical Infectious Diseases* 2002; 35: 748-753.

Buzby J.C., T. Roberts, C.T.J Lin and J.M MacDonald. “Bacterial Food Borne Disease: Medical Costs and Productivity Losses.” *Agricultural Economic Report* 741, 1996, United States Department of Agriculture, Washington, DC.

Centers for Disease Control and Prevention. “Economic costs of birth defects and cerebral palsy-United States, 1992.”*Mortality Morbidity Weekly Report* 1995; 44:694-9.

CAST (Council for Agricultural Science and Technology). “Foodborne pathogens: Risk and consequences.” *Task force report* 122. 1994, Ames, Iowa, USA.

Chomel BB, DeBess, EE, Mangiamele DM, Reilly KF, Farver TB, Sun RK, Barrett LR. “Changing trends in the epidemiology of human brucellosis in California from 1973 to 1992: a shift toward foodborne transmission.” *The Journal of Infectious Diseases* 14994; 170 : 1216-1223.

Cowden JM. “Food poisoning notification : time for rethink.” *Health Bulletin* 2000, 58:328-331.

Cowden JM, Lynch D, Joseph CA, O'Mahony M, Mawer SL, Rowe B, Bartlett CLR. “Case-control study of infections with *Salmonella enteritidis* phage type 4 in England.” *British Medical Journal* 1989; 299:771-3.

Daniels NA, MacKinnon L, Bishop R, Altekruse S, Ray B, Hammond RM, Thompson S, Wilson S, Bean NH, Griffin PM, Slutsker L. “*Vibrio parahaemolyticus* infections in the United States, 1973-1998.” *The Journal of Infectious Diseases* 2000; 181 :1661-6.

De Wit MAS, Hoogenboom-Verdegaal AMM, Goosen ESM, Sprenger MJW, Borgdorff MW. “A population-based longitudinal study on the incidence and disease burden of gastroenteritis and *Campylobacter* and *Salmonella* infection in four regions of the Netherlands.” *European Journal of Epidemiology* 2000a; 16:713-8.

De Wit MAS, Koopmans MPG, Kortbeek LM, van Leeuwen NJ, Bartelds AIM, van Duynhoven YTHP. “Gastroenteritis in sentinel general practices, the Netherlands.” *Emerging Infectious Diseases* 2001a; 7:82-91.

De Wit MAS, Koopmans MPG, van der Blij JF, van Duynhoven YTHP. “Hospital admissions for rotavirus infection in the Netherlands.” *Clinical Infectious Diseases* 2000b; 31:698-704.

De Wit MAS, Kortbeek LM, Koopmans MPG, de Jager CJ, Wannet WJB, Bartelds AIM, van Duynoven YTHP “A comparison of gastroenteritis in a general practice-based study and a community-based study.” *Epidemiology and Infection* 2001b; 127:389-97.

Delarocque-Astagneau E, Desenclos J-C, Bouvet P, Grimont PAD. “Risk factors for the occurrence of sporadic *Salmonella enterica* serotype enteritidis infections in children in France: a national case-control study.” *Epidemiology and Infection* 1998; 121:561-7.

Deneen VC, Hunt JM, Paule CR, James RI, Johnson RG, Raymond MJ, Hedberg CW. “The impact of foodborne Calicivirus disease: The Minnesota experience.” *The Journal of Infectious Diseases* 2000; 181:S281-3.

Desenclos J-C A, Klontz KC, Wolfe LE, Hoecherl S. “The risk of *Vibrio* illness in the Florida raw oyster eating population, 1981-1988.” *American Journal of Epidemiology* 1991; 134:290-7.

De Wit MAS, Koopmans MPG, Van Der Blij JF, Duynhoven YTHP. “Hospital admissions for rotavirus infections in the Netherlands.” *Clinical Infectious Diseases* 2000; 31: 698-704.

Döller PC, Dietrich K, Fillip N, Brockmann S, Dreweck C, Vontheim R, Wagner-Wiening C, Wiedenmann A. “Cyclosporiasis outbreak in Germany associated with the consumption of salad.” *Emerging Infectious diseases* 2002; 8:992-994.

Ducoffre G. *Surveillance des maladies infectieuses par un Réseau de Laboratoires de Microbiologie*. Institut Scientifique de la Santé Publique. 2002. www.iph.fgov.be

Effler P, Ieong M-C, Kimura A, Nakata M, Burr R, Cremer E, Slutsker L. “Sporadic *Campylobacter jejuni* infections in Hawaii: associations with prior antibiotic use and commercially prepared chicken.” *The Journal of Infectious Diseases* 2001; 183:1152-5.

Ekdahl K. *Communicable disease in Sweden 2001 - The annual report of the Department of Epidemiology*. Swedish Institute for Infectious Disease Control.

European Commission, Monitoring of pesticide residues in products of plant origin in the European Union, Norway and Iceland, 1999, Report SANCO/397/01-Final.

European Commission. Opinion of the Scientific Committee on Veterinary Measures relating to public health on *Vibrio vulnificus* and *Vibrio parahaemolyticus* (in raw and undercooked seafood). 2001; www.europa.eu.int.

Evengard B, Peterson K, Engman M-L. Wiklund S, Ivarsson SA, Tear-Fahnehjelm K., Forsgren M, Gilbert R, Malm GLow incidence of *Toxoplasma* infection during pregnancy and in newborns in Sweden. *Epidemiology and Infection* 2001; 127:121-7.

Fankhauser RL, Monroe SS, Noel JS, Humphrey CD, Bresee JS, Parashar UD, Ando T, Glass RI Epidemiologic and molecular trends of "Norwalk-like Viruses" associated with outbreaks of gastroenteritis in the United States. *The Journal of Infectious Diseases* 2002; (186):1-7.

Ferguson LR. “Natural and man-made mutagens and carcinogens in the human diet*.” Mutation Research* 1999; 443:1-10.

Fisher IST. The Enter-Net surveillance network - how it works. *Eurosurveillance* 1999; 4:32-55.

Food and Agriculture Organization of the United Nations / World Health Organization. Global Forum of Food Safety Regulators, Marrakech, Morroco, 28-30 January 2002. 2002a; www.who.int/fsf.

Food and Agriculture Organization of the United Nations / World Health Organization. Health implications of acrylamide in food. Report of a Joint FAO/WHO Expert Consultation 2002b.; (Geneva, Switzerland).

Food and Agriculture Organization of the United Nations / World Health Organization. Pan-European Conference on food safety and quality - 25-28 February 2002 / Final report, Budapest, Hungary. 2002c. (Rome, Italy); www.foodsafetyforum.org/paneuropean.Food and Agriculture Organization of the United Nations / World Health Organization. Joint FAO/WHO Expert Consultation on risk assessment of microbiological hazards in foods. Risk characterization of *Salmonella* spp. in eggs and broilers chickens and *Listeria monocytogenes* in ready-to-eat foods. *Food & Nutrition Paper* 72. 2001a; Food and Agriculture Organization of the United Nations (Rome, Italy).

Food and Agriculture Organization of the United Nations / World Health Organization. Joint FAO/WHO Expert Consultation on risk assessment of microbiological hazards in foods. *Food & Nutrition Paper* 71 2000; Food and Agriculture Organization of the United Nations (Rome, Italy).

Food and Agriculture Organization of the United Nations / World Health Organization. Joint FAO/WHO expert consultation on risk assessment of microbiological hazards in food. Hazard identification, exposure assessment and hazard characterization of *Campylobacter* ssp. in broiler chicken and *Vibrio* spp. in seafood. 2001b; WHO/SDE/PHE/FOS/01.4 (Geneva, Switzerland).

Food and Agriculture Organization of the United Nations / World Health Organization / Joint Institute Food Safety and Applied Nutrition. 2002. www.acrylamide-food.org

Food Standards Agency. *A report of the study of infectious intestinal disease in England*. 2000.

FoodNet. *Annual report* - 2000. http://www.cdc.gov.

Frost JA, Gillespie IA, O'Brien SJ. "Public health implications of *Campylobacter* outbreaks in England and Wales." *Epidemiology and Infection* 2002; 128:11-118.

Gallay A, Vaillant V, Boucet P, Grimont P, Desenclos J-C. "How many foodborne outbreaks of *Salmonella* infection occurred in France in 1995 ? Application of the capture-recapture method to three surveillance systems." *American Journal of Epidemiology* 2000; 152:171-7.

Garly ML, Petersen E, Pedersen C, Lundgren JD, Gerstoft J. "Toxoplasmosis in Danish AIDS patients." *Scandinavian Journal of Infectious Diseases* 1997; 29:597-600.

Gerba CP, Rose JB, Haas CN. "Sensitive populations: who is at the greatest risk?" *International Journal of Food Microbiology* 1996; 30:113-23.

Gerber A, Karch H, Allerberger F, Verweyen HM, Zimmerhackl LB. "Clinical course and the role of Shiga toxin-producing *Escherichia coli* infection in the hemolytic-uremic syndrome in pediatric patients, 1997-2000, in Germany: a prospective study." *The Journal of Infectious Diseases* 2002; 186:493-500.

Goulet V, de Valk H, Pierre O, Stainer F, Rocourt J, Vaillant V, Jacquet C, Desenclos J-C. "Effect of prevention measures on incidence of human listeriosis, France, 1987-1997." *Emerging Infectious Diseases* 2001; 7:983-90.

Goulet V, Jacquet Ch, Laurent E, Rocourt J, Vaillant V, De Valk H. "La surveillance de la listériose humaine en France en 1999." *Bulletin Epidémiologique Hebdomadaire* 2001; 34.

Griffin PM, Ostroff M, Tauxe RV, Greene K D, Wells JG, Lewis JH, Blake PA. "Illnesses associated with *Escherichia coli* 0157: H7 infections. A broad clinical spectrum." *Annals of Internal Medicine* 1988; 109:705-12.

Groseclose SL; Hall PA; Knowles CM; Adams DA; Park S; Perry F; Sharp P; Anderson WJ; Snavely K; Fagan RF; Aponte JJ; Jones GF; Nitschke DA; Worsham CA; Glynn MK; Chang M; Doyle T; Jajosky RA, and Noldy S. "Summary of notifiable diseases, Unites States, 1999." Mortality Morbidity *Weekly Report*. 2001; 48:1-104.

Grosse SD, Matte TD, Schwartz J, Jackson RJ. "Economic gains resulting from the reduction on children's exposure to lead in the United States." *Environmental Health Perspectives* 2002; 110:A310-1.

Guzewich JJ, Bryan FL, Todd EC. "Surveillance of foodborne disease I. Purposes and types of surveillance systems and networks." *Journal of Food Protection* 1997; 60:555-66.

Haeghebaert S, Le Querrec F, Vaillant V., Delarocque-Astagneau E., Bouvet P. "Les toxi-infection alimentaires collectives en France en 1998." *Bulletin Epidémiologique Hebdomadaire* 2001b; 15.

Haeghebaert S, Popoff MR, Carlier JP, Pavillon G, Delarocque-Astagneau E. "Caractéristiques du botulisme humain en France, 1991-2000." *Bulletin Epidémiologique Hebdomadaire* 2002; 14.

Haeghebaert S, Vaillant V, Bouvet P, Grimont F, Réseau de néphrologues pédiatres. “Surveillance du syndrome hémolytique et urémique chez les enfants de moins de 15 ans en France en 1999.” *Bulletin Epidémiologique Hebdomadaire* 2001a; 37.

Hald T, Wegener HC. “Quantitative assessment of the sources of human salmonellosis attributable to pork.” In *Proceedings of the 3rd International Symposium on Epidemiology and Control of Salmonella in Pork, 4-7 August 1999*; 200-5 (Washington, USA).

