The Surveillance and Control of Foodborne Diseases

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The Surveillance And Control Of Foodborne Diseases

Donal J. Daly
THE SURVEILLANCE AND CONTROL OF FOODBORNE DISEASES

DONAL DALY

MAY 1995

A Thesis submitted for the Degree of Master of Science (Environmental) to the National Council for Educational Awards.
This Thesis is dedicated to Paddy O'Connor, former Principal Environmental Health Officer with the Southern Health Board (Kerry), whose retirement after 44 years service, coincides with the completion of this work.
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ABSTRACT

THE SURVEILLANCE AND CONTROL OF FOODBORNE
DISEASES

DONAL DALY

There is great variation in the extent of surveillance of foodborne disease throughout the world. Some countries contribute important epidemiological and microbiological data to the WHO Surveillance Programme for Foodborne Diseases in Europe. While the UK is a major contributor to this programme, data from Eire has been non-existent. Trends in the incidence of foodborne disease in the UK may have particular relevance to Eire given the similarity of culture. Microbiological and epidemiological data in relation to foodborne pathogens, foods most frequently incriminated in outbreaks, places where outbreaks occur, together with the main contributory factors are discussed. The broader area of the surveillance of gastro-intestinal infectious diseases is discussed in this context, as the two most frequently isolated enteropathogens in the UK, Campylobacter and Salmonella sps. are transmitted mainly through the food chain.

Formal surveillance of gastro-intestinal infections is relatively new in EIRE. Emerging trends from the Dublin and Cork Units are discussed and compared with trends in the UK. Trends in relation to statutory notifications of food poisoning are further considered.

Finally, strategies for the control of foodborne diseases are examined. In this, it is established that the elimination of pathogens, particularly Salmonella sps. at primary breeding level in poultry is the most effective way of reducing infection in humans. However, other strategies and approaches are also considered. These include food irradiation, the concept of Hazard Analysis Critical Control Point, the role of hygiene education, and the case for establishing a formal surveillance programme for foodborne disease in Eire. The kitchen as the final line of defence is given extensive discussion. The importance of temperature control in the food service and retail grocery trade in particular is further considered. The potential for water as a source of infection and intoxication is also highlighted. Final conclusions and recommendations are outlined.
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The surveillance of disease has been defined as "the continual watchfulness over the distribution and trends of incidence through the systematic collection, consolidation and evaluation of morbidity and mortality reports and other relevant data" (Langmuir, 1963). This definition can also be applied to the surveillance of foodborne disease, the latter being further defined as "a disease of an infectious or toxic nature caused or thought to be caused by the consumption of food or water" (WHO, 1992).

Surveillance is the key to the identification of trends in the incidence of foodborne disease and has two main objectives.

1. The early detection of outbreaks to enable rapid investigation and control.

2. The monitoring of trends over a period of time of outbreaks and sporadic cases, to assess the need for prevention, and evaluate preventive measures.

Surveillance data on foodborne disease varies greatly from country to country. Some countries, like Scotland, have developed a very good system of surveillance, with others, including Ireland, to date, failing to produce reliable epidemiological and microbiological data on the occurrence of foodborne disease. This lack of appropriate surveillance data on gastrointestinal tract infectious diseases generally at national level, formed the inspiration for this thesis. It was also felt that research into this area including that from an Irish perspective would be of value.
The foregoing paragraphs summarise the background to this thesis, and the basis on which it is approached. As an Environmental Health Officer, having responsibility for monitoring hygiene standards in food premises and matters relating to foodborne disease, the writer was aware of the potential health risks associated with the manufacture/preparation of food, particularly on premises where staff are not sufficiently aware of the basic principles of food hygiene. Incidents of food poisoning frequently occur because of a failure to understand these principles, many of which are a matter of basic common sense.

Statistics on the incidence of foodborne disease in this country could not possibly represent the true picture in relation to morbidity in this area. This lead to an examination of the whole area of surveillance, including a study of identifiable trends in different countries relating to the main causative agents of food poisoning, the foods most often implicated in outbreaks, the places where food poisoning was acquired, and the main contributory factors recorded during investigations. Because of the similarity in culture, climate, eating patterns, etc. in both the UK and Ireland, data obtained from all three surveillance centres within the UK was found to be of particular interest and, consequently, will be the subject of greatest analysis and discussion. The surveillance of gastrointestinal tract infections (including foodborne infectious disease and intoxications) is highly developed in the UK in comparison to many other countries, with Scotland for example being the first country to formally contribute the WHO European Surveillance Programme for foodborne disease.

While this thesis deals primarily with the surveillance and control of foodborne disease, research will show that the broader surveillance of gastrointestinal tract diseases generally is very applicable to this theme.
This is because the two most frequently isolated pathogens reported within the three UK regions, *Campylobacter* and *Salmonella sps.* are mainly thought to be transmitted through the food chain. Consequently, the incidence of 'gastroenteritis' caused by these two enteropathogens, based on confirmed laboratory isolates, will be the subject of particular discussion. It will also be necessary to examine emerging foodborne pathogens, some of which have already gained a certain degree of media coverage. The relative importance of these microorganisms in the overall context of the surveillance of foodborne disease will be analysed.

Food poisoning is a statutory notifiable disease, unlike the reporting of gastrointestinal tract infections which is based on a separate voluntary system. It will be shown how the notifications of food poisoning is haphazard and underrepresentative. The contrast between the reported incidence of food poisoning in Ireland and the UK will be seen to be particularly significant, and the reliability of data in this area will be questioned.

Formal surveillance of gastrointestinal disease is relatively new in this country. In the absence of a national disease surveillance centre, the two Irish regional units, based in Cork and Dublin, will form the basis for discussion in this area. Data from these units will be analysed against the background of the more established surveillance of enteropathogens in England and Wales, Scotland and Northern Ireland. While these UK surveillance centres have been actively engaged in the collection, collation and reporting of epidemiological and microbiological data on gastrointestinal disease for many years, data that has already emerged from the two Irish units will be discussed and compared in this context.
The epidemic of gastrointestinal disease in the UK caused by *Salmonella enteritidis* from about 1986 onwards will be the subject of much discussion. This is principally because it will link up the surveillance of gastrointestinal disease generally with infection of the food chain, and will demonstrate how the two parallel systems of surveillance (i.e. of ‘gastroenteritis’ and foodborne disease specifically) cannot be discussed in total isolation. Discussion on the emergence of *S. enteritidis* will confirm the need for active surveillance, particularly as in the latter case, the epidemic became associated with a new food source. Further, it will be seen how one phagetype in particular predominated during the whole epidemic, and the significance this had from a public health perspective will therefore be analysed.

The final section of the thesis will deal with the control of foodborne disease. This discussion will be based on data presented in the first two parts of the thesis, which will highlight the most up-to-date epidemiological data in the occurrence of foodborne disease. As poultry will be shown to be a major vehicle of infection of the predominant foodborne pathogens, the discussion will examine, in particular, the poultry production industry, emphasising the necessity for the prevention and control of potentially dangerous pathogens at primary level. Research into this area will be examined.

Measures for the control of foodborne diseases can be approached at different levels along the food chain. While elimination of pathogens at primary level is the ideal, this is very difficult to achieve and other measures need to be applied. It is not the intention of the writer to analyse every conceivable measure of control. However, an overview of the whole approach to control will be given.
This overview will include discussion on such diverse topics as food irradiation, the Hazard Analysis Critical Control Point (HACCP) approach to food safety, the necessity for teaching and promoting hygiene education at various levels, and the importance of establishing a formal system for the surveillance of foodborne diseases.

This thesis is essentially a discussion and analysis of:

(a) The surveillance of foodborne disease and the gastrointestinal tract pathogens that are primarily transmitted through the food chain.

(b) The various approaches to food safety and, consequently, to the prevention/reduction of foodborne disease.

1.1 Summary of Principal Objectives

(1) To analyse trends in the incidence of foodborne disease in relation to:

   (a) The main causative agents and their relative significance.
   (b) The emergence of new pathogens.
   (c) The sources/vehicles of infection of these agents.
   (d) The places where outbreaks of food poisoning occur.
   (e) The main contributory factors in outbreaks.

(2) To analyse and discuss surveillance data on gastrointestinal infectious diseases, with particular reference to infections transmitted through the food chain.

(3) To analyse and discuss similar data from the two Irish regional surveillance units, and to compare these with the UK data.
To analyse and discuss various approaches to the control of foodborne disease, and to highlight areas where control can be most effectively exercised.
The Microbiology and Epidemiology of Foodborne Disease

2.1 Introduction

The surveillance of foodborne infectious diseases is concerned with the collection and collation of epidemiological and microbiological data related to such diseases, with particular emphasis on the main aetiological agents, their relative significance, and the emergence of 'new' pathogens. However, any discussion in this area should not focus solely on the causative agents, but must also include an examination of the location in which foodborne infections are acquired, the foods most often implicated, and, finally, the factors that are most likely to contribute to these incidents.

At the outset, it is important to recognise that the incidence of most communicable diseases, including those that are foodborne, are greatly under-reported. Consequently, the statistics as they exist, relate more to trends and not to the true incidence of these infections and intoxications.

The extent of surveillance of foodborne diseases is dependent on a number of factors, including:-

1. The seriousness with which a Government views foodborne and other gastro-intestinal diseases.
2. The manpower allocated in terms of laboratory staff, investigation officers, surveillance co-ordinators etc.

3. The existence of a regional or national surveillance unit with the relevant computer database for the collation, analysis and reporting of epidemiological and microbiological information on foodborne disease.

4. The motivation of general practitioners and attending hospital doctors to report cases of food poisoning when they come to their attention, and the speed with which such notifications are made to the local Director of Community Care.

5. The extent to which hospital bacteriology laboratories have kept pace with modern methods of isolation and detection of enteropathogens generally.

Surveillance is the basis for the accumulation, analysis and interpretation of statistical and epidemiological data. Recognition of emerging trends is entirely dependent on the extent of surveillance in any country. This is an important point to keep in mind in the context of this thesis. Surveillance systems will be covered more thoroughly in a separate chapter.

When comparing trends in the incidence of specific foodborne infectious diseases in various countries, certain factors should be kept in mind that may influence the outcome of such comparisons. These may, in fact, reflect differences in culture etc., rather than differences in the actual incidence of disease. Examples of these may include:-

1. The extent of surveillance of foodborne diseases.
2. The type of foods eaten - for example, a propensity for eating poultry, rice, fish and salads in different cultures.

3. Climate:- which has a strong influence, not only on the types of foods eaten, but also on the importance of temperature control with respect to food preparation and storage.

4. The particular eating patterns in different countries: - including the main places where foods are eaten (homes, schools, canteens, restaurants, etc.), the preference for specific methods of cooking etc.

In recent years, in many western countries, we have come to depend more and more on convenience foods, including 'pre-heated' and 'take-away' meals. This change in eating patterns brings with it new possibilities for foodborne disease.

5. Public awareness in food hygiene, its importance etc., including the degree of training received by foodworkers in personal and operational hygiene etc.

In Ireland, statistical and epidemiological data in relation to foodborne disease is practically non-existent. This situation is very different from that of our near neighbours in the UK, particularly Scotland, where data on outbreaks have been formally collated and reported since 1980. Indeed, Scotland was the first country to formally contribute to the WHO Surveillance Programme for foodborne infectious diseases in Europe (Sharp et al., 1988), and data initially collated and contributed by the Scottish Communicable Disease Surveillance Unit was used as a model for all other European countries which subsequently contributed to the programme. From a review of the WHO report (WHO, 1992), it can be seen that Ireland has not been contributing to the WHO programme.
For this reason, it is necessary to depend on statistical and epidemiological data from other countries to get an insight into emerging trends in the area of foodborne disease. However, as will be seen, data emerging from different countries can vary greatly, and it is not possible to draw conclusions that can automatically be applicable to the Irish situation.

2.2 Definition of Food Poisoning

When discussing trends in the incidence of foodborne infections and intoxications, it is important, at the outset, to define exactly what is meant by foodborne disease ('food poisoning').