Hall JA, Goulding JS, Bean NH, Tauxe RV, Hedberg CW. “Epidemiologic profiling: evaluating foodborne outbreaks for which no pathogen was isolated by routine laboratory testing: United States, 1982-9.” *Epidemiology and Infection* 2001; 127:381-7.

Havelaar AH, de Wit MAS, van Koningsveld, van Kempen E. “Health burden in the Netherlands due to infection with thermophilic *Campylobacter* spp.” *Epidemiology and Infection* 2000; 125:505-22.

Havelaar AH, Van Duynhoven YTHP, Nauta MJ, Bouwknegt M., Heuvelink AE, De Wit GA, Nieuwenhuizen MGM, Van Der Kar VNAJ. “Disease burden in the Netherlands due to infections with Shiga-toxin producing *Escherichia coli* O157. 2003.” *National Institute for Public Health and Environment (RIVM) Report* 28550008/2003.

Hedberg CW, David MJ, white KE, MacDonald KL, Osterholm MT. “Role of egg consumption in sporadic *Salmonella* enteritidis and *Salmonella* typhimurium infections in Minnesota.” *The Journal of Infectious Diseases* 1993; 167:107-11.

Hedlund KO, Rubilar-Abreu E, Svensson L. “Epidemiology of Calicivirus infections in Sweden, 1994-1998.” *The Journal of Infectious Diseases* 2000; 181 (Suppl.),S275-S280.

Hennessy TW, Hedberg CW, Slutsker L, White KE, Besser-Wiek JM, Moen ME, Feldman J, Coleman WW, Mondson LM, McDonald KL, Osterholm MT. “A national outbreak of *Salmonella* enteritidis infections from ice cream.” *The New England Journal of Medicine* 1996; 334:1281-6.

Herikstad H, Yang S, van Gilder TJ, Vugia D, Hadler J, Blake P, Deneen V, Shiferaw B, Angulo FJ and the FoodNet Working Group. “A population-based estimate of the burden of diarrhoeal illness in the United States: FoodNet, 1996-7.” *Epidemiology and Infection* 2002; 129:9-17.

Hodeshi M, Kazuhiro A, Shunsaku M, Satoshi T, Nobumichi S, Motonobu M, Akio O, Hiroshi Y. “Massive outbreak of Eschrichia O157:H7 infection in schoolchildren in Sakai City, Japan, associated with consumption of white radish sprouts.” *American Journal of Epidemiology* 1999; 150:787-796.

Hlady WG, Klontz KC. “The epidemiology of *Vibrio* infections in Florida, 1981-1993.” *The Journal of Infectious Disease*s 1996; 173:1176-83.

Holmes LB. “Impact of the detection and prevention of developmental abnormalities in human studies.” *Reproductive Toxicology* 1997; 11:267-9.

Hubert B, Dehaumont P, Quenum B, Pignault A. “Les toxi-infections alimentaires collectives en 1989.” *Bulletin Epidémiologique Hebdomadaire* 1990; 16:65-7.

Hutwagner LC, Maloney EK, Bean NH, Slutsker L, Martin SM. “Using laboratory-based surveillance data for prevention: an algorithm for detecting *Salmonella* outbreaks.” *Emerging Infectious Diseases* 1997; 3:395-400.

Iannoccone PM . Toxicogenomics: "The call of the wild chip". *Environmental Health Perspectives* 2001; 109:A8-11.

Inouye S, Yamashita K, Yamadera S, Yoshikawa M, Kato N, Okabe N. “Surveillance of viral gastroenteritis in Japan: pediatric cases and outbreak incidents.” *The Journal of Infectious Diseases* 2000; 181 (Suppl. 2):S270-S274.

International Clearinghouse for Birth Defects Monitoring Systems. *Congenital Malformations Worldwide*. 1991; Elsevier (Amsterdam, The Netherlands).

Kapperud G, Lassen J, Hasseltvedt V. “*Salmonella* infections in Norway: descriptive epidemiology and a case-control study.” *Epidemiology and Infection* 1998; 121:569-77.

Kapperud G, Skjerve E, Bean NH, Ostroff ST, Lassen J. “Risk factors for sporadic *Campylobacter* infections: results of a case-control study in South-Eastern Norway.” *Journal of Clinical Microbiology* 1992; 3117-21.

Käferstein FK, Motarjemi Y, Bettcher DW. “Foodborne disease control : a transnational challenge.” *Emerging Infectious Diseases 1997*; 3(503-510).

Lake RJ, Baker MG, Garrett N, Scott WG, Scott HM. “Estimated number of cases of foodborne infectious disease in New Zealand.” *New Zealand Medical Journal* 2000; 113:278-81.

Lee W-C, Lee M-J, Kim J-S, Park S-Y. “Foodborne illness outbreaks in Korea and Japan studied retrospectively.” *Journal of Food Protection* 2001; 64:899-902.

Levine WC, Smart JF, Archer DL, Bean NH, Tauxe RV. “Foodborne disease outbreaks in nursing homes, 1975 through 1987.” *Journal of the American Medical Association* 1991; 266:2105-10.

Lin M, Roche P, Spencer J, Milton A, Wright P, Witteveen D, Leader R, Merianos A, Bunn C, Gidding H, Kaldor J, Kirk M, Hall R, Della-Porta T. “Australia's notifiable diseases status, 2000.” *Communicable Disease Intelligence* 2002; 26.

Lindsay JA. “Chronic sequelae of foodborne disease.” *Emerging Infectious Diseases* 1997; 3:443-52.

Lopman BA, Reacher MH, van Duijnhoven Y, Hanon F-X, Brown D, Koopmans M. “Viral gastroenteritis outbreaks in Europe, 1995-2000.” *Emerging Infectious Diseases* 2003; 9: 90-96.

Lynt RK, Kautter DA, Read Jr RB. “Botulism in commercially canned foods.” *Journal of Milk and Food Technology* 1975; 38:546-50.

March of Dimes. *Facts and figures of birth defects*. 1999.

McNicolas AM, Kiddle E., Wright J. 1995. “Is New Zealand's recent increase in camplylobacteriosis due to changes in laboratory procedures? A survey of 69 medical laboratories.” *New Zealand Journal of Medicine*, 108 : 459-461.

Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C, Griffin PM, Tauxe RV. “Food-related illness and death in the United States.” *Emerging Infectious Diseases* 1999; 5:607-25.

Michel, P, Wilson JB, Martin SW, Clarke RC, McEwen SA, Gyles CL. « Estimation of the under-reporting rate for the surveillance of *Escherichia coli* 0157:H7 cases in Ontario, Canada.” *Epidemiology and Infection* 2000; 125:35-45.

Michino H, Otsuki K. “Risk factors in outbreaks of foodborne illness originating in schoollunch.” *Journal of Veterinary Medical Science*. 2000; 62:557-560.

Mishu B, Koehler J, Lee LA, Rodrigue D, Hickman Brenner F, Blake P, Tauxe R. “Outbreaks of *Samonella* Enteritidis infections in the United States.” *The Journal of Infectious Disease* 1994; 169: 547-52.

Morris JG, Potter ME. “Emergence of new pathogens as a function of changes in host susceptibility.” *Emerging Infectious Diseases* 1997; 3:435-42.

Mounts AW, Holman RC, Clarke MJ, Bresee JS, Glass RI. “Trends in hospitalizations associated with gastroenteritis among adults in the United States, 1979-1995.” *Epidemiology and Infection* 1999; 123 : 1-8.

Mulder RWAW, Schlundt J. *Safety of poultry meat: from farm to table*. 1999; International Consultative Group on Food Irradiation (ICGFI), FAO/IAEA/WHO.

Murray CJ, Lopez AD. *Global mortality, disability and the contribution of risk factors: Global burden of disease study.* Lancet 1997 A.D.; 349:1436-42.

Murray CJ, Lopez AD. *Mortality by cause for eight regions of the world: Global Burden of Disease Study*. Lancet 1997 B.C.; 349:1269-76.

National Academy of Sciences. *Scientific frontiers in developmental toxicology.* 2000; National Academy Press (Washington, DC, USA).

Obata H, Kai A, Morozumi S. “The trends of *Vibrio parahaemolyticus* foodborne outbreaks in Tokyo: 1989-2000.”*Kansenshogaku Zasshi* 2001; 75:485-9.

Olsen SJ, Bleasdale SC, Magnano AR, Landrigan C, Holland BH, Tauxe RV, Mintz ED, Luby S. “Outbreaks of typhoid fever in the United States, 1960-1999.” *Epidemiology and Infection* 2003, 103 : 13-21.

Powell M, Ebel, E, Schlosser W. 2001. “Considering uncertainty in comparing the burden of illness due to foodborne microbial pathogens.” *International Journal of Food Microbiology*, 69 : 209-215.

Przybylska A. “Foodborne infections and poisonings in Poland in 1999.” *Przegllad Epidemiologiczny* 2001; 55:93-102.

Rees J, Soudain SE, Gregson Norman A, Hugues Richard AC. “*Campylobacter jejuni* infection and Guillain-Barré Syndrome.” *The New England Journal of Medicine* 1995; 333:1371-5.

Rocourt J, Cossart P. *Listeria monocytogenes*. In: *Food Microbiology - Fundamentals and Frontiers*. (M.P. Doyle, L.R. Beuchat and T.J. Montville, Eds.), American Society for Microbiology (Washington DC, USA) 1997; 337-52.

Rodrigue DG, Tauxe RV, Rowe B. “International increase in *Salmonella* Enteritids: a new pandemic?” *Epidemiology and Infection* 1990; 105:21-7.

Rothenberg SJ , *et al.* "Blood lead secular trend in a cohort of children in Mexico City." II. 1990-1995. *Archives of Environmental Health* 2000; 55:245-9.

Ryan MJ, Wall PG, Adak GK, Evans HS, Cowden JM. "Outbreaks of infectious intestinal disease in residential institutions in England and Wales 1992-1994." *Journal of Infection* 1997; 34:49-54.

Schlosser O., Cervantes P. 1998. Faut-il vacciner le personnel exposé aux eaux usées contre la fièvre typhoide? Bulletin Epidémiologique hebdomadaire, 31.

Schlundt J. "Comparison of microbiological risk assessment studies published." *International Journal of Food Microbiology* 2000; 58:197-202.

Schmid H, Burnens AP, Baumgartner A, Oberreich J. "Risk factors for sporadic salmonellosis in Switzerland." *European Journal of Microbiology and Infectious Diseases* 1996; 15:725-32.

Scuderi G, Gabriella S. "A review of the salmonellosis surveillance systems in Italy: evolution during the course of time within the international framework." *European Journal Epidemiology* 2000; 16:861-8.

Shapiro RL, Altekruse S, Hutwagner, Bishop R, Hammond R, Wilson S, Ray B, Thompson S, Tauxe RV, Griffin PM and the *Vibrio* Working Group. "The role of Gulf Coast oysters harvested in warmer months in *Vibrio Vulnificus* infections in the United States, 1988-1996."*The Journal of Infectious Diseases* 1998; 178:752-9.

Smith KE, Besser JM, Hedberg CW, Leano FT, Brender JB, Wicklund JH, Johnson BP, Moore KA, Osterholm MT. "Quinolone-resistant *Campylobacter jejuni* infections in Minnesota, 1992-1998." *The New England Journal of Medicine* 1999; 340:1525-32.

Sneyd E, Lopez L, Eglinton M, McDowell R, Margolin T. *New Zealand Annual Surveillance Summary 2001*. 2002.

Sobel J, Griffin PM, Slutsker L, Swerdlow D, Tauxe RV. "Investigation of multistate foodborne disease outbreaks." *Public Health Reports*. 2002. 117 : 8-19.

Steinberg EB, Green KD, Bopp CA, Cameron DN, Wells JG, Mintz ED. "Cholera in the United States, 1995-2000 : trends at the end of the twentieth century." *The Journal of Infectious diseases* 2001; 184: 799-802.

Stern NJ, Hiett KL, Alfredsson GA, Kristinsson KG, Peiersen JR, Hardardottir H, Briem H, Gunnarsson E, Georgsson F, Lowman R, Berndtson E, Lammerding AM, Paoli GM, Musgrove, MT. *Campylobacter* spp. in *Icelandic poultry operations and human disease*. *Epidemiology and Infection* 2003; 130 : 23-32.

Studahl A., Andersson Y. "Risk factors for indigenous *Campylobacter* infection: a Swedish case-control study." *Epidemiology and Infection* 2000; 125 : 269-269-275.

Swaminathan B, Barrett TJ, Hunter SB, Tauxe RV, CDC PulseNet Task Force. "PulseNet: The molecular subtyping network for foodborne bacterial disease surveillance, United States." *Emerging Infectious Diseases* 2001; 7:382-9.

Swaminathan B, Matar GM. “Molecular typing methods. 1993”; in: *Diagnostic molecular microbiology, principles and application* (DH Persing, TE Smith, FC Tenover and TJ White, eds.), *American Society of Microbiology* (USA).

Tappero JW, Schuchat A, Deaver KA, Mascola L, Wenger J. “Reduction in the incidence of human listeriosis in the United States: Effectiveness of prevention efforts?” *Journal of the American Medical Association* 1995; 273:1118-22.

Tauxe RV. “Emerging foodborne diseases: An evolving public health challenge.” *Emerging Infectious Diseases* 1997; 3:425-34.

Tauxe RV. “Surveillance and investigation of foodborne diseases - Roles of public health in meeting objectives for food safety.” *Food Control* 2001; 78:31-41.

Thomson G, DeRubeis D, Hodge M, Rajanayagam C, Inman RD. “Post-*Salmonella* reactive arthritis: late clinical sequelae in a point source cohort.” *The American Journal of Medicine* 1995; 98:13-9.

Thornley C, McDowell R, Lopez L, Baker M. *Annual Summary of outbreaks in New Zealand* 2001. 2002.

Tompkins DS, Hudson MJ, Smith HR, Eglin RP, Wheeler JG, Brett MM, Owen RJ, Brazier JS, Cumberland P, King V, Cook. “PEA study of infectious intestinal disease in England: microbiological findings in cases and controls.” *Communicable Disease and Public Health* 1999; 2:108-13.

United States Environmental Protection Agency. www.epa.gov.

Vaillant V, Caumes E, De Valk H, Mesnage V, Griffon AM. “Intoxication alimentaire a la ciguatera: savoir l'évoquer même en l'absence de voyage.” *Bulletin Epidémiologique Hebdomadaire* 2001; 38.

Verhaegen J, Charlier J, Lemmens P, Delmée M, Van Noyen R, Verbist L, Wauters G. “Surveillance of human *Yersinia enterocolitica* infections in Belgium: 1967-1996.” *Clinical Infectious Diseases* 1998; 27:59-64.

Wall PG, de Louvois J, Gilbert RJ, Rowe B. “Food poisoning: notifications, laboratory reports, and outbreaks - where do the statistics come from and what do they mean?” *Communicable Disease Report Review* 1996; 6:R93-R100.

Wang Y, Thornton I, Farago M. “Changes in lead concentrations in the home environment in Birmingham, England over the period 1984-1996.” *The Science of the Total Environment* 1997; 207:149-56.

Watanabe T. “Reduced cadmium and lead burden in Japan in the past 10 years.” *International Archives of Occupational and Environmental Health* 1996; 68:305-14.

Wheeler JG, Sethi DM, Cowden J, Wall PG, Rodrigues LC, Tompkins DS, Hudson MJ, Roderick PJ on behalf of the Infectious Intestinal Disease Study Executive. “Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance.” *British Medical Journal* 1999; 318:1046-50.

World Health Organization. *Biomarkers in Risk Assessment: Validity and Validation* 2001a.; Environmental Health Criteria - EHC 222 (Geneva, Switzerland).

World Health Organization. GEMS/Food Total Diet Studies, *Report of the 2nd International Total Diet Workshop, 4-14 February 2002, Brisbane, Australia 2002*; (Geneva, Switzerland).

World Health Organization. *Global surveillance of foodborne disease: developing a strategy and its interaction with risk analysis - Report of a WHO consultation, Geneva, Switzerland 26-29 November 2001.* 2002 a, WHO/CDS/CSR/EPH/2002.21 (Geneva, Switzerland).

World Health Organization. *Methods for foodborne disease surveillance in selected sites - report of a WHO consultation 18-21 March 2002, Leipzig, Germany*. 2002 b, WHO/CDS/CRS/EPH/2002.22 (Geneva, Switzerland).

World Health Organization. *The increasing incidence of human campylobacteriosis.* 2001; WHO/CDS/CSR/APH 2001.7 (Geneva, Switzerland).

World Health Organization. *Infant exposure to certain organochlorine contaminants from breast milk: a risk assessment,* GEMS/Food international dietary survey. 1998; (Geneva, Switzerland).

World Health Organization. *Methods on foodborne disease surveillance in selected sites - 18-21 March,* Leipzig (Germany). in press b.

World Health Organization. *Neurotoxicity risk assessment for human health: Principles and approaches* 2001b.; Environmental Health criteria - EHC 223 (Geneva, Switzerland).

World Health Organization. *Principles and methods for assessing allergic hypersensitization associated with Exposure to Chemicals* 1999 A.D.; Environmental Health Criteria - EHC 212 (Geneva, Switzerland).

World Health Organization. *Principles and methods for assessing direct immunotoxicity associated with exposure to Chemicals* 1996; Environmental Health Criteria - EHC 180 (Geneva, Switzerland).

World Health Organization. *Principles for the assessment of risks to human health from exposure to chemicals.* 1999b.; Environmental Health Criteria (EHC) 210 (Geneva, Switzerland).

World Health Organization. *Toxic oil syndrome: current knowledge and future perspectives.* WHO Regional Publications European Series 1992; 42:Geneva (Switzerland).

World Health Organization. *Understanding the BSE threat. WHO/CDS/CSR/EPH/2002*. 2002 c Geneva (Switzerland). a.

World Health Organization / United Nation Environment Protection. *Public health impact of pesticides used in agriculture.* 1990; (Geneva, Switzerland).

Yazdanpanah Y, Beaugerie L, Boelle PY, Letrillart L, Desenclos J-C, Flahault A. “Risk factors of acute diarrhea in summer - a nation-wide French case-control study.” *Epidemiology and Infection* 2000; 124: 409-416.

PART II: THE COSTS OF FOODBORNE DISEASE

EXECUTIVE SUMMARY

66. This study provides a brief survey of the literature on the economic costs typically associated with foodborne disease, discusses some common methodologies employed to quantify these costs and presents some quantitative estimates to show their approximate magnitude and policy importance. Differences about what costs to measure and how to measure them, combined with serious data limitations, make comparisons across pathogens, over time or among countries extremely difficult. Still, for policy-makers faced with decisions based on cost-benefit analysis of measures to reduce foodborne disease, it is essential to have the best possible estimates of the economic costs involved.

67. Increasing demands for regulatory accountability have required governments to make greater use of cost-benefit analysis in evaluating policy changes. Some studies consider the aggregate cost of incidences of a single disease with its complete elimination as the point of reference. Others focus on evaluating the costs and benefits of alternative government programs, which are aimed at reducing the prevalence of pathogens in the food supply. There are two approaches for evaluating the economic costs of (or benefits of a reduction in) foodborne disease: cost-of-illness (COI) and willingness-to-pay (WTP).

68. The cost-of-illness approach (COI) is based on the premise that the reduction in national output, which arises as a consequence of an incidence of a foodborne-disease measures the reduction in welfare that it causes. An accounting approach is adopted which sums up medical expenses, foregone earnings of affected individuals and associated productivity losses to employers. The COI approach concentrates only on the direct costs incurred by those actually suffering from the disease and ignores the benefit that every individual experiences as a result of having to devote less resources to achieving their preferred health status. It also fails to recognise that consumers willingly accept some reduction in their health status (i.e. higher health risk) because the costs of obtaining minimum risk are prohibitive at the margin.

69. The willingness-to-pay (WTP) approach attempts to estimate the value society places on publicly provided risk reduction by estimating an individual's willingness-to-pay for reductions in risk. The method essentially combines a monetary evaluation of the disutility of being ill with the estimated cost-of-illness, together with an estimate of the preventative expenditure an individual is willing to pay for a given pathogen level. Although more difficult to apply, the WTP approach can give a broader estimate of economic costs than COI because it makes it possible to include quality-related aspects that cannot be translated into identifiable short-term illnesses (*i.e.* preventative expenditures).

70. The study also examines some of the additional economic considerations of foodborne disease, incurred by industry (beyond productivity losses) or, in some cases, governments, including disease eradication, litigation costs, product recalls, market impacts and the impact on value of firms. While the economic framework for such studies is less well developed, these costs can be very significant and should be taken into consideration when designing food safety regulations and, in particular, when carrying out a full cost-benefit analysis of regulatory options.

71. Empirical evidence is rather piecemeal and non-comparable but the common message is that the economic costs associated with foodborne disease represent a significant economic burden on consumers, the food industry and governments. The study concludes by suggesting there is a need to improve our understanding of these economic costs to allow policy makers to design appropriate prevention strategies to lower health risk.

I. INTRODUCTION

72. Reports by the World Health Organisation (WHO) have concluded that, despite improvements in many areas, foodborne disease caused by microbiological hazards and chemical contaminants continues to be a growing public health concern (WHO, 2002; OECD, 2002). The direct and indirect economic costs[8] associated with foodborne disease are known to be high but actual quantitative estimates are difficult to obtain. Differences about what costs to measure and how to measure them, combined with serious data limitations, make comparisons across pathogens, over time or among countries extremely difficult. Still, for policy-makers faced with decisions based on cost-benefit analysis of measures to reduce foodborne disease, it is essential to have the best possible estimates of the economic costs involved.

73. This consultant's report is intended to complements an earlier WHO study, commissioned by the OECD on the incidence of foodborne disease in the OECD area [AGR/CA/APM(2002)28]. It provides a brief survey of the literature to identify the types of economic costs typically associated with foodborne disease, discusses some common methodologies employed to quantify these costs and presents some quantitative estimates of economic costs to show their approximate magnitude and policy importance.

74. The objective of this report is to draw attention to the limited development of a solid conceptual framework and relative lack of information on the economic costs of foodborne disease. The report consists of three main sections. Section II examines various approaches to measuring what are generally considered the main costs of foodborne disease - medical costs and productivity losses while Section III provides some empirical results from a number of OECD Member countries. Section IV goes on to briefly examine the additional costs to the agri-food industry associated with foodborne disease such as disease eradication, litigation costs, product recalls and longer term market impacts.

II. APPROACHES TO MEASURING THE ECONOMIC COSTS OF FOODBORNE DISEASE

75. When determining the economic costs of foodborne disease, it is important to begin by placing the measure in context. Increasing demands for regulatory accountability have required governments to make greater use of cost-benefit analysis in evaluating policy changes (Caswell, 1998). This method is based on the Kaldor-Hicks compensation principle which states that a policy intervention is justified if the beneficiaries could potentially compensate the losers and still leave both groups better off. The application of the method therefore begins with a clear understanding of the nature of the policy intervention and an identification of the beneficiaries and the losers. Without such a framework the meaning of the measure of cost is very likely to be ambiguous and may result in double counting.