Some countries consider a disease as foodborne only when an aetiological agent is identified in the patient as well as the food. Others define all cases of diarrhoeal disease to be foodborne in origin, although some of the recognised foodborne pathogens, such as *Salmonella sps.* may be transmitted via a food vehicle or from person to person through the faecal-oral route. Therefore, precise comparison of national figures is not always possible and, consequently, wide variations in morbidity may be explained by the different manner of reporting and categorising infections. However, notwithstanding these shortcomings, an analysis of available statistical and epidemiological data in this area will still be of benefit.

The problem of the absence of a common definition for food poisoning within the UK, for example, was the subject of an advisory paper by the Committee on the Microbiological Safety of Food (Anon(A), 1992). This paper was necessary to assist uniformity of notifying practices by medical practitioners throughout the UK, since, as is the case in Ireland, this term is nowhere defined in legislation.
The Committee in its report proposed the following definition which is that currently used by WHO, and which has been adopted for administrative purposes since 1980 in Scotland (Collier et al., 1981).

"Any disease of an infectious or toxic nature caused, or thought to be caused by the consumption of food or water"

A closer look at this definition will perhaps clarify some matters, viz:

- The definition includes 'disease' in the broader sense, regardless of presenting symptoms. So other non-typical gastrointestinal manifestations of illness (e.g. neurological symptoms) are included, which is desirable if the full burden of food and waterborne disease is to be apparent from further statistical analysis.

- Suspicion of disease/illness caused by the consumption of food and water is sufficient for inclusion. This is important for surveillance and investigative purposes, as proving that a suspect food is responsible for a food poisoning incident is usually difficult. Very often, one has to depend on epidemiological evidence rather than microbiological confirmation (serotyping, phagotyping etc.) to conclude that a particular food has caused illness.
Illness caused by toxic chemicals, whether biological or man-made is included. While most food poisoning illnesses are caused by microorganisms (particularly bacteria, but occasionally viruses) rather than chemicals, it is worth remembering that two of the most widespread outbreaks of food poisoning ever to occur in Europe and Ireland were caused by chemical contamination of food, the former resulting from the contamination of cooking oil with pesticide in Spain in 1981, and the latter caused by the contamination of cucumbers by another pesticide (‘Aldicarb’) in Eire in the summer of 1992.

- Allergens and food intolerance are not included in the definition as these refer specifically to a person's individual response to a food (e.g. shellfish) rather than the quality of the food itself.

- Water is included in the definition and an illness is regarded as being 'foodborne' even though the vehicle of infection may have been water. There may be a conflict here in Ireland with the inclusion of water in the definition of food poisoning. The Health Act of 1947 defines food as:–

"Every article used for food or drink by man, other than drugs or water"

Therefore, while water is included under the WHO definition of foodborne illness (food poisoning), albeit for administrative purposes, it is excluded from the definition of food in Irish Law. It is, therefore, possible that many infectious diseases would not be included for statistical purposes as being foodborne (‘food poisoning’) if the WHO definition of the latter is not clearly understood and adopted by relevant authorities, such as the Department of Health and the medical profession generally.
While it could be argued that waterborne infectious diseases would be notified anyway as 'gastro-enteritis' (i.e. medical condition rather than mode/vehicle of transmission), the latter being a notifiable disease under the Infectious Disease Regulations 1981, it is important to understand that the 'umbrella' term 'gastro-enteritis' is only notifiable if contracted by children under 2 years of age. Other notifiable infectious (and potentially waterborne) diseases, such as salmonellosis, bacillary dysentery, cholera, typhoid and paratyphoid, are notifiable without reference to age.

While the above comments could be regarded as being of little significance given that 'gastro-enteritis' and 'food poisoning' are greatly under-reported anyway, the definition of food poisoning (to include water, given that water is the basic ingredient in most food processes) needs to be clarified in this country, as has been the case in the UK.

2.3 Surveillance Programmes for Foodborne Diseases in Europe and North America

There are three surveillance programmes for the collation of epidemiological and statistical data in relation to foodborne illness (outbreaks) in Europe and North America.

**EUROPE:** WHO surveillance programme for the control of foodborne infections and intoxications (Berlin).

**CANADA:** Foodborne disease reporting centre, Food Directorate, in collaboration with the Bureau of Chemical Hazards, Environmental Health Directorate (Ottawa).
UNITED STATES: Centre for Infectious Diseases, Enteric Disease Branch, Centre for Disease Control (Atlanta).

Having reviewed the most up-to-date available data from these surveillance programmes, the following broad statements can be made:-

1. In all countries, it is accepted that the majority of food poisoning incidents are not reported or recorded.

2. For the vast majority of recorded food poisoning outbreaks, the causative agent or the food vehicle is not proven.

3. Where the causative agent is identified, the vast majority are of microbiological (as opposed to chemical) origin - mostly bacteria.

4. *Salmonella sps.* are the most frequently recorded causative agent where a microorganism has been identified.

5. Meat and meat products account for the vast majority of outbreaks where a food vehicle has been identified.

6. The place where most food poisoning outbreaks are recorded can vary from country to country with, for example, the home being the most frequently recorded location in the UK and Austria; catering or food service establishments in the US; schools in France and canteens in E. Germany.
7. The epidemiological data contributed to these programmes can vary greatly between countries. Within the European surveillance programme, (5th report) the UK, France, Spain (unlike Ireland) and others are significant contributors. In North America, the Canadian surveillance unit provides complete epidemiological data. In fact the Canadian and the UK communicable disease surveillance systems are arguably the best in the world.

Point no. 2 above in relation to the failure to record a pathogen or a food vehicle in so many outbreaks is very relevant, as it puts the degree of success of a surveillance programme into context. It further shows the importance of both the public and the medical profession bringing incidents of food poisoning to the attention of public health authorities at the earliest possible moment, as speed of investigation is vital if full microbiological and epidemiological data is to be ascertained and collated. Even where full epidemiological information is available, there still remains the difficulty of confirming a microbiological agent. Speed of investigation increases the opportunity of collecting 'left-over' foods and clinical specimens as, for example, in the case of enteroviruses (e.g. Norwalk agent) peak shedding of particles occurs within the first 48 hrs of symptoms appearing (Appleton, 1990).

Table 2.3.1 gives a detailed breakdown of the relative role played by the different pathogens in various countries during the 1980s, with Scotland giving this breakdown for the whole decade (1980-1990).

Closer scrutiny of these figures must be qualified, as very often statistical data can be misleading if not read in context. For example, some countries, when tabulating causative microorganisms, include 'chemical' or 'other toxic substances' in their total figures (e.g. Denmark). The Netherlands includes food poisoning outbreaks by 'spoilage' substances, which is not defined.
<table>
<thead>
<tr>
<th>COUNTRY</th>
<th>YEARS</th>
<th>No. of Outbreaks</th>
<th>Salmonella (%)</th>
<th>Campylobacter (%)</th>
<th>Cl. perfringens (%)</th>
<th>S. aureus (%)</th>
<th>Bacillus sp. (%)</th>
<th>E.coli</th>
<th>Enteroviruses (%)</th>
<th>Cl. botulinum</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCOTLAND</td>
<td>1980-90</td>
<td>2226</td>
<td>1852 (83%)</td>
<td>221 (10%)</td>
<td>71 (3%)</td>
<td>17 (0.7%)</td>
<td>25 (1%)</td>
<td>4</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>ENGLAND &amp; WALES</td>
<td>1986-89</td>
<td>2687</td>
<td>2350 (87%)</td>
<td>213 (8%)</td>
<td>38 (1.4%)</td>
<td>85 (3%)</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>DENMARK</td>
<td>1985-89</td>
<td>141</td>
<td>26 (18.4%)</td>
<td>2 (1.4%)</td>
<td>29 (20%)</td>
<td>12 (8.5%)</td>
<td>10 (7%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NETHERLANDS</td>
<td>1985-89</td>
<td>132</td>
<td>40 (30%)</td>
<td>10 (7.5%)</td>
<td>12 (9%)</td>
<td>8 (6%)</td>
<td>24 (18%)</td>
<td>6</td>
<td>4.5%</td>
<td></td>
</tr>
<tr>
<td>SPAIN</td>
<td>1985-89</td>
<td>2639</td>
<td>2338 (89%)</td>
<td>2</td>
<td>25 (1%)</td>
<td>173 (6.5%)</td>
<td>3</td>
<td>17</td>
<td>19 (0.7%)</td>
<td></td>
</tr>
<tr>
<td>W. GERMANY</td>
<td>1985-89</td>
<td>136</td>
<td>90 (66%)</td>
<td>4</td>
<td>7 (5%)</td>
<td>16 (12%)</td>
<td>4</td>
<td>7</td>
<td>5%</td>
<td>48</td>
</tr>
<tr>
<td>FRANCE</td>
<td>1985-89</td>
<td>1070</td>
<td>885 (83%)</td>
<td>88 (8%)</td>
<td>64 (6%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>U.S.A.</td>
<td>1983-87</td>
<td>909</td>
<td>342 (38%)</td>
<td>28 (3%)</td>
<td>24 (3%)</td>
<td>47 (5%)</td>
<td>16 (2%)</td>
<td>7</td>
<td>41 (4.5%)</td>
<td>74 (8%)</td>
</tr>
<tr>
<td>CANADA</td>
<td>1984-86</td>
<td>413</td>
<td>156 (38%)</td>
<td>30 (7%)</td>
<td>48 (12%)</td>
<td>54 (15%)</td>
<td>38 (9%)</td>
<td>11</td>
<td>(0.157)</td>
<td>6</td>
</tr>
</tbody>
</table>

Source: European Countries, (WHO, 1992)
USA, (Bean et al, 1990).
CANADA, (Todd, 1991).

SCOTLAND: (Sharp et al, 1982-1992))

1. Scotland:- all agents
2. England & Wales:- "Bacteriological" only.
3. Denmark: 'Incidents, includes "chem & toxic substances"
4. Netherlands: includes "spoilage"
5. Spain:- includes 'toxic' & 'various agents'
6. W. Germany: "Microbiological only" - excluding Botulism outbreaks
7. France: - includes "others".
8. U.S.A.:- Includes "chemicals & Parasites".
9. Canada: - Microbiological" only

*Outbreaks where an agent was recorded
In the latest report on Canadian data figures for outbreaks by 'plants' (e.g. algae, mushrooms etc.); 'chemicals' (e.g. pesticides, metals, solvents, etc.); and 'animals', (e.g. insect infestations) are included. However, only the microbiological data is presented here. The United States includes 'Scombrototoxic poisoning' incidents (presumably histamine poisoning) under the heading of 'CHEMICALS', whereas the Canadian figures do not refer to this type of fish poisoning at all.

The Germans quote the figures for botulism separately in their report, and state that these are unproportionately high and, therefore, unreliable/unrealistic (e.g. when compared to the total number of *Salmonella* outbreaks). This is because incidents of botulism are given priority within their surveillance programme with presumably more thorough investigations. Within the UK surveillance systems, England & Wales present separate tabulations for foodborne disease (i.e. caused by common bacterial pathogens) and 'other acute foodborne infections and intoxications' (incl. Brucellosis, Listeriosis, Hepatitis, Rotavirus and Scombrototoxic), whereas the Scottish surveillance authorities present one tabulation for 'foodborne disease outbreaks' and include bacterial, viral, chemical, scombrotoxic and 'unspecified' outbreaks in their total figures (Sharp et al. 1981-92).

Statistics on the incidence of foodborne disease in any country are only as reliable as the extent of surveillance and reporting in that country. This is evidenced, for example, by details of outbreaks given in Table 2.3.1 for both the US and Canada.
Looking specifically at the number of recorded outbreaks caused by *Campylobacter, Cl. perfringens, S. aureus*, and *Bacillus sps.* and considering:

(a) The vast difference in population - by a factor of 10 approx. in favour of the US.

(b) The time scale referred to in the reports, i.e. 5 yrs (1983/87) in the case of the US, and 3 yrs (1984/86) in the case of Canada.

It can be seen that, for each of these foodborne pathogens, higher outbreak figures were recorded in Canada than in the US. For *Salmonella* outbreaks, the US figures appear to be only slightly double the number recorded in Canada. So, in the absence of reliable statistical and epidemiological data in any country, details of outbreaks as presented in annual reports can only give an indication of trends in that country and not the true incidence of infection.

However, notwithstanding this, it is still possible to draw constructive conclusions, and make certain statements in relation to the data presented in Table 2.3.1. This summarises data obtained from a review of surveillance programmes for foodborne diseases in Europe, US and Canada.
2.4 International Trends in The Microbiology and Epidemiology of Foodborne Disease

Trends in relation to foodborne disease will be discussed under the following headings:-

2.4.1. Causative agents, their relative significance etc.
2.4.2. Foods most often implicated in food poisoning outbreaks.
2.4.3. The places where food poisoning outbreaks are acquired.
2.4.4. Major contributory factors in food poisoning outbreaks.

2.4.1 Causative Agents

1. Salmonella

This microorganism is by far the most important causative agent in recorded food poisoning outbreaks in the developed world, causing well over 80% of outbreaks in the UK, 87% in France, although causing a significantly lower percentage of outbreaks in North America (37%/38%). It is, however, interesting to note that Denmark is the only country in Table 2.3.1 where Salmonella has not been recorded as the principal causative agent.

In this case, Cl. perfringens has been more frequently isolated.

This has probably more to do with eating patterns as Cl. perfringens outbreaks most often involve institutional type catering, frequently implicating minced beef and stew-type dishes.
Furthermore, institutional outbreaks of food poisoning are probably more likely to be reported and investigated than those that occur in other types of catering establishments, and it is possible that surveillance in Denmark may concentrate to a greater degree on institutional type catering.

The significant role of *Salmonella sps.* was confirmed in another report (Sockett, 1991) which examined outbreaks of food poisoning implicating manufactured foods in England and Wales during the period 1980-89. In this report, *Salmonella sps.* were identified as causing illness in 132 (45%) of the 248 outbreaks where a causative agent was identified, although outbreaks associated with manufactured foods were quite small (5%) as a proportion of all outbreaks reported to the Communicable Disease Surveillance Centre (CDSC) during this decade. However, the sheer volume of manufactured foods on sale, and the wide distribution of these products (e.g. processed meats, etc.) meant that outbreaks due to *Salmonella sps.* had the potential to affect many people over a wide geographical area.

2. Camplylobacter

This caused anything from 221(10%) outbreaks (where the aetiological agent was identified) in the case of Scotland (1980-1990), to only two outbreaks in Spain (1985-1989). In the case of France, *Camplylobacter sps.* received no particular mention, but together with "others" (i.e. *Yersinia, B. cereus* and *Scombrotoxin*) accounted for 33(3%) of recorded outbreaks during this period (1985/89). Overall, *Camplylobacter sps.* do not tend to be implicated in recorded outbreaks of foodborne illness, as most incidents occur as sporadic cases, which themselves may be part of more widespread (i.e. not a point-source) and unrecognised outbreaks.
This introduces the importance of having a national surveillance system (which will be dealt with fully in the following section) for recording the incidence of gastrointestinal tract infections in communities. *Campylobacters sps.* are now recognised in the UK as the most frequently isolated pathogen under surveillance, having surpassed *Salmonella sps.* - a point perhaps not generally recognised.

England & Wales give separate tabulations for *Camplylobater sps.* in the latest WHO report (WHO, 1992) and record 192 outbreaks (implicating chicken and milk mostly) during the period 1986-89.

### 3. *Clostridium perfringens*

Again there was great variation in the recording of this microorganism, ranging from 20% of all confirmed 'incidents' of food poisoning in Denmark, to less than 1% in the case of Spain. It is not possible to easily explain discrepancies such as this. As most recorded outbreaks in both countries occur in the home, the difference may lie in the types of foods usually eaten in both countries.

The point has already been made about the particular association between minced beef, stews etc. and outbreaks of *Cl. perfringens*. A slight discrepancy also exists within the UK itself, where, notwithstanding the difference in timescale, almost 8% of outbreaks were caused by *Cl. perfringens* in England & Wales as against 3% in Scotland. Canada recorded double the number of outbreaks than that recorded in the US, but given the vast difference in population, (x10) and the longer time scale of the US report (5 years), it is impossible to sustain the fact that the US figures in any way represent the true incidence of this type of food poisoning.
4. **Staphylococcus aureus**

This type of 'true' food poisoning (with enterotoxin production on food during multiplication of bacteria) often results from human failure to observe the proper standard of personal hygiene, or from the use of raw milk in the manufacture of certain foods (e.g. some cheeses).

There was also a significant variation in the reporting of confirmed outbreaks of Staphylococcal food poisoning. For example, this microorganism caused 64 (15.5%) of 413 microbiologically confirmed outbreaks in Canada during 1984-86, while it only accounted for 17 (1%) of 2226 outbreaks in Scotland (1980-90). Both these countries have extremely detailed reporting systems and this difference cannot be simply explained by the fact that the Canadian total (413) refers to microbiological figures only, whereas the Scottish totals (2226) include less frequently identified agents such as 'chemical' contamination and histamine ('scombroid') poisoning.

The large difference in time span covering both reports would, however, have a bearing on the results. Given the relatively short incubation period of *S. aureus* food poisoning of 2-4 hrs, this may reflect more prompt reporting of this type of food poisoning in Canada, resulting in quicker investigation and retrieval of 'left-over' foods. However, this is very difficult to substantiate. The 38 (1.4%) outbreaks reported in England & Wales confirm that this microorganism plays only a relatively minor role in food poisoning incidents in the UK. Apart from W. Germany and Canada however its incidence never surpassed 10% of all outbreaks in the countries referred to in Table 2.3.1
5. **Bacillus cereus**

The most significant point here was the evident difference between the Netherlands (18% of outbreaks) and Spain (0.1%) in the reporting of this microorganism. Indeed, the fact that there were only 3 confirmed outbreaks of this type of food poisoning in Spain over the 5 year period (1985/89) confirms the extent of under-reporting and investigation of outbreaks of food poisoning caused by *B. cereus* given that rice, the food most often implicated in this type of intoxication, is a fairly common food in Spanish dishes.

The comparatively high incidence of *B. cereus* in the Netherlands can be explained by the fact that 30% of all foods implicated in outbreaks (1985/89) were associated with Chinese meals (presumably implicating rice).

In England & Wales 85 (3%) of 2687 outbreaks (1986/89) were confirmed as being caused by *Bacillus sps.* and another report (Anon(A), 1990), which looked at 58 outbreaks of *B. cereus* food poisoning for 1986/88 (England & Wales) found that rice was confirmed as the implicated food in 13 of these.

The 58 outbreaks recorded in the latter report, while still not reflecting the true incidence, is still far more reliable than the 3 outbreaks recorded in Spain between 1985-1989. However, with the exception of The Netherlands (18%) and to a lesser degree Canada (9%), *B. cereus* appears to be of little significance as a causative agent in food poisoning outbreaks generally. An exception to this would be in countries or communities where rice forms a major part of the diet. In Western societies, the vast increase in volume of ethnic catering in recent years is a major contributory factor in the emergence of this pathogen in food related illness.
6. **Clostridium botulinum**

This is a rare but quite often fatal form of food poisoning. Reference has already been made to the apparent high incidence in W. Germany, where surveillance of this particular type of food poisoning is very comprehensive.

In the US, there were 74 (8%) confirmed outbreaks in the 5 year period 1983-87. The number of cases per outbreak was very small (140 cases total), but there were 10 deaths recorded among these cases. Most (56) of the 74 incidents occurred in the home, with meat, fruit and vegetables being the foods most often implicated. Given the strong association between this type of food poisoning and preserved foods, it is likely that the majority of *Clostridium botulinum* food poisoning incidents in the US were acquired from home preserving/canning procedures.

While this is a very rare form of food poisoning in this part of the world, one cannot be too complacent because of its relatively high mortality rate. As recently as June 1989, it was reported in the UK that 27 people became ill after eating yoghurt flavoured with hazelnut conserve (O’Mahony et al, 1990). Investigations into the outbreak concluded that the implicated conserve had been sweetened with aspartame rather than sugar, and enquiries into the method of preservation of the conserve indicated that heat used in the manufacturing process was inadequate to destroy *Clostridium botulinum* spores.

Certainly, given the fatality rate of this type of food poisoning, modern food manufacturers producing long shelf-life foods in which anaerobic conditions favour the survival and growth of *Clostridium botulinum* bacteria, cannot be too careful with their preservation procedures.
7. Enteroviruses

While Table 2.3.1 may indicate that these microorganisms (mostly SRSVs of the Norwalk group) are of little significance when compared to other well recognised pathogens, the difficulty in detecting them in foods, together with their prompt shedding from the human bowel (within 48 hrs), means that they are more often epidemiologically associated with foodborne illness than confirmed microbiologically.

Enteroviruses generally (incl. adeno-, calci-, rota- etc.,) are mostly transmitted from person to person via the faecal oral route, although one study (Ho et al, 1989) suggested that droplet spray from the vomitus of cases can be another mode of transmission.

While Small Round Structured Viruses (SRSV) cause about 90% of foodborne outbreaks implicating viruses, and currently account for 2-5% of confirmed cases of viral gastroenteritis reported to the CDSC (England & Wales), (Fig. 2.4.1), other viruses, particularly that which causes Hepatitis A (HAV) have also been shown to be transmitted via food. The relatively low reported incidence of viral 'food poisoning' can be accounted for by:-

(a) The difficulty in isolating viruses from foods

While HAV can be grown in cell culture, virus isolation has never been achieved from food (Teo, 1992). The long incubation period of Hepatitis A (2-6 weeks) obviously presents its own difficulty with tracing and confirming a vehicle of infection. Further, with infectivity being greatest in the 48 hours before onset of jaundice (Teo, 1992), the taking and examining of faecal specimens is inappropriate.
(b) The delay in collection of clinical specimens

20%-40% of non bacterial outbreaks of gastroenteritis have been attributed to the *Norwalk agent* (Ho, 1989). Despite this, and despite the fact that very often clinical and epidemiological evidence is compatible with a viral source, the actual identification and confirmation of viral outbreaks of foodborne gastro-enteritis does not usually occur. In the opinion of this writer, one major cause of this is the delay in submitting stool specimens for electron microscopy (EM). The submission of two stool specimens (for bacteriological and virological examination) simultaneously is suggested, where epidemiological evidence may suggest a viral cause, given that viral particles fall below detectable levels so quickly. Alternatively, the retention of an early stool specimen, pending the result of the original sample sent for bacteriological examination, is recommended.

The vast majority of viral gastroenteritis occurs in children, with the rota virus (normally not foodborne) being the causative agent in approx. 85% of cases. **Fig 2.4.1** below compares the various enteroviruses reported in England & Wales in 1990. It clearly shows the dominance of the rota virus in viral gastroenteritis.

**Figure 2.4.1**

![Reports of Gastroenteritis in England & Wales 1990.](image)

Confirmed foodborne outbreaks of viral infection are of concern in this paper only. Research shows that in the vast majority of viral outbreaks of foodborne illness shellfish are implicated as the principal food vehicle (mostly Molluscs). A total of 107 outbreaks of food poisoning associated with eating bivalve molluscan shellfish (affecting 3,500 people) were recorded by the Communicable Disease Surveillance Centre (CDSC) of England & Wales in the 10 year period 1981-90 (Anon, 1991). This compares with 23 outbreaks (affecting 1,000 people) in the previous 10 years. This apparent increase in illness due to shellfish consumption may have been due to increased consumption of shellfish in the first instance, and better detection methods for viral particles in particular.

Table 2.4.1 below summarises the breakdown of causative agents of illness resulting from the consumption of molluscan shellfish in England & Wales during the period 1965-88.

Table 2.4.1  Outbreaks of Illness Associated with Molluscan Shellfish in England and Wales 1965-88

<table>
<thead>
<tr>
<th>Type of Outbreak</th>
<th>No. of Outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial Food Poisoning</td>
<td>12</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>17</td>
</tr>
<tr>
<td>Viral Gastroenteritis</td>
<td>37</td>
</tr>
<tr>
<td>Paralytic Shellfish Poisoning (PSP)</td>
<td>1</td>
</tr>
<tr>
<td>Red Welk Poisoning</td>
<td>1</td>
</tr>
<tr>
<td>Unknown*</td>
<td>101</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>169</strong></td>
</tr>
</tbody>
</table>

*Features often characteristic of viral gastroenteritis.