76. There are many different methods used to evaluate the costs of foodborne disease. These differences severely compromise the degree to which the estimates obtained from such studies can be compared. In part, this is a natural consequence of the fact that many studies have different objectives, or more specifically, different points of reference to which the *status quo* is compared. For example, some studies (Sockett, 1995) consider the aggregate cost of incidences of a single disease with its complete elimination as the point of reference. Others (Buzby *et al, 1997*) focus on the evaluating the costs and benefits of alternative government programs which are aimed at reducing the prevalence of pathogens in the food supply, and here the point of reference is the alternative policy options.

---

[8] The distinction between direct and indirect costs is itself a source of ambiguity in measures of the cost of foodborne disease. Many of the studies reviewed below classify costs differently and insufficient detail is publish to attempt harmonisation.

77. Broadly speaking there are two approaches for evaluating the economic costs of (or benefits of a reduction in) foodborne disease: cost-of-illness (COI) and willingness-to-pay (WTP). The basic economic theory which serves as a framework for these approaches is described in Annex I.

Cost-of-Illness Approach

78. The cost-of-illness approach (COI) is based on the premise that the reduction in national output, which arises as a consequence of an incidence of a foodborne-disease measures the reduction in welfare that it causes. An accounting approach is adopted which sums up medical expenses, foregone earnings of affected individuals and associated productivity losses to employers. Roberts and Sockett (1994) argue that the costs to an individual or family of a foodborne disease (in this case salmonellosis) are represented by the lost opportunities to carry out day-to-day activities, the expenses caused by the illness and the pain, suffering and occasionally death that arise from the disease. Once the costs to the individual of varying degrees of severity of infection have been estimated they are combined with epidemiological information on cases and outbreaks of disease to estimate the aggregate costs. One problem with this approach is that the epidemiological information is notoriously underreported, for example, Busby *et al* (1997) note that only 1-5% of cases are reported in the US; Roberts and Socket (1994) suggest a range of 1-10% in the UK. For the United States, Mead *et al.* (1999) have estimated the actual number of acute foodborne illnesses, hospitalizations and deaths and these numbers can be used in economic analysis. Missing from the Mead et al. numbers are the chronic complications that occur in a small percentage of cases, but are associated with most pathogens (Foegeding and Roberts, 1994). These are important since the economic costs of possibly life-long complications, such as kidney failure, mental retardation, and paralysis, have a high economic cost to society (see table at end of review).

79. Some authors (Buzby *et al,* 1996) extend the list of costs to include the costs to industry. They argue that production and regulatory costs arising from the existence of the disease should be included in the COI. The differences between the lists of costs that are included in the measures developed by Roberts and Sockett (1994) and by Buzby *et al* (1996) illustrate the point made in the introduction about the lack of comparability between alternative measures arising from differences in the aims of the studies. Because Buzby *et al* are interested in comparing the costs and benefits of alternative programs, one of the benefits of moving from the *status quo* is an elimination of the costs associated with the existing regulatory programme.

80. The major criticism of the COI approach is that it provides only a partial estimate of the economic costs. The COI approach concentrates only on the direct costs incurred by those actually suffering from the disease and ignores the benefit that every individual experiences as a result of having to devote less resources to achieving their preferred health status. It also fails to recognise that consumers willingly accept some reduction in their health status (*i.e.* higher health risk) because the costs of obtaining minimum risk are prohibitive at the margin. Berger *et al* (1987) argue that an approach more closely based on a microeconomic model of consumer optimisation is more appropriate.

81. Whilst the COI approach can be criticised, it has some practical advantages. While the amount of data required (*e.g.* incidence of disease) may be substantial, it is often readily available in most developed countries. However, the problem of under reporting noted above means that considerable effort has to be applied in obtaining accurate estimates. For example, a one-year long study in the UK by the Food Standards Agency (FSA 2000a) based on a sample of 4 888 people estimated that 20% of the population suffered from infectious intestinal disease of which only 3% saw a physician.

Willingness-to-Pay Approach

82. The willingness-to-pay (WTP) approach attempts to estimate the value society places on publicly provided risk reduction by estimating an individual's willingness-to-pay for reductions in risk. The method essentially combines a monetary evaluation of the disutility of being ill with the estimated cost-of-illness, together with an estimate of the preventative expenditure an individual is willing to pay for a given pathogen level. Empirical studies which use the WTP approach emphasise its usefulness in evaluating the intangible costs associated with foodborne disease. Although more difficult to apply, the WTP approach can give a broader estimate of economic costs than COI because it makes it possible to include quality-related aspects that cannot be translated into identifiable short-term illnesses (*i.e.* preventative expenditures). Caswell (1998) considers the alternative methods available in practice for estimating WTP.

83. The most common method is to use contingent valuation or experimental auctions. The methods essentially offer the consumer the choice between two products one of which is subject to a 'normal' risk of being contaminated with a pathogen and the other which is guaranteed pathogen free. Elicitation methods are used to evaluate the consumers WTP for the latter product. In a study of this type, Shin *et al. (1992)* argue that WTP is a measure of the morbidity cost associated with the disease and that this should be added to direct costs obtained with a cost-of-illness approach to obtain the total cost associated with the disease.

84. Conjoint analysis or choice experiments are sometimes argued to more accurately represent the consumers shopping experience. Participants are presented with an array of consumption options all of which have differing attributes and in particular will include food safety and price. By observing consumers choices between the different attribute bundles it is possible to elicit the value placed on each attribute. To obtain accurate measures for food safety, conjoint analysis requires that consumers be provided with full information on acute foodborne disease outcomes, including their duration, severity, and the probability of complications that may be life-long.

85. Hedonic pricing methods relate the price paid for a good to a bundle of attributes including food safety. Using this approach, it is possible to apportion the price paid between these attributes. It has also been proposed that because jurors are also food consumers, the value of damages awarded against parties liable for the introduction of a food borne pathogen can be taken as a measure of WTP. Finally Caswell notes that the benefits of improved access to foreign markets that would result from a lowering of non-tariff barriers to trade may provide a measure of WTP. Thus under an assumption of competitive markets, the costs incurred by a country in denying access to its markets can be taken to be a measure of that countries willingness to pay to avoid the disease risk associated with the potential imports. Clearly the assumption is unrealistic however as many food import restrictions are in place for reasons other than to ensure food safety.

Comparing COI and WTP Approaches

86. A number of attempts have been made to develop theoretical models that allow the relationship between WTP and COI to be compared. Berger *et al.* (1987) develop a general framework in which to evaluate an individual's willingness-to-pay for changes in their health status and show that WTP and COI are only equivalent under extremely restrictive circumstances.[9] Berger *et al.* conclude that there are "no plausible assumptions which can be made to simplify the WTP measure to COI".

---

[9] In particular, when the individual derives utility from a given health status directly in addition to the consumption possibilities that it allows and when an individual spends money defensively in order to avoid illness, the COI measure is inappropriate.

87. Antle (2000a) considers WTP in the more specific context of food safety. The approach he adopts is more comprehensive than that in Berger *et al* (1987). He analyses the whole market for food safety in an attempt to better understand the nature of the market failure and to determine more clearly the case for market regulation. He argues that food safety can be modelled as a quality attribute of food but that it is subject to unusual information asymmetries where producers and consumers are probably both ill informed to varying degrees. The demand side of the model is similar to that of Berger *et al* except that it makes health status dependent on the individual's food consumption. From the model of demand, Antle is able to demonstrate that COI is an underestimate of WTP.

88. These theoretical models show, at least conceptually, that WTP is the more accurate measure of the cost of foodborne disease. They also suggest there is a danger of double counting in the empirical method adopted by Shin *et al.* (1992) in which WTP is seen as supplementary to COI. The work of Antle also shows that a clear understanding of the 'market' for food safety is required if we are really to understand the case for market regulation. It implies that we take a step back from the application of cost-benefit analysis of a given intervention to consider what the best form of intervention is for tackling the specific problem that arises in the case of food safety.

89. Whilst it can be argued that WTP is preferable to COI on theoretical grounds, there are some impediments to its practical implementation. In particular it is possible that, where a consumer does not bear the full cost of the disease, their WTP may not equate to the theoretical concept. One approach that may be fruitful here is through the use of choice experiments where the implications of changed public expenditure that results from changes in the regulatory regime may be incorporated into the attribute bundles offered to the consumer.

III. EMPIRICAL ESTIMATES OF THE ECONOMIC COSTS OF FOODBORNE DISEASE

90. The following studies were selected to illustrate the diverse range of empirical approaches that have been used to estimate the economic costs of foodborne disease, and to provide some evidence on the relative magnitude of those costs.

Australia

91. Food Standards Australia New Zealand (formerly ANZFA) estimated the cost of foodborne illness to the Australian community using a COI approach. The number of incidences is estimated using three different surveys, and the commonly used adjustment factor of 10 applied to the notified cases, to extrapolate to the population. Three out of the four methods (including the adjusted notified cases) give an estimate between 4 and 5.4 million cases per year. Direct costs of a food borne disease incidence are based on the productivity loss and medical costs, including hospitalisation. These costs are then doubled to account for the indirect costs giving an average cost for each illness of AUD 630 (USD 366). Combining this figure with the number of incidences gives a total cost of AUD 2.6 billion (USD 1.5 billion) per year. More recent work (Food Science Australia 2002) reports the cost of foodborne illness in Australia to AUD 1.67 billion (USD 0.96 billion) a year.[10]

92. In 2001 the Department of Health and Ageing commissioned a national study entitled *Food Safety Management Systems, Costs, Benefits and Alternatives* (Australian Department of Health and Ageing, 2001). This report outlines the costs and benefits of a range of food safety management systems and implementation strategies. In Australia enhanced surveillance for foodborne illness is undertaken by OzFoodNet, a collaborative project with Food Standards Australia New Zealand (FSANZ) and State and

---

[10] A detailed description of the composition of this figure is given in the cited report.

Territory and Commonwealth health authorities OzFoodNet, through the National Centre for Epidemiology and Population Health, conducted a national survey of gastroenteritis during 2001-2002. Of the 17.2 million cases of gastroenteritis each year in Australia, there are 5.4 million cases that are conservatively estimated to be due to contaminated food resulting in the loss of 6.5 million days of paid work. When the calculations (COI approach) in the *Food Safety Management Systems, Costs, Benefits and Alternatives* report are adjusted for the most recent and more accurate estimation of foodborne illness, the estimated cost to Australia is AUD 3.75 billion (USD 1.99 billion) annually.

Canada

93. Todd (1989a) used a COI approach to estimate the annual average cost of a comprehensive range of foodborne diseases in Canada for the period 1978-82. Based on six different methods to adjust for underreporting (from .06 to 4% of cases reported), he used the median of 350 to scale up reported incidence of each disease. The study differentiated between incidences that arise in food processing and those that arise elsewhere and estimated the costs per case of each disease by examining 67 incidents across North America and one case from the UK over the 1962-85 period, converted to 1985 prices. The types of economic costs measured included the loss of business to the food producer, retailer, or server; medical care and hospitalisation of the cases; income lost because of illness or carrier state, and the value of homemakers' time and leisure time and costs of the investigation of the outbreak. Table 1 presents the average cost per case for the range of diseases considered.

94. To account for deaths arising from foodborne disease, the study combined valuations of life for individuals of differing ages and sexes with epidemiological information on the age and sex profile of deaths caused by the disease to produce an a average valuation of foodborne fatalities. As epidemiological data is only available for *salmonellosis, typhoid fever, shigellosis* and *parasitic helminthic* diseases, the average cost of a fatality arising from all other diseases was assumed to be the same as that resulting from *salmonellosis*. The number of fatalities arising for each disease is calculated by taking the ratio of reported fatalities to reported cases of the disease and multiplying this by the actual number of cases computed in the manner described above.