Source: Appleton H. (1990)
The important point here is that, of the 68 outbreaks where an aetiological agent was identified, 54 (79.4%) were attributed to viruses, 37 (68%) of which were described as 'viral gastroenteritis' (most probably SRSVs), and 17 (31%) were due to Hepatitis (HAV). This compares with 12 (17.6%) outbreaks caused by bacterial pathogens recorded for this period. The predominance of viral particles in outbreaks may be related to present methods of depuration of molluscs. In Ireland and the UK, this mostly involves cleansing by immersion in clean sea water for 36-48 hours which has been sterilised by ultraviolet light. Molluscs are filter feeders which concentrate organic matter (incl. microorganisms) within their flesh from the surrounding sea water. During immersion in clean sea water, molluscs expel waste material including bacteria but enteroviruses are not expelled with the same efficiency. This latter problem creates implications for public health, and this situation is further compounded by the resistance that viruses present to heat treatment.

Molluscs, such as cockles and mussels are, at least, 'cooked' to some degree before consumption, whereas oysters are mostly eaten in their raw state. Oysters were consequently the most commonly recorded shellfish (75 outbreaks) in England & Wales in the 10 year period (1981-90), with cockles next (12 outbreaks), and then mussels (4 outbreaks). 'Mixed shellfish' were recorded in a further 16 outbreaks (Anon, 1991).

Thorough heat treatment is the only sure way to destroy enteroviruses and, in the case of HAV, inactivation takes approx. 90 seconds if the core temperature of the shellfish flesh is held at 85°C-90°C (Appleton, 1990). As in so many incidents of 'gastroenteritis/food poisoning' where a causative agent is never isolated or identified, the possibility of a viral cause should be kept in mind. Finally, in incidents where molluscan shellfish, in particular, are implicated, viruses should be immediately suspected.
2.4.1.1 Emerging Bacterial Foodborne Pathogens

The bacteria discussed to this point are by far the most commonly recognised causative agents in foodborne illness. However, over the past decade, 'new' food poisoning agents have been emerging which have been linked epidemiologically and, in some incidents, confirmed bacteriologically to foodborne transmission. These include:

(a) *Listeria monocytogenes.*

(b) *E.Coli 0157*

Further, with regard to *Salmonella sps.*, these being the principal food poisoning agents in recorded outbreaks in most countries and comprising of over 2,200 serotypes, there has been a 'shift' or a change in the relative prevalence of serotypes during the 1980s. The emergence of one serotype, in particular *S. enteritidis*, also needs to be addressed and discussed, as this has posed a much greater threat to public health than *Listeria* and *E.coli* mentioned above.

For this reason, it is proposed to follow up this chapter with discussion on the emergence of *Salmonella enteritidis* and *Campylobacter jejuni* - now the two most important and significant microorganisms with regard to foodborne illness.
(a) **Listeria monocytogenes**

While *L. monocytogenes* has long been recognised as a human pathogen, confirmation of foodborne transmission has been rare. In fact, it was not until a series of outbreaks in the early 1980s that attention was focused on food as a possible vehicle of infection. These include:-

1. **Canada 1981**: (Lund, 1990) Outbreak affecting 7 adults and 34 perinatal cases. Two of otherwise healthy adults died and, of the perinatal cases, there were 5 abortions and stillbirths. Of the 25 live births, the case fatality rate was 27%. The food implicated was coleslaw, the cabbage coming from fields fertilised with manure from a flock of sheep in which Listeriosis had recently occurred.

2. **Massachusetts, US 1983**: (Lund, 1990) Outbreak affecting 49 people. 42 immunocompromised adults, 12 of whom died, and 7 perinatal cases with 2 deaths.

An association was demonstrated between many of the cases and the consumption of pasteurised milk from a single plant. While *L. monocytogenes* was not isolated from the pasteurised milk itself, it was however isolated from:

(a) A milk filter.
(b) Raw milk.
(c) Cows supplying milk to the plant.
3. **California 1985:** (Lund, 1990) Outbreak affecting 1,142 cases. 93 in pregnant women of Hispanic origin and 49 in non-pregnant adults. There were 48 deaths in all, including 20 foetuses, 10 neonates and 18 non-pregnant adults, the majority of which had a predisposing condition.

Epidemiological evidence linked the outbreak with a brand of 'Mexican style' soft cheese, and it was suggested that some of the milk used in the production of the cheese had not been pasteurised.

4. **Switzerland 1983-87:** (Lund, 1990) An epidemic occurred in the canton of Vaud, with 122 cases (61 materno-foetal, and 61 adults) with a case fatality rate of 27%.

The outbreak was attributed to infected 'Vacherin Mont D'or' cheese, and the environment in the ripening cellars was found to be heavily contaminated with *L. monocytogenes*.

5. **France 1992:** (Anon, 1992), (Weigen, 1992) 131 cases (subsequently updated to 279 cases) with 29 deaths recorded.

The outbreak strain in this incident was similar to that of the Californian and Switzerland outbreaks (serovar 4b), so it is possible that soft cheese may again have been implicated (implicating raw milk originally). Epidemiological investigations were ongoing at the time of writing.

In England & Wales, there have been documented cases, where organisms of the same serogroup and phagetype were isolated from both the patient and the incriminated food. **Table 2.4.2** below summarises these cases.
Again, serotype 4b appears to have particular association with foods made from milk. In the 1986 case, a woman (36) became ill with meningitis caused by *L. monocytogenes* (Bannister, 1987). She had eaten soft cheese from which a similar organism had been isolated. Finally, it is also worth noting that, with neonates, *L. monocytogenes* is the 3rd most common cause of bacterial meningitis after *E. coli* and *S. agalactiae* (Schlech et al., 1983).

**Table 2.4.2  Confirmed Foodborne Listeriosis-England & Wales.**

<table>
<thead>
<tr>
<th>YEAR</th>
<th>CASES</th>
<th>FOOD VEHICLE</th>
<th>SEROTYPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1986</td>
<td>1</td>
<td>Imported Soft Cheese</td>
<td>4b</td>
</tr>
<tr>
<td>1988</td>
<td>1</td>
<td>UK Goats Milk Cheese</td>
<td>4b</td>
</tr>
<tr>
<td>1988</td>
<td>1*</td>
<td>Cook-Chill Chicken</td>
<td>4b not fully</td>
</tr>
<tr>
<td>1988</td>
<td>1**</td>
<td>Vegetable Rennet</td>
<td>4b serotyped</td>
</tr>
</tbody>
</table>

*Stillbirth
**Miscarriage

**Source:** Various - In: PHLS Microbiology Digest 1989 6(2).

Since mid-1987, an active surveillance programme has existed in Scotland to monitor human Listeriosis. While this possibility accounts for the highest number of cases recorded in the UK (7 per million population), it is thought that an 'outbreak' situation - again caused by the serogroup 4b. existed from mid '87 to mid '89 (*Fig. 2.4.2*). By 1990, the figure had returned to what is regarded as its presumed baseline level. *(Note: these refer to surveillance figures which do not implicate any specific source of infection or mode of transmission).*
Laboratory reports of isolates of L. monocytogenes in the UK, 1980 - 1993: Isolation rates per 100,000 of population.

Adapted from surveillance data on gastrointestinal infections from CD(S)U, PHLS-CDSC, DHSS-NI.
Surveillance in England & Wales showed a more pronounced increase in laboratory reports of Listeriosis in the late 1980's from a low of 75 cases in 1980 to a high of 281 in 1988. As in Scotland, however, there was a fall in reported cases in 1989, despite more alert surveillance that year, so it is possible that during 1987/88 there was indeed an outbreak of Listeriosis in the UK. Laboratory reports from Northern Ireland, while of little overall significance (32 reported cases between 1983-1992), were also highest during the 1987/88 period.

Fig. 2.4.2 compares the incidence of Listeriosis within the UK on a rate per population basis. As can be seen, at no time did the incidence exceed 1 per 100,000, with surveillance data confirming a peak during 1987/88.

UK is not a notifiable disease in the UK (or indeed in Eire). However, in June 1989, the Scottish Social Services Committee reported on the problem of food poisoning and UK in particular. In the 2nd (follow-up) report (Anon(B), 1990) in 1990, this select committee made a number of recommendations including:

1. That careful consideration be given to designating UK as a notifiable disease.

2. That all pregnant women should be told exactly what the risks are of contracting listeriosis.

3. That in the case of stillbirth, where the cause of death is not evident, L. monocytogenes should be routinely looked for as a possible cause of death.

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1. PHLS - CDSC laboratory reports.
2. CD(S) U laboratory reports.
3. DHSS N.Irl. laboratory reports.
4. That high-risk foods (e.g. soft cheese, pate, coleslaw) should be stored at/or below 3°C, and that proposed amendments to the Food Hygiene Regulations should stipulate this.

In Ireland, the Ministers for Health and Agriculture & Food were sufficiently concerned about reports of increasing incidence of *L. monocytogenes* in the country that they referred the matter to the Food Safety Advisory Committee who reported on the matter in December 1989 (FSAC(A), 1989).

In this report, it is stated that while accepting that "there is overwhelming evidence that Listeriosis is primarily transmitted by food, there was no evidence to suggest that food was implicated in the 9 reported cases of Listeriosis during the previous 3 years (1986/89)". They complete their report by issuing 13 recommendations. These are varied but include:-

1. Confirmation of the Social Services Select Committees (UK) recommendation that vulnerable foods should be stored at a max. of 3°C.

2. There should be continued monitoring of known food sources for this pathogen.

3. Groups at risk should be made aware of the hazards of Listeriosis and advised regarding preventive measures via the Health Promotion Unit of the Department of Health.

4. That all milk intended for human consumption in any form should be pasteurised.

5. That foods like poultry and pâté should be thoroughly cooked during preparation, e.g. to temperature of at least 74°C.
Confirmed cases of foodborne Listeriosis are extremely low given the ubiquity of the microorganism and its prevalence in so many foods. Many surveys have been carried out to determine the contamination rates of foods. One such study (Sheridan, Duffy, 1992) looked at the occurrence of *L. monocytogenes* from a selection of meat and meat products in Ireland (Table 2.4.3). The results showed that, with the exception of minced beef, there was little variation between *Listeria* rates found in other countries and in Ireland. Examiners were surprised about this as the contamination rate in beef before mincing was quite high in the Irish survey (25%), leading one to assume that contamination rates for minced beef would be similar.

Table 2.4.3 Occurrence (%) of *L. monocytogenes* in Irish Meat Samples Compared to Other Countries.

<table>
<thead>
<tr>
<th>PRODUCTS</th>
<th>IRELAND</th>
<th>OTHER COUNTRIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beef</td>
<td>25.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Minced Beef</td>
<td>5.0</td>
<td>21.0</td>
</tr>
<tr>
<td>Chicken</td>
<td>30.0</td>
<td>31.0</td>
</tr>
<tr>
<td>Cooked Meats</td>
<td>10.0</td>
<td>17.0</td>
</tr>
<tr>
<td>Frozen Meats</td>
<td>18.0</td>
<td>21.0</td>
</tr>
</tbody>
</table>


The 10% contamination rate in cooked meats, which is low in comparison to other countries (17%), is still quite alarming, given that *Listeria* is not particularly heat tolerant, therefore, probably indicating postcooking contamination. Initial counts ranged from 5-50 (cfu/g) but, as the infective dose is not known, (and is thought to vary from person to person) initial numbers of organisms in foods are important, particularly as such foods may be eaten by vulnerable groups (e.g. pregnant women).
A survey carried out in 1990 in Belfast into prepacked ready-to-eat sandwiches (Wilson, 1990) - found Listeria present (in 25g) in 57 (13%) of 433 sandwiches examined. This was quite similar to L. monocytogenes found in cooked meats in the aforementioned Irish survey. However, in 3 (0.7%) of the sandwiches, Listeria spp. were found in numbers greater than 10^2 per gram.