95. The total cost of foodborne disease in terms of illness and death in Canada was CAD 1.334 billion (USD 977) on average between 1978 and 1982. While methodologies were not described, a study by Curtin (1984), cited in Buzby *et al.* (1996), estimated the annual cost of human *salmonellosis* to be CAD 84 million (USD 65 million), while Mayers and Couture (1999), cited in Food Standards Australia and New Zealand (2002), estimated the average cost per case of foodborne illness in Canada was about CAD 1 000 (USD 675).

Table 1. Estimates of Average Costs of Foodborne Disease in Canada (Todd, 1989)

Etiologic agent	Average cost per case arising from mishandling at food-service establishments, markets, homes, farms and in the community (1985 $).	Average cost per case arising from mishandling at food processing establishments, (1985 $).
Salmonella	1 067 (USD 781)	118 925 (USD 87 061)
Clostridium perfringens	246 (USD 180)	
Staphylococus aureus	12 909 (USD 9 450)	24 040 (USD 17 599)
Bacillus cereus	391 (USD 286)	
Shigella	375 (USD 275)	
Campylobacter jejuni	1 331 (USD 974)	
Eschericia coli	3 276 (USD 2 398)	
Clostridium botulinum	23 904 (USD 17 499)	8 181 242 (USD 5 989 196)[1]
Salmonella typhi	10 139 (USD 7 422)	349 786 (USD 256 066)
Giardia lamblia	9 536 (USD 6 981)	
Listeria monocytogenes		11 543 (USD 845)
Ciguatera poison	3 383 (USD 2 477)	
Solanine from potatoes	24 (USD 18)	
Average	6 220 (USD 4 553)	

1. Includes loss of business and recall costs.

Finland

96. Peltola *et al.* compared the results of a WTP study with previous estimates from the COI approach with respect to the Finnish Salmonella Control Program. According to a survey, respondents would be willing to spend an additional EUR 70 (USD 66) annually to finance the current level of salmonella control in Finland. Previously, benefits were estimated to be about EUR 3.5 (USD 3.3) annually using COI approaches.

Germany

97. Krug and Rehm (1983), cited in Roberts and Sockett (1994) and Todd (1989), is a cost-benefit study of human salmonellosis in West Germany in 1983. It was assumed that 81% of the human cases were derived from consumed foods. The incidence of foodborne disease was established at 13.4 times the number of reported cases, for a total of 385 545 cases. Using a COI approach, the average cost per case was estimated at DM 161 (USD 130), for a total national cost of about DM 62 million (USD 50 million).

New Zealand

98. Scott *et al.* (2000) and Roberts *et al.* (2000) conducted a COI study for 10 infectious food borne diseases in New Zealand. They break incidences of the diseases into three categories: Do not present to the public health system; Visit the general practitioner (GP) and Hospitalisation with the final category broken down into the subcategories: recover; long-term illness and death. The number of people presenting to the GP are adjusted for underreporting by a factor 10 for salmonellosis, shigellosis and yersinosis, and a factor

of 5 for campylobacteriosis. The costs per case in $NZ reported in Table 2 includes direct medical costs; direct non-medical costs and the costs of reduced productivity.

Table 2. Estimated Unit Costs of Infectious Foodborne Disease in New Zealand (Scott, 2000)

Disease	Cost per case ($NZ)
Campylobacteriosis	533 (USD 242)
Salmonellosis	526 (USD 239)
Shigellosis	253 (USD 115)
Yersinosis	891 (USD 405)
Listeriosis	55 434 (USD 25 197)
Verotoxigenic *Escherichia Coli*	10 231 (USD 4 650)
Typhoid	3 834 (USD 1 743)
Hepatitis	4 432 (USD 2 015)
Toxins	221 (USD 100)
Small structured round viruses	204 (USD 93)

99. The total cost of foodborne disease cases was estimated at NZD 55.1 million (USD 25 million), consisting of NZD 2.1 million (USD 0.9 million) direct medical costs, NZD 0.2 million (USD 0.1 million) direct non-medical costs, NZD 48.1 million (USD 22 million) in lost productivity, and NZD 4.7 million (USD 2.1 million) in intangible cost of loss-of-life. The incidence of *campylobacteriosis* accounts for most of the foodborne illness costs. The total cost of potentially foodborne infectious disease was estimated to be NZD 88.8 million (USD 40.4 million). An estimate of NZD 215.7 million (USD 98 million) for all cases of infectious intestinal diseases, including non-foodborne pathogens or for which no pathogen was identified, suggests foodborne illness was accountable for about 40% of the total IID costs.

Sweden

100. Lindqvist *et al.* (2001) study the incidence, causes and costs of foodborne illnesses in a one-year (1998/99) study of the municipality of Uppsala, Sweden. The estimated average cost per illness was SEK 2 164 (USD 246) to society and SEK 500 (USD 57) to the patient. The annual cost of foodborne illnesses in Sweden was estimated to be SEK 1 082 million (USD 123 million).

United Kingdom

101. Roberts and Sockett (1994) applied a COI approach to *Salmonella* infections in the UK. By examining survey evidence, they derive a multiplication factor of 38 to apply to the notified cases to obtain actual incidence. They used two approaches to compute the average cost of infection. One categorises patients according to the medical care that they receive and the other uses a subjective measure of severity that is provided by the respondents in the survey. Combining these estimates with the incidence data gave an estimated range for overall cost of GBP 350-502 million (USD 538-772 million). Sockett (1993) also reports an estimated range of values for the COI of salmonella infection of GBP 231 – 331 million (USD 355-409 million).

102. The Food Standards Agency (FSA, 2000a) examined the costs of infectious intestinal disease (IID) in England, based on a detailed survey. Clearly, foodborne disease does not map perfectly onto this category but there is a strong relationship. In terms of computing the COI, it is notable that the survey differentiates between social classes and collected actual data on the costs incurred by individuals. Table 3 presents the average costs for the diseases covered, distinguishing between the costs incurred for those who present to the GP and those that do not.

Table 3. Average Costs per Head of Infectious Intestinal Disease in England (Food Standards Agency, 2000)

Organism	Average costs for those presenting to GP (GBP)	Average costs for those not presenting to GP (GBP)
Salmonella	76.50 (USD 117.15)	3.75 (USD 5.67)
Campylobacter	274.77 (USD 420.78)	28.17 (USD 43.14)
Enterovir *E. Coli*	188.74 (USD 289.03)	30.80 (USD 47.17)
C. Dificile	44.90 (USD 68.76)	41.05 (USD 62.86)
Rotavirus	74.59 (USD 114.23)	0
Small structured round viruses	151.02 (USD 231.27)	14.48 (USD 22.17)

103. The overall cost of IID in the UK is estimated by the FSA at GBP 742.8 million (USD 1 137.5 million). A very limited WTP study was also carried out by the FSA. This was largely of a qualitative nature. It found that 60% of respondents were willing to pay more for safer food, with about one-half willing to pay up to 10% more to ensure 'the lowest possible risk'.

United States

COI Approach

104. Todd (1989b) carried out a similar study for the US as that discussed above for Canada. The study uses a number of estimates for the incidence of foodborne disease, including the factor of 350 applied in the Canadian study to scale-up the reported incidences. Moreover, the estimated costs per incidence are very similar to those obtained for the Canadian study. The conclusion reached is that the cost of illness, death and business loss due to foodborne disease is USD 8.4 billion. Microbiological diseases (bacterial and viral) represent 84% of the US costs, with salmonellosis and staphylococcal intoxication being the most economically important diseases (USD 4 billion and USD 1.5 billion per year, respectively).

105. Buzby *et al.* (1996) use a COI approach to evaluate the costs of foodborne disease in the US for six bacterial pathogens all found in animal products - *Salmonella, Campylobacter jejuni, Escherichia coli 0157:H7, Listeria monocytogenes, Staphylococcus aureus and Clostridium perfringens*. For the first four diseases, the incidence and corresponding costs were broken down into a number of severity categories. In the cases of *E* coli disease and *listeria* some chronic complications were also considered. In converting reported cases to actual cases for salmonella a factor of between 10 and 100 was used, following the work of Chalker and Blaser (1988) and it is interesting to note that this factor is significantly lower than that used by Todd. For the other diseases, detailed epidemiological information was used to obtain estimates of the true level of incidence, which on the whole are claimed to be conservative.

106. Table 4 provides the estimates obtained by Buzby *et al.* (1996) together with the costs per case, excluding death, where the estimates are weighted averages across the severity categories. In addition, it is estimated that the COI associated with Guillain-Barre syndrome[11] is USD 358.8 million (see also Buzby *et al.*, 1997). The overall costs estimated by Buzby *et al.* (1996) are USD 2.9- 6.7 billion per year. The study suggests microbial pathogens in food cause an estimated 6.5 to 33 million cases of human illness and up to 9 000 deaths in the United States each year.

---

[11] Guillain-Barre syndrome is a complication arising from infection with foodborne Campylobacter.

Table 4. Costs per Case and Total Costs of Foodborne Disease in the US (Buzby *et al.,* 1996)

Pathogen	Cost per case (1993 USD)	Total Cost (1993 USD billion)
Campylobacter jejuni or *coli*	437	0.6-1.0
Clostridium perfringens	6 487	0.1
Eschericia coli	5 000	0.2-0.6
Listeria monocytogenes	-	0.2-0.3
Salmonella	523	0.6-3.5
Staphylococcus aureus	763	1.2

107. Buzby and Roberts (1997) report that seven foodborne pathogens (Campylobacter jejuni, Clostridium perfringens, Escherichia coli 0157:H7, Listeria monocytegenes, Salmonella, Staphylococcus aureus, and Toxoplasma gondii) cost the US an estimated USD 6.5-34.9 billion (1995 dollars) annually. The wide differences between this estimate and that of Busby *et al.* (1996) is due more to the methodology than to the inclusion of the additional pathogen Toxoplasma gondii.[12]

108. More recent work by the US Centers for Disease Control and Prevention (CDC) estimates that foodborne diseases cause approximately 76 million illnesses, 325 000 hospitalizations, and 5 000 deaths each year (Mead *et al*., 1999). ERS has estimated the annual US economic costs incurred for five major bacterial pathogens: *Escherichia coli* O157 and other STECs (and associated hemolytic uremic syndrome), *Campylobacter* (and associated Guillain-Barré syndrome), *Listeria monocytogenes*, and *Salmonella*. ERS has developed outcome trees for the illnesses caused by those pathogens, showing the costs incurred and the number of cases by the severity of disease: no physician visit, physician visit, hospitalization, premature death, and chronic complications. The current ERS methodology is a combination of the COI approach for valuing acute illnesses and WTP approach for valuing deaths. The latest ERS estimates of medical costs, productivity losses, and value of premature deaths for diseases caused by five foodborne pathogens is USD 6.9 billion per year (Table 5).

109. ERS is developing a cost-calculator to show how different assumptions change the estimates of the costs of foodborne disease. Salmonellosis is the first disease for the cost-calculator and will be available on the ERS website in early 2003.

---

[12] This wide range is attributable to alternative methods of valuing a human life. The Landefeld and Seskin method used in the 1996 and 1997 Buzby *et al.* studies estimated ranges of USD 9.3-12.9 billion and USD 6.5-13.3 billion, respectively. The 1997 study also used a hedonic method to value life for a second estimate which resulted in a range of USD 19.7-34.9 billion. Thus, the range of USD 6.5-34.9 billion is the outer limits of the two methods of estimation.

Table 5. Estimated annual costs due to selected foodborne pathogens, 2000[1]

Pathogen	Estimated annual foodborne illnesses[2]			Costs[3]
	Cases	Hospitalizations	Deaths	
	Number			*USD billion*
Campylobacter spp	1 963 141	10 539	99	1.2
Salmonella	1 341 873	15 608	553	2.4
E. coli O157	62 458	1 843	52	0.7
E. coli, non-O157 STEC	31 229	921	26	0.3
Listeria monocytogenes	2 493	2 298	499	2.3
Total	3 401 194	31 209	1 229	6.9

1. These estimates of foodborne illness costs are based on new data and improved methodologies for valuing costs, so they are not directly comparable to earlier ERS estimates of the costs of foodborne disease.
2. Data from the Centers for Disease Control and Prevention, Mead *et al*., 1999, Food-Related Illness and Death in the UnitedStates(http://www.cdc.gov/ncidod/eid/vol5no5/mead.htm).
3. The total estimated costs include specific chronic complications in the case of *Campylobacter* (Guillain-Barré syndrome), *E. coli* O157 (hemolytic uremic syndrome), and *Listeria monocytogenes* (congenital and newborn infections resulting in chronic disability or impairment). Estimated costs for *Listeria monocytogenes* exclude less serious cases that do not require hospitalization.

Source: http://www.ers.usda.gov/Briefing/FoodborneDisease/features.htm.

Willingness to Pay (WTP) approach

110. Giamalva *et al.* (1997) report the results of a non-hypothetical laboratory experiment, where participants were found to be willing to pay an average of USD 0.71 for the right to exchange a typical meat sandwich for a sandwich irradiated to eliminate the potential risk of foodborne bacteria. Moreover, 68.3% of the participants were willing to pay at least some positive amount. The study also concluded that the value of reduced risk, as estimated by aggregated WTP, greatly exceeded the estimated direct costs of foodborne disease, and the estimated costs of irradiation for many food products.[13]

111. Shin *et al.* (1992) attempt to include the cost of pain and suffering associated with a foodborne disease through consumers' willingness-to-pay to avoid it. They estimated that, for each meal that may be potentially contaminated, participants in this study would be willing to pay USD 0.55 to eliminate *Salmonella* and USD 0.81 cents to eliminate *Trichinella spiralis*. Note that the risk of trichinosis in the United States is very rare, while salmonellosis is one of the most common causes of foodborne illness (Mead *et al.*, 1999). What was measured in this study was consumers' perceptions of risk, not their willingness to pay for actual foodborne illness risks.

IV. ADDITIONAL ECONOMIC CONSIDERATIONS OF FOODBORNE DISEASE

112. Sections III and IV considered the measurement of medical costs and productivity losses associated with foodborne disease, using the cost-of-illness and willingness-to-pay approaches. This Section examines some of the additional economic considerations of foodborne disease, incurred by industry (beyond productivity losses) or, in some cases, governments. While comprehensive information is

---

[13] The authors found a positive relationship between WTP and the perceived risk of foodborne disease, and a negative relationship between WTP and years of education.

not available, some anecdotal evidence and several empirical studies are presented to illustrate the additional economic costs associated with disease eradication, litigation costs, product recalls, market impacts and the value of firms.

113. It is always difficult to identify and measure these additional costs, although the task is made somewhat easier if the event is restricted to a single corporate entity (and information is made public). When an *E. Coli* outbreak in 1993 was traced to the US hamburger retail chain, Jack-in-the-Box, the immediate economic impacts of the outbreak included suspended hamburger sales, recalled meat from distributors, and increased cooking times/temperatures. Experts were called in to design an entirely new food-handling system. Losses of between USD 20 and USD 30 million were projected for 1993, although the quick response resulted in substantially lower actual losses (Goff, 1999).

114. A recent FAO report, based on country surveys and an extensive literature review, provides some indication of the additional economic costs to industry of recent foodborne disease outbreaks in selected countries (FAO, 2002). As the report examines animal diseases and, therefore, does not relate solely to foodborne disease, the figures provide some indication of the magnitudes involved. The cost estimates are broken down into direct costs (costs associated with compensation and control) and indirect costs (costs to agriculture and related industries). The results are summarised in Table 6 below:

Table 6. Costs of Recent Animal Disease Outbreaks (USD million)

Disease	Direct Costs	Indirect Costs	Total
BSE UK (1996-97)	2 433	1 395	3 828
CSF Netherlands (1997-8)	1 321	1 019	2 340
FMD UK (2001)	3 558	5 646	9 204
FMD Korea (2000)	266	n.a.	266
FMD Japan (2000)	15	n.a.	15

Source: FAO, 2002; for Korea, Ministry of Agriculture and Foresty of Korea.

115. Such comprehensive reports are rare. Most of the quantitative work in this area has been of an *ad-hoc* nature, focusing on a specific outbreak and/or specific regulatory measures introduced to deal with an incidence of foodborne disease. While not a comprehensive review, this Section presents a number of studies to illustrate the major areas of additional industry costs and the potential magnitude of such costs.

The Cost of Eradication

116. In some cases the risks associated with an outbreak of a disease are considered to be so severe that measures are implemented to ensure its complete elimination from the food system at source. This type of procedure is implemented when a single source for the pathogen can be isolated. Usually this means that the source can be traced to the primary production stage and a slaughter policy implemented to remove the source. We consider two examples of this type of policy, the BSE and salmonella control programs that are implemented in the UK.

117. In response to the Bovine Spongiform Encephalopathy (BSE) outbreak in the UK, controls for both live animals and meat were introduced and strengthened from the late 1980s to limit the exposure of humans to infected meat and of animals to infected feed.[14] At the height of the BSE crisis, in 1996, the ban

---

[14] Apart from the requirement that suspected cases of BSE and the offspring of confirmed cases be notified and destroyed, the two principal controls to keep infected material out of the food chain were the ‘Over Thirty Month’ (OTM Rule) and the removal of parts of the body that carry the highest demonstrable levels

on the use of cattle over 30 months old in food, pharmaceutical and cosmetic products cost the British Government GBP 480 (USD 749) per animal in compensation (of which the EU paid 70%). In addition, a special subsidy was paid to renderers of GBP 1.5 million (USD 2.3 million) per week and GBP 100 (USD 156) per veal calf was given in compensation to encourage their slaughtering. The Government also announced a GBP 50 million (USD 78 million) programme to support beef prices. The total cost of the British measures was approximately GBP 600 million (USD 936 million) in 1996 (Dnes, 1996). Since 1996, the estimated total annual direct costs of the BSE controls were of the order of GBP 550 million (USD 858 million). The breakdown between industry and government costs of the three methods of control is given in the following Table 7.

Table 7. Annual Costs of BSE in the UK
GBP (USD) million

	OTM Rule	Feed Ban	SRM	Total
Industry	95 (152)	14 (22)	18 (29)	127 (204)
Government	400 (642)	2 (3)	23 (37)	425 (682)
Total	495 (795)	16 (26)	41 (66)	552 (886)

Source: UK Food Standards Agency (2000b).

118. *Salmonella enteritidis* first emerged as a frequent infection of poultry in Great Britain in 1987 (O'Brien, 1988), although by the beginning of 1993 the UK Government had already spent GBP 5.5 million (USD 8.3 million) on the slaughter of 287 layer flocks and 77 breeder flocks (Veterinary Record, 1993).[15] At the same time, there was a dramatic increase in the prevalence of *Salmonella* food poisoning in humans in Great Britain. This was largely due to an increase in Salmonella Enteritidis PT 4 infection. Extensive media coverage led to a sharp drop in egg consumption of around 90% (North and Gorman, 1990). The incidence reached a peak in the early 1990s when, to restore public confidence, the Government introduced a comprehensive package of control measures. The cost to the Government and egg producers of increased surveillance and regulatory measures, sales losses and the slaughter of over a million birds was estimated at GBP 70 million (USD 125 million). The incidence of human salmonelosis remained broadly stable until 1998 when a significant fall was recorded throughout most of the UK. In 1998, 23 361 laboratory confirmed cases of salmonellosis were reported in the UK (MAFF, 2000). Since 1998, the level of laboratory confirmed cases has fallen further, with the lowest level since the late 1980s of 16 983 in 2000 (DEFRA, 2002), although there was a slight increase in 2001, to 18 420. (Source: DEFRA 2003)

Litigation Costs

119. Food firms, such as manufacturers, retailers and restaurants, have economic incentives to produce safer food in order to avoid foodborne illness lawsuits and the potential compensation that they may have to pay to those affected and their families. Product liability can be a powerful mechanism for shifting the costs of foodborne illness from the persons who become ill to the firms responsible for the contaminated product. However, high transaction and information costs combined with the structure of the legal system limit the effectiveness of the litigation for compensating consumers and providing firms with signals to

---

of infection (Specified Risk Material (SRM)). The principal control to prevent infectivity re-entering cattle and to reduce the incidence of BSE was a ban on feeding mammalian meat-and-bonemeal (MBM) to any farmed livestock. (FSA, 2000).

[15] Incidents due to S. enteritidis in chickens rose from 36 in 1986 to 111 in 1987 and 401 in 1988 (HMSO, 1989).

produce safer food (Buzby, Frenzen and Rasco, 2001). Although Clark (2000) suggests that as we are increasingly able to identify the source of a foodborne illness, the power of litigation to shape industry behaviour about food safety will increase.

120. Internationally, filing of a lawsuit is becoming an immediate response to outbreaks of foodborne illness with negotiations leading to multi-million dollar settlements (Morton, 1998). Buzby, Frenzen and Rasco (2001) found that, of a sample of 175 foodborne illness lawsuits resolved in court during 1988-1997, 31.4 percent resulted in some compensation paid by firms. The median award by juries for injuries due to pathogen-contaminated food products was USD 25 560 (1998 dollars). Buzby, Frenzen and Rasco also classified the defendants in foodborne illness court cases by firm type, during the period 1988 to 1997, the results are shown in Table 8.

Table 8. Defendants in Foodborne Illness Court Cases by Firm Type

Defendant	**Total Defendants**[a]	
	Number	**Per cent**
Restaurants	74	31.6
Foodstores	27	11.5
Distributors	11	4.7
Manufacturer[b]	29	12.4
Parent	60	25.6
Other	33	14.1
Total	234	100.0

Source: Buzby, Frenzen and Rasco, 2001.

[a] Of the 178 court cases, 43 had multiple defendants for an overall total of 234 defendants.

[b] Includes producers such as dairies and egg farms.

121. Some observers believe that nearly all food firms have at least some insurance coverage against foodborne illness due to a firm’s products (Clark, 2000). Comprehensive information about product liability insurance coverage in the food industry is not readily accessible because the insurance industry is highly competitive and data about premiums and paid claims are valuable market information. One example of the insurance available to food firms is the ‘products contamination coverage’ sold by the insurance subsidiary of the National Food Processors Association. This coverage includes assistance to deal with regulatory investigations and media inquiries, as well as product testing and compensation for the costs of product recalls, lost profits, and damage to brand names. Many food firms might obtain less comprehensive coverage. (Buzby, Frenzen and Rasco, 2001)

Product Recall and Market Impact

122. Product recalls and lost market sales are important aspects of the costs to industry associated with the outbreak of a foodborne disease. Food recalls and withdrawals are also expensive undertakings for businesses with loss of stock, loss of consumer confidence and potentially bad publicity. There are also costs associated with re-establishing goodwill and market share. This is true not only for the company involved but may also have a flow-on effect on whole industry sectors and to a country’s international reputation as a supplier of safe food. Even if a business is not responsible for a food poisoning incident, it can expect a downturn in sales as consumer confidence in a particular sector is affected. This can result in significant loss of sales in both domestic and international markets.