In 1989, the PHLS reported on a comprehensive survey involving 18,337 foods (incl. sandwiches, pates, liquid egg, bottled water, soft and hard cheese, ice-cream, milk, cooked fish, pizza, gravy etc.) (McLauchlin, Gilbert, 1990), Listeria spp. was present in 1,814 (10%), with L. monocytogenes found in 1,159 (6%) of all samples. L. monocytogenes was present in 116 (17%) of ready-to-eat pates which is alarming, with a 7% contamination rate for sandwiches (type not stated) - slightly lower than the 10% contamination rate for 'cooked meats' in the Irish survey.

In Ireland, Listeriosis is only notifiable as 'food poisoning' (if thought to be foodborne in the opinion of a medical practitioner). It is not specifically notifiable otherwise. While Listeriosis is a rare infectious disease in Ireland, the overall ubiquity of Listeria spp., in which quite a wide variety of foods appear to be contaminated, together with its relatively high mortality rate, makes the surveillance of L. monocytogenes an important public health issue.
(b) *E. Coli 0157*

*E. coli* microorganisms form part of the facultatively anaerobic commensal flora of the gut of many warm blooded animals including man. However, it is well recognised that pathogenic strains exist, (Doyle, 1990) (Gross and Roberts, 1990) such as:-

1. Enterotoxigenic *E. coli* (ETEC) - producing a heat stable or heat labile enterotoxin, the most common cause of travellers diarrhoea. A large waterborne outbreak has been described (Doyle, 1990) in the US in 1975 with, at least, 2,000 people being infected.

2. Enteropathogenic *E. coli* (EPEC) - this strain has been associated with numerous outbreaks of infantile enteritis, but is no longer a major public health problem in this part of the world.

3. Enteroinvasive *E. coli* (EIEC). This causes a dysentery-like disease, affecting all age groups and has been shown to be food related. An outbreak in the US in 1971 was associated with imported cheese, and affected 387 people (Doyle, 1990).

4. A fourth group has been described in recent years. This group (VTEC), which produces a verocytotoxin and first described in Canada in the late 1970s, was subsequently recognised in the US in the early 1980s as being responsible for two clinical syndromes.

   (a) Haemorrhagic Colitis (HC).
   (b) Haemolytic Uraemic Syndrome (HUS).
During the 1980s, the incidence of infection due to VTEC (serogroup 0157) increased dramatically in the US and Canada, and these organisms are now recognised as a major public health problem.

Summer outbreaks due to *E. coli 0157* are quite common now in the UK. While epidemiological evidence may suggest a food source in many such outbreaks, it has proved impossible so far to confirm this microbiologically. In fact, as far as the writer is aware, *E. coli 0157* has only been isolated from implicated food on one occasion to date in the UK. Examples of some outbreaks in the UK in recent years include:-

(a) 16 cases in N.W. England in 1991, with a case control study showing significant association between infection and the consumption of a locally produced yoghurt (Anon(A), 1991)

(b) 22 of 93 persons who attended a christening reception became ill in June 1987 - 6 of whom were hospitalised. There was an association between eating a pork pie or turkey roll and becoming ill (Anon, 1987).

(c) 16 cases of *E. coli 0157* infection (4 of whom needed dialysis) in Lothian, Scotland were associated with eating in a local restaurant. Notable features of this outbreak were the wide range in incubation periods (1-14 days) and the occurrence of secondary spread through asymptomatic carriers (Marsh et al, 1991)
(d) 4 confirmed cases of a possible waterborne outbreak in Grampian, Scotland in June 1990. Further investigations revealed that several other school children in the locality had been 'off sick' with diarrhoea for a couple of days concurrent with the 4 confirmed cases. Water samples taken at the homes of these 4 cases showed heavy contamination with faecal coliforms, although \textit{E. coli 0157} was not isolated in samples tested (Dev. et al, 1991).

(e) In February 1990, \textit{E. coli 0157} was isolated from 2 children in South Wales who were reported to have eaten cooked sausages of the same brand purchased at a local butcher (Anon(B), 1991).

The largest outbreak ever recorded was in the US in January 1993, affecting 230 bacteriologically confirmed cases in Washington State. Some of the cases developed Haemolytic Uraemic Syndrome (HUS) and one died. Investigators have linked the outbreak to the consumption of 'hamburgers' (beef burgers) from a fast food chain, and \textit{E. coli 0157} was isolated from implicated batches of ground beef (Anon(A), 1993).

\textit{E. coli 0157} is only emerging in this part of the world as a foodborne pathogen, as this particular strain of \textit{E. coli} has only come to the attention of surveillance authorities in recent years. Obviously, the recorded incidence of food poisoning outbreaks is greatly under-reported but, with improved isolation methods and increased interest in the organism, it is probable that many more outbreaks will be reported in the future. Referring back to Table 2.3.1, only the Canadian surveillance unit specifically mention \textit{E. coli 0157} in their annual reports (11 incidents).
While many European countries refer to 'E.coli' outbreaks, the particular strain is not clarified (WHO, 1992). However, in outbreaks in which foods of cattle origin are implicated and where investigators have failed to identify the more common foodborne pathogens, a search should be made for *E.coli 0157* as the possible causative agent. Little is known about contamination rates of foods with *E.coli 0157*. However, surveys carried out in the US (Doyle, 1990) of retail raw meats and poultry indicate contamination rates for ground beef (3.5%), pork (1.5%), poultry (1.5%) lamb (2.3%). Evidence from a survey of mince beef carried out within the Southern Health Board region suggests a contamination rate with this pathogen of 1%. (J. Boland, EHO, and H. Cowman, Food Hygiene Lab. Cork, - personal communications).

As *E.coli 0157* is not an unusually heat resistant microorganism, it is very likely that most outbreaks result from the undercooking of ground beef (minced meat), or possibly from the consumption of contaminated raw milk. Cross contamination from raw minced beef to cooked meats may also be a contributory factor, particularly as, in recent years, many 'traditional' butcher shops have diversified to include the cooking, cutting and display of cooked meats on the premises. Food operators need to be educated to minimise the opportunity for cross contamination.

The reported incidence of *E.coli 0157* is much greater in Scotland than in the rest of the UK, increasing significantly in 1988 and peaking in 1991 (Fig. 2.4.3). This large difference is difficult to interpret, although it may be explained by a greater awareness and, therefore, more intensive surveillance within the Scottish unit. The incidence, by comparison, appears to be much lower in England & Wales and in Northern Ireland, with the former on only one occasion (1992) reaching a rate of 1/100,000 population. Fig. 2.4.3 demonstrates these rates recorded by the the 3 UK surveillance centres for the years 1983/93.
Laboratory reports of isolates of E. coli 0157 in the UK, 1980-1993

Adapted from CD(S)U, PHLS-LEP, DHSS-NI reports
2.4.1.2 Major Causative Agents Of Foodborne Illness in the UK

*Salmonella sps.* and *Campylobacter sps.* have emerged to become the two most frequently isolated enteropathogens in gastro-intestinal disease surveillance in the UK. Their relative significance for 1991 and 1992 in England & Wales in comparison to other intestinal tract pathogens can be seen in Fig. 2.4.4, with similar data for Scotland in Fig. 2.4.5. Throughout the 1980s, there has been considerable increase in the reporting of gastrointestinal infections by the PHLS, and this has been largely due to increases in laboratory reports of *Campylobacter* and *Salmonella* infections. (Fig. 2.4.6). There have been significant increases in laboratory reports of *Campylobacter sps.* since the early 1980s, with *Salmonella sps.* showing a roughly corresponding rate of increased isolations since 1985 in particular (Fig. 2.4.6).

The relative significance of *Campylobacter sps.* and *Salmonella sps.*, expressed as a percentage of total isolates, is slightly smaller as can be seen from the Scottish data (Fig. 2.4.5). It is, nevertheless, very clear that these are also the two most prevalent enteropathogens reported in Scotland.

What is also significant is that, in both surveillance systems, *Campylobacter* isolates exceeded those for *Salmonella* by an average 10 percentage points for 1991 and 1992, with the 3rd most frequently isolated enteropathogen (*rotavirus*), playing a relatively equal role (14%). ‘Other’ gastrointestinal tract infections are of greater significance in the Scottish surveillance unit, accounting for approx. 36% of all isolates, whereas in England & Wales, they accounted for approx. 27% of all isolates.
Table 2.4.4

Surveillance of Gastrointestinal Tract Infections

1991 N=94,034

- Cryptosporidium: 6%
- Shigella: 11%
- Rotavirus: 14%
- Campylobacter: 35%
- Salmonella: 24%
- Others: 10%

1992 N=120,106

- Cryptosporidium: 4%
- Shigella: 15%
- Rotavirus: 15%
- Campylobacter: 32%
- Salmonella: 26%
- Others: 9%

Reference: Adak, G.K., 1993, Personal Communication

Figure 2.4.4

Surveillance of Gastrointestinal Tract Infections
Figure 2.4.5
Surveillance of Gastrointestinal Tract Infections


1991 N=11,487
Cryptosporidium 17%
Shigella 6%
Campylobacter 30%
Salmonella 20%
Others 14%

1992 N=17,206
Cryptosporidium 6%
Shigella 23%
Campylobacter 28%
Salmonella 17%
Rotavirus 14%
Others 12%


45
Main Intestinal-tract Infectious Agents
Laboratory Reports from England and Wales, 1980-1992

Adapted from PHLS-CDSC reports, *1992 figures provisional.
Looking at the overall picture throughout the last decade or so, one can see the complete dominance of *Campylobacter sps.* and *Salmonella sps.* in the surveillance of gastro-intestinal infectious diseases in England & Wales (Fig. 2.4.6). While Fig. 2.4.6 refers to total isolates of both *Campylobacter sps.* and *Salmonella sps.* (England & Wales), Fig. 2.4.7 and Fig. 2.4.8 give the isolation rates on a population basis throughout the UK. This allows for more direct comparison of the incidence of Campylobacteriosis and Salmonellosis within the UK. For *Campylobacter sps.* (Fig. 2.4.7), it can be seen that the reported incidence of infection caused by this enteropathogen has been much lower in Northern Ireland than in the rest of the UK during this period, with Salmonella infections showing a similar trend (Fig. 2.4.8).

*Salmonella sps.* cause the vast majority of foodborne outbreaks in England & Wales (e.g. 900-1000 outbreaks are currently reported each year to CDSC) (Anon(B), 1992), with *Campylobacter sps.* accounting for the largest number of individual isolates reported by medical laboratories throughout the UK. This probably implies that Campylobacter enteritis occurs mostly as a sporadic infection, with Salmonellosis incidents having a point-source (e.g. a hotel function) and, therefore, more likely to be reported and investigated. However, it is very likely that outbreaks of food poisoning caused by *Campylobacter* are greatly underreported and many of the sporadic cases may actually be part of more widespread (as opposed to point-source) outbreaks, with poultry being a likely vehicle of infection.

*Salmonella sps.* have long been recognised as major foodborne pathogens whereas *Campylobacter sps.* only emerged as 'new' food poisoning agents in the late 1970s with the development of selective media (Pearson, Healing, 1992). There was a dramatic increase in routine isolations of these pathogens in the following years as is shown in Fig. 2.4.6.
Laboratory reports of isolates of Campylobacter spp in the UK, 1980-1993

Adapted from CD(S)U, PHLS-CDSC and DHSS-NI reports.
Laboratory reports of isolates of Salmonella spp in the UK, 1980-1993

Adapted from CD(S)U, PHLS_CDSC and DHSS_NI reports.
Campylobacter enteritis is caused by two closely related species (C. jejuni & C. coli - 100 serotypes) but, in most parts of the world, C. jejuni is the predominant species causing 80-90% of infection (Skirrow, 1990). There are, on the other hand, 2,200 serotypes of Salmonella sps., although fewer than 200 cause human illness in the UK in any year (Baird-Parker, 1990).

As more laboratories began to identify and report Campylobacter sps., isolates increased to such a degree that, by 1981, (England & Wales)\(^1\); 1985 (Scotland)\(^2\) and 1991 (Northern Ireland)\(^3\); Campylobacter had clearly overtaken Salmonella as the most frequently isolated enteropathogen. Much of the increased infection is certainly food related as Campylobacter sps. have been isolated from a wide range of foods including poultry, milk and water. However, the epidemiological and bacteriological evidence is much less clear (when compared to Salmonella sps.), as a lack of discriminating and reliable techniques for epidemiological analysis had, until relatively recently, hindered our understanding of Campylobacter food poisoning.

Referring back to Table 2.3.1, it can be seen that there was a total of 2,350 recorded Salmonella outbreaks in England & Wales during 1986-89. Of these 2,350 outbreaks, 672 were caused by S. typhimurium, 1,107 by S. enteritidis, and 571 by 'other' Salmonella sps. (WHO, 1992). There is no specific reference in this table to outbreaks caused by Campylobacter sps. as, in WHOs 5th report (1992), the CDSC contribute separate data for Campylobacter under the heading 'Foodborne Campylobacter Outbreaks - by Incriminated Foods' (Unlike other pathogens where the breakdown is recorded under 'causative agent'). Under the former heading, it is stated that for 1986-89 there were 192 recorded outbreaks of Campylobacter food poisoning although, in only 41 (21.3%) outbreaks, was the suspected food mentioned.

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1  PHLS - CDSC laboratory reports.
2  CD(S) U laboratory reports.
3  DHSS N.Irl. laboratory reports.
In only 4 of these 192 outbreaks was there epidemiological and bacteriological evidence of an association of food with illness. It would appear that, in the vast majority of outbreaks, a suspected food was assumed to have been incriminated without clear epidemiological evidence, not to mention bacteriological confirmation. Of the incriminated food mentioned, both milk and chicken account for most of the outbreaks recorded. These two food sources are also well recognised as common vehicles of infection for *Salmonella* sps., so it is likely that these two pathogens have common reservoirs of infection.

This is a very important point to note with regard to the control and prevention of both forms of gastro-intestinal diseases. It has been noted that clinical manifestations of both illnesses can be similar, but the intensity and duration of abdominal pain is on average greater for Campylobacter enteritis. The average incubation period for Salmonellosis tends to be shorter (12-36 hrs) whereas, for Campylobacteriosis, it is usually about 3 days. Further, chronic carriage for healthy people who have become infected with *Campylobacter* sps. is unknown, while it is well recognised that the excretion phase for Salmonellosis can continue for 3 months or more after first becoming infected (Smyth, 1989). This may have serious implications for public health as far as food service establishments are concerned, particularly if infected food operators do not practice or observe proper standards in personal hygiene.

From a food hygiene point of view, one major difference between both microorganisms is that *Salmonella* sps. readily multiply on foods at ambient temperatures, whereas *Campylobacters* sps. require living organisms or mammalian hosts to multiply (Skirrow and Benjamin, 1980). This may also explain why there are far more recorded outbreaks implicating *Salmonella* sps., given that storage of food at ambient/room temperature for extended periods is a major contributory factor in many food poisoning outbreaks.
While several foods have been implicated in incidents of salmonellosis and campylobacteriosis, two foods in particular have been associated very frequently with such incidents, i.e. poultry and milk. *Campylobacter jejuni* is a natural constituent of poultry faeces and most isolations from wildlife in the UK are from birds. While up to 25% of cows may be carriers (Beumer et al., 1988), other domestic animals such as pigs (usually *C. coli*), sheep, dogs and cats may also harbour these microorganisms.

*Campylobacter sps.* are present in nearly all surface waters which, if distributed untreated, can lead to major outbreaks of Campylobacter enteritis. One such outbreak involving approx. 680 of 1,000 inhabitants of a Northern Norway community has been described (Melby et al., 1991), where it was suggested that 3 serotypes of *C. jejuni* were the causative agents. However, *Campylobacter sps.* have only rarely been isolated from drinking water during suspected outbreaks.

In natural waters they survive in viable but nonculturable forms (Rollins, Colwell, 1986) and in the Norway outbreak (where 2 isolation techniques were applied), growth of colonies from tap water samples showing characteristics consistent with those of *Campylobacter sps*, was obtained from only one of the two techniques applied (Rollins, Colwell, 1986).

Epidemiological data from many countries has confirmed the role of poultry as a source of human infection of *Campylobacter*. Further, data from England & Wales for the years 1981-88 shows that increased incidence of Campylobacter enteritis in humans reported during this period paralleled an increase in the consumption of fresh chickens. (Fig. 2.4.9).
Figure: 2.4.9

Campylobacter Infections and Chicken consumption

- Fresh chicken consumption
- Campylobacter Infections

Adapted from PHLS, CDSC (British Food Information Service).
With 300 million chickens being sold in the UK annually (Smith, 1989), this major source of *Campylobacter* (and *Salmonella*) infection requires extreme care as far as their handling, preparation and storage is concerned. Indeed, a survey of commercially reared flocks in Ireland indicated that as many as 92% were found to be positive for *Campylobacter sps.* (FSAC(A), 1991) with a different study finding counts of up to $1.5 \times 10^6$ per bird in fresh chickens, and up to $2.4 \times 10^7$ in unevicerated birds (Hood et al., 1988). In England & Wales, milk is the most common food item reported to be associated with *Campylobacter* outbreaks, with 9 reported outbreaks affecting 872 people recorded during the period 1987-89 (Sockett, 1991). (There were 7 milkborne incidents, all involving unpasteurised milk caused by *Salmonella sps.* during this period). In Scotland, milkborne outbreaks of food poisoning declined greatly in the years after 1983, when the sale of raw milk was effectively banned. Such outbreaks have since been virtually eradicated with, for example, recorded outbreaks declining by one-third between the 1980/82 period (27 outbreaks) and 1986/88 (9 outbreaks) (Sharp, 1989). The Richmond Committee in their report (Anon(D), 1991) drew attention to the hazards associated with the consumption of untreated milk. These hazards were primarily concerned with infections caused by *Salmonella* and *Campylobacter sps.* but, despite this, and in contrast to Scotland, the sale of raw milk has not been banned in England & Wales.

The role of *Salmonella sps.* in the incidence of foodborne infections is so well documented that it is not necessary to elaborate on this (Table 2.3.1). Major outbreaks have been described implicating, ‘manufactured’ foods (Sockett, 1991); mayonnaise (Mitchell et al., 1989); fresh shell eggs (Mawer et al., 1989); combi-oven cooked eggs (Ahmed et al., 1992); scrambled eggs (Feng-Ying et al., 1988); salami (Cowden et al., 1989); rotisserie chickens (Riley et al., 1988) mousse (Merrington, Morris 1988) poultry (Woolaway et al., 1988), and raw milk (Dillon, 1984).
As with Campylobacteriosis, poultry meat poses the greatest risk of causing foodborne Salmonellosis. In Scotland, of the 1380 households and general outbreaks recorded, between 1980 and 1985, a food vehicle was only identified in approx. two thirds (Sharp et al, 1988). However, where the actual food vehicle was identified in these outbreaks, poultry meat was incriminated most frequently (54%). In England & Wales, during the five year period 1987-91, 128 outbreaks (affecting 3,500 people) of food poisoning associated with poultry meat were reported to the CDSC amounting to 14% of all general (i.e. non-domestic) outbreaks recorded in this period (Anon(C), 1991). Table 2.4.4 reflects the significant role played by poultry meat as the suspected vehicle of infection in outbreaks of salmonellosis for various periods between 1959 and 1985 in England & Wales. It can be readily seen that, apart from the period (1959-62), poultry meat was recorded as the suspected vehicle of infection in a third to a half of all incidents where a food vehicle was identified.

Table 2.4.4  
Salmonella Food Poisoning: Suspected Vehicles of Infection in Family and General Outbreaks (England & Wales)

<table>
<thead>
<tr>
<th>Years</th>
<th>Total No. of Vehicles</th>
<th>Vehicles associated with poultry</th>
</tr>
</thead>
<tbody>
<tr>
<td>1959-62</td>
<td>70</td>
<td>9 (12.9%)</td>
</tr>
<tr>
<td>1969-72</td>
<td>127</td>
<td>66 (52.0%)</td>
</tr>
<tr>
<td>1981-83</td>
<td>347</td>
<td>179 (51.3%)</td>
</tr>
<tr>
<td>1984-85</td>
<td>177</td>
<td>57 (32.2%)</td>
</tr>
</tbody>
</table>


The association between infection caused by *Salmonella sps.* and poultry meat is not surprising given the demand for poultry meat in this part of the world in the first instance, and contamination rates recorded in various surveys.
Table 2.4.5 summarises surveys carried out in England and in Scotland since 1979. The surveys undertaken in England refer to chickens bought by Environmental Health Officers, mainly in various retail outlets including butcher shops, supermarkets, frozen food outlets etc. The surveys show that contamination rates (all *Salmonella sps.*) vary from 79% in the 1979/80 survey, to 48% in the latest (1990) (Roberts, 1991). While this might appear to indicate that the overall rate of *Salmonella* contamination of chicken carcasses may be declining, this would need to be substantiated with up-to-date surveys. What is perhaps more significant is the increase in the rate of isolation of *S. enteritidis PT4* from a low of zero in the earlier survey to 21% in the 1990 survey. This latter issue had serious implications for public health in the UK, generally in the latter half of the 1980s, resulting in an epidemic of *S. enteritidis PT4* food poisoning, implicating poultry and egg products with a predominance of cases infected with *S. enteritidis PT4*. This will be discussed in greater detail later.

A major survey in Scotland between February 1988 and March 1989 that examined *Salmonella* contamination rates of chicken carcasses delivered to the kitchen of a long-stay hospital (psycho-geriatric) confirmed the 1990 survey in England (Reilly et al, 1991). In this particular survey, of a total of 477 chicken carcasses screened during this 13 month period, 214 (45%) of individual carcasses carried one or more *Salmonella* serotypes and every single consignment examined contained affected carcasses. Simultaneous to carcass screening, sewers draining the residential accommodation (excl. kitchen effluent) were also monitored. Thirty (38%) of the 79 'Moore Swabs' put down were positive for *Salmonella sps.*, and there was a statistically significant association between the *Salmonella* serotypes isolated from chickens and those isolated from the sewers (swabs) the following week.
Poultry meat does not play as significant a role in incidents of salmonellosis in the US as it does in the UK. While *Salmonella* sps. are not unusually heat resistant, some experts in the US believe that their countries passion for rare beef (which incidently carries very low levels of *Salmonella*) explains why beef has been implicated in more reported outbreaks of salmonellosis than poultry (USDA, 1988). The incidence of salmonellosis in the US would certainly be much greater if the *Salmonella* carriage rate in beef was similar to that for poultry, or indeed if the rate of poultry consumption was higher in the population.

Table 2.4.5  
Salmonella in Retail Chickens

<table>
<thead>
<tr>
<th>Salmonella</th>
<th>Proportion containing Salmonellae</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frozen</td>
</tr>
<tr>
<td>A. F.C. (1990) All sps.</td>
<td>79/146 (54%)</td>
</tr>
<tr>
<td>S. enteritidis PT.4</td>
<td>33/146 (23%)</td>
</tr>
<tr>
<td>B. FHL (1987) All sps.</td>
<td>65/101 (64%)</td>
</tr>
<tr>
<td>S. enteritidis PT4</td>
<td>29/101 (20%)</td>
</tr>
<tr>
<td>C. FHL (1979/80) All sps.</td>
<td>79/100 (79%)</td>
</tr>
<tr>
<td>S. enteritidis PT4</td>
<td>0/100</td>
</tr>
<tr>
<td>D. Scotland (1988/89)</td>
<td>N/S</td>
</tr>
</tbody>
</table>

NE = Not Examined.  
FC = Food Chain Lab (PHLS).  
NS = Not Stated.  
FHL = Food Hygiene Lab (PHLS)

Sources:  
The Emergence of S. enteritidis PT4

Surveillance indicators confirm that an epidemic of unprecedented proportions occurred in the UK during the latter half of the 1980s, and the most noticeable trend was reflected in the relative prevalence of the two most frequently isolated serotypes, i.e. *S. typhimurium* and *S. enteritidis*. The former had been the predominant serotype right through the 1960s and 1970s, with *S. enteritidis* remaining the 2nd or 3rd most prevalent serotype in human infections of salmonellosis. *S. virchow* was the second most common serotype in 1984, but has since been relegated to third place and is likely to remain so.