123. Wong *et al.* (2000) give an indication of the economic significance of product recalls as a consequence of the presence of pathogens which would lead to the US Food and Drug Administration (FDA) initiating legal action if the recall did not take place. The FDA was the entity most often responsible for detecting microbial contamination of foods and cosmetics (33% of all such recalls), followed by state regulatory agencies (24%), and manufacturers/retailers (21%). Table 9 indicates the number of products recalled classified by type of food product and pathogen respectively:

Table 9. Recalls of Food Products in the US 1994-1998 by Food Type and Microbial Organisms Isolated from Recalled Food Products

Product type	Number of product recalls	Microbe or Toxin	Number of product recalls
Dairy	22	*Bacillus cerreus*	3
Seafood	12	*Clostridium bottulinum*	38
Pastry	11	Coliforms	36
Salad	9	*E. coli* O157:H7	16
Sandwich	9	*Lactobacillus*	12
Dip/Sauce	8	*Listeria monocytogenes*	813
Beverage	7	*Salmonella*	143
Vegetable	6	*Staphylococcus aureus*	69
Grain	6	Other bacteria	8
Condiment	3	Hepatititus A	33
Fruit	2	Norwalk-like viruses	61
		Mold/yeast/fungi	96
Total	95	Total	1 328

Source: Wong *et al.*, 2000.

124. Contamination of minced beef with enterohaemorrhagic E. coli sold by Hudson Foods, Inc. in the US in 1997 resulted in the recall of 12 million kilograms of product. In this case, an initial small product recall multiplied significantly when it was found that product reworking practices in the plant left no break in the potential chain of contamination from early June through to August (USDA).

125. An outbreak of *Listeriosis* in the US, in August 1998, was responsible for more than 70 cases of illness and 16 deaths in 14 states. Investigators believe that construction dust laced with *listeria* contaminated meats being processed at the Bil Mar Foods plant in Michigan. Bil Mar Foods voluntarily recalled an estimated 35 million pounds of specific production lots of hot dogs and deli meat that might have been contaminated. This led to a number of further scares associated with meat and other products. The scale of the recall was immense with the Sara Lee corporation alone recalling an estimated 16 million kg of meat. Another large product recall in 1999, due to possible contamination by *Listeriosis*, involved 30 million pounds of meat and poultry products, produced by Thorn Apple Valley at an expected cost of between USD 1 and USD 7 million. (Cornell Cooperative Extension 1999)

126. In Australia a 1997 *salmonella* incident involving mettwurst (pork sausage) resulted in closure of the business, cost the bakery owners AUD 16 000 (USD 11 869) in fines and led to an insurance settlement of AUD 750 000 (USD 556 380)). Following this incident, mettwurst sales throughout Australia fell by 40%. There have been reports of 400 to 500 small goods producers going out of business as a result. It is estimated that the downturn in trade following this incident cost the Australian small goods industry over AUD 400 million (USD 297 million). Similarly, a 1996 *salmonella* contamination of peanut butter cost the company involved over AUD 55 million (USD 43) and there was a flow-on effect to another company

which lost AUD 100 000 (USD) in sales of its peanut butter muesli bars. (Food standards agency Australia and New Zealand, 2002)

127. An outbreak and subsequent recall of product associated with a New Zealand supermarket delicatessen counter cost the outlet around AUD 1.5 million (USD 0.8 million) in recall costs, consultants fees, reparation, product loss and lost custom. Over the next six months, the supermarket chain as a whole lost AUD 3 million (USD 1.6 million) in reduced sales. (Food Standards Agency Australia and New Zealand, 2002).

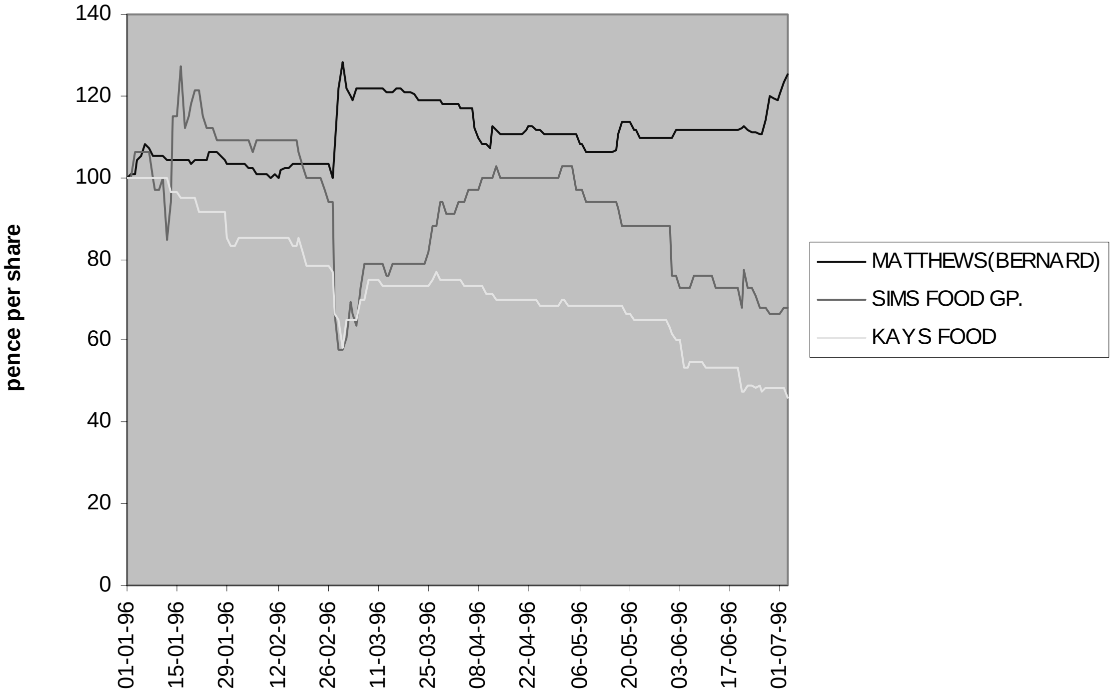
128. A major review of the market and regulatory impact of the BSE crisis in Europe by the GIRA consulting firm estimated that, by the end of 2001, 1.7 million tonnes of beef was removed from consumption, beef export volumes within the EU and to third countries dropped by one-third, and producer prices for finished cattle were 10-15% lower than before the crisis. (IMS, 2001). Caskie, Davis and Moss (1999) estimated net losses in income for Northern Ireland of 0.5% of regional GDP from the impact of BSE with job losses of up to 0.6% of regional employment. About 77% of the income losses and 87% of the job losses were in the beef sector, primarily beef production.

The Impact on the Value of Firms

129. An outbreak of foodborne disease (or related animal disease) will lower the expectations of investors regarding the profitability of firms producing the affected product. As a consequence the share price of such firms can be expected to decline and, if one accepts the efficient markets hypothesis (Fama, 1970), the decline in the market value of the firm could be taken to be an estimate of the expected costs to the firm of the incident.

130. Figure 1 shows the evolution of the share prices of three British food manufacturing companies during the first six months of 1996. All three companies show a substantial change in price coinciding with the announcement that new variant CJD was potentially attributable to the consumption of meat from animals infected with BSE. Bernard Matthews is primarily involved in the production of poultry and experienced an increase in their share price whilst the other two companies which have interests in beef manufacturing show a decline in price.

Figure 1. Share Price of Three British Food Manufacturing Companies (first 6 months 1996)



131. Thomsen and McKenzie conducted an event study[16] to evaluate the effects of federally supervised meat product recalls on the value of publicly owned meat and poultry companies in the US. They concluded that only the most serious class of product recall, where there is a probability that consuming the product will cause serious adverse health consequences or death, have an impact on shareholder value. It is found that this type of recall lowers the shareholder value of the company by between 1.5 and 3%.

132. Henson and Mazzocchi conduct an event study to determine the impact of the BSE crisis on the equity prices of 24 food manufacturing firms. The purpose of an event study is first to identify whether the impact of an event is significant and second to enable the comparison of changes in the levels of returns obtained in firms of different sizes as a consequence of the incident. Returns on the shares of dairy, animal feed and beef firms were found to be are negatively impacted by the BSE scare whilst those of other-meat manufacturers were positively affected. The size of the negative impact is greatest for beef manufacturers and lowest for dairy firms whilst the positive impact on the returns to other-meat producers is smaller in magnitude than the negative effects on both the beef and animal feed firms.

133. In the US Thorn Apple Valley *listeriosis* recall noted above, Sales fell by USD 16.6 million over the previous year. This resulted in estimated losses of USD 2.02 per share in its second quarter ended December 1999, including a charge of USD 8 million for international restructuring and a loss of USD 2.8 million on the disposal of the fresh pork division (Meat Industry Insights, 1999).

---

16. Event studies provide a method whereby the impacts of ‘food scare’ on the stock price of firms can be determined. The method first entails the estimation of the ‘normal’ returns on a security against which the actual returns can be compared in order to determine the ‘abnormal’ returns. When abnormal returns coincide with the food-scare event it is concluded that the event has a significant impact on the value of the firm(s).

V. SUMMARY OBSERVATION

134. This report provides an overview of the costs associated with foodborne disease. Attention is drawn to the economic importance of these costs, the wide range of methodological approaches and empirical results and the general lack of comprehensive and/or comparable quantitative information.

135. Sections II and III indicate the almost exclusive reliance on the COI approach to estimating the cost of foodborne disease. Whilst these estimates serve a useful purpose in raising awareness of the substantial costs associated with these diseases, they are of limited value in designing food safety policy because of the limitations in terms of their partiality that have been discussed. Initially what is required is a clear understanding of the nature of the market failure that exists in insuring consumers against foodborne disease risk. It is not sufficient to use zero incidence of the disease as a baseline in estimating the loss in welfare that arises from their incidence. It is probable that consumers (and taxpayers) are willing to pay for a reduction in the risk associated with the presence of foodborne pathogens but the resource cost that is associated with reducing the risk to the minimum is likely to be prohibitive. In short we have to consider what the socially optimal level of foodborne disease risk is. Second, estimates of the benefit that will be derived by consumers through correcting this market failure are required in order to evaluate the welfare gain which would ensue. The WTP approach, possibly implemented through choice experiments, may provide a better approach to obtaining such estimates as long as consumers are given information on the foodborne disease outcomes and probability of disease.

136. Section IV examined some of the additional economic considerations of foodborne disease for industry and governments. While the evidence reported is limited to a few anecdotal and very specific cases, it does show that the economic costs associated with disease eradication, litigation, product recalls, market impacts and share values can be very significant. Such additional economic costs should be taken into consideration when designing food safety regulations and, in particular, when carrying pout a full cost-benefit analysis of regulatory options. The methods that are reviewed in section IV are largely of an *ad-hoc* nature, they rely on an accounting approach which focuses on readily identifiable costs associated with a disease outbreak.

137. Whilst significant efforts have been made to connect the COI and WTP approaches to their theoretical economic underpinnings, the economic framework for studies of the costs to industry is less well developed. A notable exception to this is Antle (2000b). One can argue that the omission of an explicit framework which accounts for optimising behaviour and possible substitution amongst alternative products by the firm is likely to overestimate the costs of an incident. Intuitively a firm is likely to attempt to minimise the negative impacts of an outbreak by rearranging its product mix and other aspects of its business. A simple accounting approach may not accurately reflect the impacts of such changes on a business. For this reason the approach suggested by Antle is a significant step forward because, by estimating a cost function, some of these omissions can be rectified.

138. As noted in the earlier OECD study on the incidence of food borne disease prepared by the WHO, “in spite of some very successful efforts, the burden of foodborne disease remains high. FBD has been brought to the attention of consumers and policy-makers during the two last decades because of some highly publicised outbreaks caused by micro-organisms 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.”