The emergence of *S. enteritidis* from about the mid-80s throughout the UK was so drastic that by 1987 (Scotland)\(^1\) and 1988 (England & Wales)\(^2\) and (Northern Ireland)\(^3\), this serotype exceeded *S. typhimurium* for the first time. Figs. 2.4.10-2.4.12 outline the relative significance of the two main *Salmonella* serotypes for the years 1990-92 in the UK, with the predominance of *S. enteritidis* serotypes, particularly *phagetype 4* clearly evident in Scotland and England & Wales, although less so in Northern Ireland.

For the first time also, a new food source became epidemiologically implicated in increased outbreaks of salmonellosis which coincided with increased isolations of *S. enteritidis*, particularly *phagetype 4 (PT4)*. While, traditionally, poultry was the food most frequently implicated in outbreaks of *Salmonella* food poisoning, in the latter half of the 1980s, eggs and egg based products (e.g. Mayonnaise etc.) became increasingly incriminated, until it became clear to public health and surveillance authorities that they were dealing with an epidemic of unprecedented proportions.

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1. CD(S)U laboratory reports (Sharp, 1993)
2. PHLS - CDSC laboratory reports (Adak, 1993)
3. DHSS, N. Irl laboratory reports (Mitchel 1993)
Foods made from raw eggs in particular were identified as a major source of infection. Some reported incidents of these have previously been referred to. Other examples include outbreaks implicating home made ice-cream and egg based desserts (Cowden et al, 1989). In the U.K, the Department of Health became so concerned about increased illness associated with the consumption of eggs that it issued a series of circulars in 1988, advising the public of the potential hazard, and recommending that eggs be adequately cooked before consumption (DoH, 1988). Caterers were also advised to only use pasteurised eggs for food preparation.

Eggs were also reported to be associated with increased incidents of salmonellosis in the north eastern states of the US. For example, a series of outbreaks of food poisoning involving one particular restaurant chain has been described (Feng-Ying et al, 1988). An outbreak in one of these restaurants resulted in at least 71 illnesses, with 17 persons known to have been hospitalised. Scrambled eggs, served at a breakfast bar were implicated as the vehicle of infection in the incident.

The latest annual report from France (1991), (Anon(B), 1993) which confirmed that *S. enteritidis* was the most common serotype in recorded outbreaks of food poisoning (49%), identified eggs and egg products as the most commonly incriminated food. In fact, in France in 1991, outbreaks caused by eggs and egg products were double the number implicating poultry and the other meats.
Relative significance of the main Salmonella serotypes: England and Wales

Relative significance of the main Salmonella serotypes: Scotland

Reference: Sharp, J.C.M., 1994, Personal communication
Relative significance of the main Salmonella serotypes: Northern Ireland

Reference: Mitchell, D., 1993, Personal communication
The increase in isolations of *Salmonella sps.* in humans in the UK since 1985, has been almost entirely due to the phenomenal increase in reports of *S. enteritidis PT4*, which clearly reached epidemic proportions by the late 1980s. For example, in England & Wales in 1990, *S. enteritidis* accounted for 62% of all *Salmonella* isolates and PT4 accounted for 78% of these. This is in stark contrast to total isolates of *S. typhimurium* which accounted for only 18.4% of all *Salmonella* isolates for that year. In 1991, *Phagetype 4*, on its own, accounted for 48% of all *Salmonella* isolates from humans, and confirms that the explosive increase in isolations of *Salmonella sps.* was mainly due to an epidemic of this phage type.

Several other countries have reported a very high incidence of *S. enteritidis PT4* infections, and the microorganism has been isolated from human cases and foods from at least 10 European countries, and from countries as far apart as Japan and Argentina (Baird-Parker, 1990).

While eggs have been epidemiologically and microbiologically incriminated in several outbreaks of food poisoning, meat from broiler chickens, also contaminated with *S. enteritidis PT4*, has been an important cause of outbreaks and sporadic cases. The successful adoption of *S. enteritidis PT4* in poultry flocks during the 1980s seems to have followed the pattern of the avian adopted serotype *S. pollorum*. Infection in the parent stock, particularly in layers, can permit transovarian infection, resulting in the contents of intact shell eggs becoming contaminated. During the late 1980s, however, and despite the epidemic, the incidence of this was thought to be very low.

One survey by the PHLS in England in 1988 examined 2,000 eggs from suspect sources. *S. enteritidis PT4* was cultured from the internal contents of only 2 eggs (PHLS, 1989).
As it can be difficult in practice to trace eggs to the source flocks, it has been suggested that veterinary studies would be more beneficial if aimed at establishing infected flocks. Further, the continued investigation of flocks shown to be linked to incidents of egg associated food poisoning would also be a much more rewarding exercise (PHLS, 1989). Another extensive survey carried out by PHLS in 1991 studied 7,045 six-egg samples of British eggs and 8630 6-egg samples of imported eggs, found that 47 (0.7%) of the former and 19 (0.2%) of the latter samples were contaminated with *S. enteritidis*. The majority of these were Phagetype 4 (Louvois, 1992). The Richmond Committee in their report (Anon(C), 1990) has acknowledged this unprecedented epidemic of Salmonellosis due to *S. enteritidis* PT4, and made reference to a PHLS working party set up in 1987 to carry out an epidemiological study into the epidemic. This study showed that the foods most commonly eaten in the week before onset of symptoms included egg sandwiches, egg based dishes and poultry. The Committee recognised the importance of this study, and concluded that "there is little doubt that the increase (in *Salmonella* isolations/cases) has been due not only to poultry meat, but also to the contents of intact shell eggs.

The UK Government was so concerned about the alarming increase in cases of salmonellosis that it introduced a series of measures to limit *Salmonella* infection in food animals, with the aim of minimising the introduction of this microorganism into the human food chain. These measures (Zoonosis orders) will be discussed in the final section of the thesis, in which the various approaches necessary to reduce or minimise foodborne infections and intoxications in humans will be analysed.

*Salmonella* sps. are the most commonly recorded causative agents in point-source outbreaks of foodborne disease, with *Campylobacter* sps being the most frequently isolated enteropathogens in clinical laboratories in the UK.
Both microorganisms very often share a common vehicle of infection (e.g. poultry and raw milk), although *Salmonella sps.* are also thought to be spread directly from person to person, whereas there is little known about this mode of transmission for *Campylobacter sps.* All measures aimed at reducing the incidence of foodborne disease must target these two pathogens primarily, and the success of such preventative measures should be judged against the decline (or increase) in the incidence of these two infections in a community.

2.4.2. Implicated Foods

Foods that tend to be implicated in reported food poisoning outbreaks have already been discussed to some extent in relation to the major foodborne pathogens, particularly *Salmonella* and *Campylobacter sps.* Various foods that have already been mentioned include poultry, meat products generally, eggs and egg products, milk and shellfish.

Records show that, in most food poisoning outbreaks, the incriminated food vehicle is not established or confirmed, and a review of data from the surveillance of foodborne disease in Scotland confirms this. Table 2.4.6 summarises this data for the years 1980-90 when, during this period, investigations failed to confirm the incriminated food in 60% of outbreaks.
Table 2.4.6  Outbreaks of Food Poisoning where the implicated food was established: Scotland 1980-1990

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No. Outbreaks Recorded</td>
<td>147</td>
<td>238</td>
<td>276</td>
<td>287</td>
<td>256</td>
<td>185</td>
<td>184</td>
<td>234</td>
<td>219</td>
<td>196</td>
<td>175</td>
</tr>
<tr>
<td>No. Outbreaks where food vehicle confirmed</td>
<td>74</td>
<td>121</td>
<td>117</td>
<td>99</td>
<td>109</td>
<td>69</td>
<td>63</td>
<td>81</td>
<td>97</td>
<td>66</td>
<td>48</td>
</tr>
</tbody>
</table>

Summary: Total: 1980-1990: 2388
Food Vehicle confirmed: 944 (39.5%)

Source: Sharp et al, 1981-92

For England during the years 1986-89, information about suspected food vehicles was given in 24% of total outbreaks, but this varied greatly with the type of aetiological agent (WHO, 1992).

For example, a suspect food was recorded for only 15% of *Salmonella* outbreaks, compared with 81% for *Cl. perfringens*, 100% for *S. aureus* outbreaks and 90% for outbreaks caused by *Bacillus sps*. It has been suggested that the low proportion of *Salmonella* outbreaks for which a suspect food vehicle is mentioned is a result of the large number of family outbreaks of salmonellosis recorded, about which little information is given. Again, it is the larger community or point-source (e.g. hotel function) outbreaks that are more likely to be reported.

In the UK, poultry (particularly chicken) is clearly the most common food implicated in food poisoning outbreaks, followed by other meats and meat products such as beef, pork, ham, meat pies, stews etc.
Several other foods are recorded, including eggs, milk and 'other/mixed' foods, but these are of much lesser consequence when compared to total numbers of recorded outbreaks involving poultry and other meat products (WHO, 1992).

The WHO report (1992), however, reflects a very different situation with regard to implicated foods when individual countries are compared to the UK. For example:

A. **SPAIN**: Egg/Mayonnaise was recorded in 40% of all reported outbreaks with 'meat' being implicated in only 3%. Interestingly, there is no reference to poultry as an implicated food in outbreaks during 1985/89, which, can only be put down to a failure on the part of the Spanish Health Authorities to carry out full epidemiological and microbiological investigations into outbreaks. The failure to record poultry specifically as a vehicle of infection is very surprising given *Salmonella* carriage rates in poultry generally, and Spain's very warm climate.

B. **FRANCE**: France, like Spain, also records eggs as the most frequently implicated food in outbreaks during 1988/89 accounting for 160 (32%) of all outbreaks reported. This is repeated in the latest report for 1991 (Anon(B), 1993), with eggs and egg products (e.g. mayonnaise, mousse etc.) being the vehicle of infection in 121 (31.5%) of the 384 outbreaks investigated. Other foods implicated included meat and poultry 62 (16%), milk and milk products 22 (6%) and fish/seafood 21(6%). (This is in contrast to Scotland where, in 1991, in 109 (73%) of the 150 outbreaks reported that year a food vehicle was not identified) (Sharp, Reilly, 1993). In a further 79 (21%) outbreaks, no specific food item could be determined.
C. **DENMARK:** Denmark records meat and meat products as being implicated in 37 (20%) recorded outbreaks, followed closely by fish and shellfish 31 (17%), with poultry being implicated in only 14 outbreaks (7%). This gives a combined total for all meat products (including poultry) accounted for 51 (27%) of all outbreaks.

D. **NETHERLANDS:** Statistics from The Netherlands reported 'Chinese foods' as being implicated in 29.7% of all outbreaks where a food vehicle was recorded during 1985/89. This may reflect particular preference for these food in Holland, and within the classification of "Chinese foods", it is probable that chicken, rice and eggs were the foods most likely to have caused illness. The exceptionally high proportion (18%) of all known outbreaks where *B. cereus* was recorded as the causative agent, (second only to Salmonella-30%), confirms the significance of Chinese food in foodborne disease in Holland, as rice is the food most often implicated in *B. cereus* food poisoning incidents.

E. **CANADA:** Reports from Canada are similar to the UK figures in that meat and poultry are the foods most often associated with reported outbreaks. For example, for the years 1984/86, of a total of 2,385 outbreaks where a food vehicle was recorded, 'meat' was responsible for 537 (22.5%), and poultry 340 (14.2%). These were followed by bakery foods 254 (10.6%), and dairy foods (7.75%).
F. UNITED STATES: Figures for the US are not very reliable and cannot reflect the true incidence of foodborne disease in the US. This is indicated by the fact that, for the years 1984-86, there were only 25 recorded outbreaks where poultry (chicken & turkey) was implicated as a vehicle of infection, with a further 15 implicating dairy foods. It is hoped that, with improved surveillance, more reliable data will be available in the future.

Discussion

As foods are often disposed of before investigations can begin, it is very difficult to obtain a microbiological confirmation that a particular food vehicle was responsible for illness. Investigators for the most part must rely on epidemiological data collected during enquiries and interviews before a suspected food vehicle can be identified.