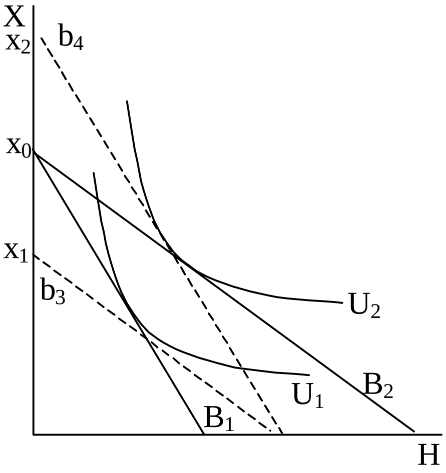
ANNEX I: MEASURING CHANGES IN CONSUMER WELFARE

This very simple model formally represents the benefit that is the result of an exogenous policy intervention and shows how it may be evaluated. The model serves to establish a framework in which the general approaches to measuring the costs of foodborne disease can be considered.

The choices facing a consumer when deciding how safe they wish to be in their choice of food consumption can be represented with an indifference curve diagram (Figure 1) - the X axis is the level of consumer consumption and H represents health status (*e.g.* risk of foodborne disease). Line B_1 represents the trade-off (budget constraint) between consumption and health, arising because the consumer incurs a cost in attaining a higher health status, for example in the higher price of a healthier diet and higher levels of medical expenditure.

The indifference curve U_1 joins points which yield the same level of satisfaction (utility) to the consumer. The further away from the origin that the indifference curve is located, the higher the level of satisfaction associated with the points along its length. The highest level of satisfaction that can be attained is that associated with U_1 and the consumer obtains this by choosing the health status and consumption level represented by the point of tangency between B_1 and U_1.

Annex Figure 1. The effects of improved food safety on welfare



The effects of a reduction in the level of foodborne disease can be represented by a reduction in the opportunity cost to the consumer of achieving an increase in their health status. Thus, B_1 moves to B_2 because the amount of consumption required to achieve a given increase in health status is reduced (*e.g.* the consumer spends less to avoid foodborne disease). As a consequence, the consumer obtains maximum satisfaction at the point of tangency between U_2 and B_2 and consumer welfare is increased.

In evaluating the effect of such a change, the object is to measure the increase in satisfaction obtained (willingness to pay) as the consumer moves from U_1 to U_2. There are two theoretical approaches to obtaining such a measure. One is to take money away from the consumer thereby such that consumer satisfaction returns to U_1 (*i.e.* moving from B_2 to b_3) with the cost measured as the difference between x_0 and x_1.

The second approach is to identify the amount of money we would have to give to the consumer for an equivalent increase in satisfaction (utility) to that which is obtained from the reduction in the level of foodborne disease (*i.e.* moving the budget constraint B_1 to b_4) with the cost measured as the difference between x_2 and x_0.

Note that these two approaches will not generally result in the same cost estimate.

REFERENCES

Antle, J.M. (2000a). “Economic Analysis of Food Safety” In: B. Gardner and G. Rausser (Eds.) *Handbook of Agricultural Economics,* Vol. 2, North Holland Publishing Co. Amsterdam.

Antle, J.M. (2000b). “No Such Thing as a Free Safe Lunch: The Cost of Food Safety Regulation in the Meat Industry,” *American Journal of Agricultural Economics* 82 310-322.

Australian Department of Health and Ageing, Food Safety Management Systems, Costs, Benefits and Alternatives, 2001, http://www.health.gov.au/pubhlth/strateg/foodpolicy/pdf/alternatives.htm

Berger, M.C., G.C. Blomquist, D. Kenkel, and G.S. Tolley (1987). “Valuing Changes in Health Risks: A Comparison of Alternative Measures,” *Southern Economic Journal* 53:967-84.

Brown, R. (2001). “BSE - A Painful and Continuing Saga,” International Meat Secretariat *News Letter,* No 261 - 15 October 2001. http://www.meat-ims.org/IMS_Pages/info_exch.htm

Buzby J.C., Allos B.M. and Roberts T. (1997). “The Economic Burden of *Campylerbacter*-Associated Guillain-Barre Syndrome,” *Journal of Infectious Diseases* 176(suppl. 2) 192-197.

Buzby, J. C., Frenzen, P.D. and Rasco, B. (2001). “Product Liability and Microbial Foodborne Illness,” Food and Rural Economics Division, Economic Research Service, US Department of Agriculture. *Agricultural Economic Report No*. 799.

Buzby J,C and T. Roberts (1997). “Economic Costs and Trade Impacts of Microbial Foodborne Illness,” *World Health Statistics Quarterly* 50 pp 57-66.

Buzby J.C., T. Roberts, C.T.J Lin. and J.M MacDonald. (1996). “Bacterial Food Borne Disease: Medical Costs and Productivity Losses,” *Agricultural Economic Report* 741, United States Department of Agriculture, Washington, DC.

Caswell J.A. (1998). “Valuing the Benefits and Costs of Improved Food Safety and Nutrition,” *Australian Journal of Agricultural and Resource Economics* 42(4) pp 409-424.

Caskie, P., Davis, J. and Moss, J. E. (1999). “The Economic Impact of BSE: a Regional Perspective,” *Applied Economics,* 31, 1623-1630.

Chalker, R.B. and Blaser, M.J. (1988). “A Review of Human Salmonellosis: III Magnitude of Salmonella Infection in the US,” *Review of Infectious Diseases.* 10,1 111-124.

Clark, B. T. Attorney at Marler Clark. Personal Correspondence in 2000 quoted in Buzby, Frenzen and Rasco (2001).

Cornell Cooperative Extension (1999). *Multistate Outbreak of Listeriosis*. Cornell University, US (January/February).

DEFRA (2003). *Zoonoses Report: United Kingdom 2001*. Department for Environment, Food and Rural Affairs, London.

Dnes, A. W. (1996). “An Economic Analysis of the BSE Scare,” *Scottish Journal of Political Economy*, 43, 343-348.

Fama E.F. “Efficient Capital Markets: A Review of Theory and Empirical Work,” *Journal of Finance* 25 383-343.

FAO (2002) *Animal Diseases: Implications for International Meat Trade* Committee on Commodity Problems, Intergovernmental Group on Meat and Dairy Products, 19th Session, Rome 27-29 August 2002.

FAO/WHO (2002). Statistical Information on Foodborne Disease in Europe - Microbiological and Chemical Hazards, Conference paper, FAO/WHO Pan-European Conference on Food Safety and Quality, Budapest, Hungary, 25-28 February 2002. http://www.foodsafetyforum.org/paneuropean/index_en.htm

FSA (2000) *A Report on the Study of Infectious Intestinal Disease in England*, London: The Stationary Office.

FSA (2000b). *Review of BSE Controls*. Food Standards Agency, London.

Foegeding, P.M. and T. Roberts, co-chairs, Council for Agricultural Science and Technology Task Force. 1994. *Foodborne Pathogens: Risks and Consequences.* 87 pages. Ames IA: Council for Agricultural Science and Technology.

Food Science Australia and Minter Ellison Consulting. *National Risk Validation Project: Final Report* http://www.health.gov.au/pubhlth/strateg/foodpolicy/pdf/validation.htm.

Food Standards Australia and New Zealand (2002) *Incidence of Foodborne Illness* Internet website: http://www.foodstandards.gov.au/mediareleasespublications/publications/foodsafetystandardscostsandbenefits/incidenceoffoodborne633.cfm

Guimalva J.N., Bailey W.C. and Redfern M. “An Experimental Study into Consumers’ Willingness-to-Pay For an Irradiated Meat Product,” *Journal of Food Safety* 17(3) 193-202.

Goff, R. (1999). “Coming Clean,” *Forbes*, 163 (10), 156-158.

Henson S. and M. Mazzocchi. “Impact of Bovine Spongiform Encephalopathy on Agribusiness in the UK: Results of an Event Study of Equity Prices”, *American Journal of Agricultural Economics* 84(2) 370-386.

HMSO (1989). *The Report of the Chief Veterinary Officer: Animal Health* 1988. Her Majesty’s Stationary Office, London.

Krug, W. and Rehm, N. (1983). Nutzen-Kosten-analyse de salmonellose-bekampfung. Schriftenreihe des Bundesministers fur Jugens, Famile und Gesundheit, Berlin.

Lindqvist R., Andersson, Y., Lindback, J., Wegscheider, M., Eriksson, Y., Tidestrom, L., Lagerqvist-Widh, A., Hedlund, K.O., Lofdahl, S., Svensson, L. and Norinder, A., “A One Year Study of Foodborne Illnesses in the Municipality of Uppsala, Sweden,” *Emerging Infectious Diseases* 7(3) pp588-592.

Mead, Paul S., Laurence Slutsker, Vance Dietz, Linda F. McCaig, Joseph S. Bresee, Craig Shapiro, Patricia M. Griffin, and Robert V. Tauxe. 1999 "Food-Related Illness and Death in the United States." *Emerging Infectious Diseases* 5(5):607-625, accessed 10/8/99 <http://www.cdc.gov/ncidod/eid/vol5no5/mead.htm>.

Morton, E. (ed.) (1998). "Lawyers Feed on Outbreaks", *Food Protection Report*, 14, 1-2.

North, R. and Gorman, T. (1990). "Chickengate: An Independent Analysis of the Salmonella in Eggs Scare," *Health Unit Paper* No. 10, IEA Health and Welfare Unit, The Institute of Economic Affairs, London.

O'Brien, J. D. P. (1988). *Salmonella Enteritidis Infection in Broiler Chickens*, Veterinary Record, 122-214.

OECD/WHO (2002). *Food Safety Compliance: A Report on Foodborne Diseases in the OECD Area.*

Peltola, J., Aakkula J., Maijala R., and Siikmaki J. *The Value of the Finnish Salmonella Control Program in Consumer's Eyes,* Unpublished paper: Agrifood Research Finland, P.O. Box 3, 0004111 Helsinki, Finland.

Roberts J.A. and Sockett P.N. (1994). "The Socio-economic Impact of *Salmonella Enteritidus* Infection," *International Journal of Food Microbiology* 21:117-129.

Scott W.G., Scott H.M., Lake R.J. and Baker M.G. (2000). "Economic Cost to New Zealand of Foodborne Infectious Disease," *The New Zealand Medical Journal* 113 pp281-4.

Shin S-Y, Kliebenstein, J.K., Hayes D.J. and Shogren J.F. (1992). "Consumer Willingness to Pay for Safer Food Products," *Journal of Food Safety* 13:51-59.

Sockett, P.N., (1995). "The Epidemiology and Costs of Diseases of Public Health Significance, In Relation to Meat and Meat Products," *Journal of Food Safety* 15 91-112.

Thomsen M.R. and A. M. McKenzie (2001). "Market Incentives for Safe Foods," *American Journal of Agricultural Economics* 82(3) 526-538.

Todd E.C.D (1989a). "Preliminary Estimates of Costs of Foodborne Disease in Canada and Costs to Reduce Salmonellosis," *Journal of Food Protection* 52 (8) pp586-594.

Todd E.C.D. (1989b). "Costs of Acute Bacterial Foodborne Disease in Canada and the United States," *International Journal of Food Microbiology* 9 pp313-326.

USDA http://www.fsis.usda.gov/oa/recalls/prelease/pr015-97.htm.

Veterinary Record (1993). "Government to End Slaughter of Salmonella-Infected Layers", *Veterinary Record*, 132-174.

Wong S., D Street, S.I. Delgado and K.C. Klontz (2000). *Recalls of Foods and Cosmetics due to Microbial Contamination Reported to the US Food and Drug Administration* Journal of Food Protection 63(8) pp1113-1116.

http://www.ers.usda.gov/Emphases/SafeFood/

http://www.ers.usda.gov/briefing/FoodborneDisease/

http://www.ers.usda.gov/briefing/FoodSafetyPolicy/