The suggestion of retaining samples of the main food items for 3 to 5 days or so after meals is a very good idea but, perhaps not practical in the day to day affairs of a busy restaurant, given the demands of time, storage space, labour etc.

For institutional catering and for 'once-off' catering functions, particularly like those carried on in marquees, social clubs etc., where facilities and staff training in hygiene might not be of the required level, it is advisable that, for example, a sample dinner be put aside (and refrigerated) for a few days to be available for analysis should an outbreak occur. This is presently not the norm but would be a very worthwhile proposal in certain catering situations. In the event of a massive food poisoning outbreak occurring, sample foods would be readily available for bacteriological examination at the outset of an investigation.
It has been confirmed that foods of animal origin (poultry, other meats, eggs, dairy produce etc.) are most often implicated in food poisoning incidents. These are 'high risk' foods, and any campaign undertaken to reduce or minimise the incidence of food poisoning must target these foods with the aim of eliminating, or at least reducing to acceptable levels, initial bacterial counts in the raw product.

Proper surveillance, together with adequate follow-up investigations in situations where food poisoning incidents are reported, will ultimately identify existing or emerging high risk foods. This was borne out in the identification of an association between increased human isolates of *S. enteritidis* PT4 and eating eggs and egg products in the UK in the latter half of the 1980s. Surveillance reports have also suggested an association between eating beef burgers and *E.coli* 0157 infection, and between listeriosis in vulnerable groups, and eating soft cheeses made from raw milk, and meat pate. What may be a very high risk food in one country (e.g. fish products in Japan), may not present the same degree of hazard in other countries, as eating patterns and cooking procedures vary.

What really matters is that high risk foods and emerging foodborne pathogens are clearly identified so that preventative measures can be put in place to deal with any potential foodborne hazard that may arise from time to time.

2.4.3. Places where Outbreaks occur

The place where a food poisoning incident occurs refers normally to the place where the food is prepared or mishandled, and where it is eaten. However, with the huge increase in fast food outlets in recent years, foods may not be eaten in the place of preparation.
The same applies, for example, to picnic foods, and to airline foods where there may be a significant time interval (and distance) between initial preparation and final consumption of the food.

Food poisoning outbreaks are more normally associated in the public eye with large-scale catering, as occurs in hotels, restaurants, institutions etc. However, statistics for the UK generally disprove this common perception, and again using the Scottish example (Table 2.4.7), it can be seen that for the 11-year period 1980-90, 73% of recorded outbreaks of foodborne illness were in private households, with the remaining 27% being described as 'general outbreaks' (i.e. outbreaks affecting two or more persons not confined to a private household).

It is important to note however, that the actual number of persons involved (cases) is much greater in general outbreaks, (e.g. involving hotels, restaurants, institutions etc.) where common foods are eaten by a much greater number of people and normally at one 'sitting'. It is for this reason, that 'general outbreaks' are more likely to attract greater publicity. They, of course, also differ from household outbreaks in that illness normally results from the carelessness of other people, whereas in the case of household outbreaks, illness is acquired through the failure to observe some of the basic principles of food hygiene (e.g. undercooking poultry etc.) by members of one's own household.
Table 2.4.7: Place of Outbreak of Food Poisoning - Scotland 1980-1990

<table>
<thead>
<tr>
<th>YEARS</th>
<th>TOTAL</th>
<th>HOUSEHOLD</th>
<th>GENERAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980-90</td>
<td>2388</td>
<td>1738 (73%)</td>
<td>650 (27%)</td>
</tr>
<tr>
<td>1980-90</td>
<td>17,146</td>
<td>4577 (26.6%)</td>
<td>12,569 (73.4%)</td>
</tr>
</tbody>
</table>

Source: Communicable Disease (Scotland) Unit. Surveillance Programme for foodborne infections and intoxications. Adapted from individual Annual Reports 1980-90

The WHO report on foodborne infectious diseases in Europe (WHO, 1992) does not give any breakdown between household and general outbreaks for England & Wales, but another report (Anon, 1988) gives this information for the years 1984 and 1985 (Table 2.4.8).

These statistics confirm the Scottish figures as, during 1984 and 1985, there were more household outbreaks than general outbreaks, although the breakdown was not as significant as for Scotland (1980-90). (Total no. of outbreaks = 1,102 - Household: 57%; General: 43%).

However, when one examines closely the breakdown of the general outbreaks, and compares this with outbreak data for private households, it is clear that the home is by far the single most frequently recorded location where illness is actually acquired. Table 2.4.8 gives this breakdown for 1985 by aetiological agent (with 1984 figures in brackets).
Table 2.4.8  Food Poisoning Outbreaks - Reported by MOEH, EHOs and Laboratories by Place of Outbreak and Aetiological Agent England and Wales 1985. (1984)

<table>
<thead>
<tr>
<th>Location of outbreak</th>
<th>Salmonella sp.</th>
<th>Cl. perfringens</th>
<th>S. aureus</th>
<th>B. cereus &amp; B. Spp</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Private homes</td>
<td>248(358)</td>
<td>6(4)</td>
<td>2(2)</td>
<td>3(3)</td>
<td>259(367)</td>
</tr>
<tr>
<td>Rests/Receptions</td>
<td>38(53)</td>
<td>17(22)</td>
<td>2(3)</td>
<td>7(16)</td>
<td>64(94)</td>
</tr>
<tr>
<td>Hospitals</td>
<td>17 (20)</td>
<td>17(17)</td>
<td>1(-)</td>
<td>1(-)</td>
<td>36(37)</td>
</tr>
<tr>
<td>Institutions</td>
<td>3(5)</td>
<td>12(8)</td>
<td>2(-)</td>
<td>2(-)</td>
<td>19(13)</td>
</tr>
<tr>
<td>Schools</td>
<td>2(6)</td>
<td>4(1)</td>
<td>2(-)</td>
<td>(-)</td>
<td>8(7)</td>
</tr>
<tr>
<td>Shops</td>
<td>4(10)</td>
<td>(-)</td>
<td>1(-)</td>
<td>(-)</td>
<td>5(10)</td>
</tr>
<tr>
<td>Canteens</td>
<td>6(2)</td>
<td>3(10)</td>
<td>-(1)</td>
<td>1(3)</td>
<td>10(16)</td>
</tr>
<tr>
<td>Farms</td>
<td>3(9)</td>
<td>(-)</td>
<td>(-)</td>
<td>(-)</td>
<td>3(9)</td>
</tr>
<tr>
<td>Infected Abroad</td>
<td>9(12)</td>
<td>-(1)</td>
<td>(-)</td>
<td>(-)</td>
<td>9(14)</td>
</tr>
<tr>
<td>Other</td>
<td>21(18)</td>
<td>2(-)</td>
<td>1(1)</td>
<td>-(1)</td>
<td>24(20)</td>
</tr>
<tr>
<td>Unspecified</td>
<td>21(47)</td>
<td>3(5)</td>
<td>1(-0)</td>
<td>1(-)</td>
<td>26(52)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>372(540)</td>
<td>64(68)</td>
<td>12(7)</td>
<td>15(24)</td>
<td>463(639)</td>
</tr>
</tbody>
</table>

Note: Salmonella sps were identified as the aetiological agent in 912 (83%) of all outbreaks.

In contrast to this data for the UK, an analysis of places of outbreaks in other countries reveals a different trend. For example, statistics for The Netherlands show that only 10% of all known outbreaks were acquired in private homes, with restaurants and cafes being responsible for the vast majority (81%) (WHO, 1992). The French figures show that during 1985-89, schools were the single most frequently recorded place of outbreak 208 (29%), followed by households 165 (23%) and restaurants 128 (18%).
The latest report for France (1991), (Anon(B), 1993), however, follows the UK trend, with private homes being the place most frequently recorded with 144 (22%), although the vast majority of cases (2,665) were recorded in the 61 (9%) outbreaks that occurred in school canteens.

There is no specific breakdown available for the places of outbreaks in West Germany but it is stated in the 1992 WHO report that ‘central kitchens in hospitals, homes for the elderly and similar establishments were found as a source of a number of foodborne disease outbreaks, with restaurants being another place where outbreaks had their origin’.

The Spanish figures would again be more in line with those in the UK, with almost half (48.2%) of all recorded outbreaks during 1985/89 originating in private homes, followed by restaurants and ‘bars’.

The Canadian report (Todd, 1991) gives a breakdown of data for ‘places where food was mishandled by specific aetiology’. For 1984 and 1985 this report confirms food service establishments as the places where most mishandling took place with 1,000 (7%) incidents, followed by ‘homes’ with 346 (3%) incidents. (These figures include all incidents, including chemical, plant, microbiological etc, as well as incidents where an aetiology was not established, which was the case with the vast majority of incidents).

A further breakdown of food service establishments reveals that hotels and restaurants are the locations where most recorded mishandling occurs. Interestingly enough, almost the same number of ‘mishandling incidents were recorded for ‘homes’ and ‘hotels/restaurants’, but when one includes all the incidents recorded in all food service establishments, (e.g. fast-food outlets, clubs, institutions, canteens etc.) the total figures for food service establishment outnumbers those for ‘homes’ by almost 3 to 1.
Discussion

The true figures for food poisoning incidents recorded at private homes is very difficult to estimate, but appears to be much higher than is generally known. Further, family outbreaks are less likely to be reported, as they involve smaller numbers of people than outbreaks occurring in catering/point-source establishments and, as already mentioned, those responsible for household outbreaks involve members of one's own family. 'General' outbreaks account for the greatest number of cases, and while fewer in number than 'household' outbreaks, the potential hazard to public health is much greater given the vast numbers of people served in catering establishments. Further, a person infected with *Salmonella sps.* or *Hepatitis A* virus (HAV), for example, working in a large restaurant has much greater implications for public health than someone who only prepares food for the immediate family.

Outbreaks of food poisoning in private homes and catering establishments very often result from a failure by food preparation personnel to adhere to some of the very basic principles of food hygiene. As many domestic kitchens are small, with limited storage space, it is probable that cross-contamination from raw food is a common contributory factor to food poisoning incidents in the home.

However, it is likely that the undercooking of foods (especially meats) and allowing these to be stored for prolonged periods at room temperature, and by inadequate reheating are just other important contributory factors - See following discussion.
It is important that educational or food hygiene awareness programmes should be geared towards the home - where most recorded outbreaks of food poisoning occur. However, it is also important that each country should establish for itself the places where most food poisoning incidents occur, so that the maximum resources can be directed to areas that present the highest risk. Finally, it is vital for each country to have an adequately funded food poisoning surveillance programme and, as far as Ireland is concerned, it is not necessary to look any further than the Scottish example as a model to follow and to hopefully emulate.

2.4.4. Main Contributory Factors To Outbreaks of Food Poisoning

This aspect of the epidemiology of foodborne disease is very important if we wish to get a thorough insight into how food poisoning incidents occur. Where possible, all the contributory factors should be recorded when completing investigations into outbreaks of food poisoning. During such investigations, Environmental Health Officers very often have to rely on the memory of catering staff to recall cooking operations, food storage arrangements etc. on a particular night.

This is much easier in instances where the actual causative microorganism has a short incubation period (e.g. *S. aureus, B. cereus*) due to early receipt of notifications by Health Boards. Where microorganisms with long incubation periods are implicated (e.g. *Campylobacter* and *Hepatitis A*) precise recall of events may not be very clear. Further, the retrieval of leftover foods, while quite possible in the former case, is highly unlikely with illnesses that have a long incubation period. The ideal outcome to any investigation into a food poisoning outbreak is to match the causative microorganism (incl. the phagetype) in the incriminated food with that identified in clinical specimens.
This allows the EHO to determine how an outbreak may have occurred, and what factors were likely to have contributed to its occurrence. However, all too often the implicated food and causative agents are not confirmed, and one has to rely on epidemiological data collected during investigations to establish the possible contributory factors to an outbreak.

Contributory factors to food poisoning outbreaks have been recorded by a few people, and when these are closely examined it is very clear that food poisoning most often results from a failure on the part of food preparation personnel to adhere to the very basic principles of food hygiene. Data relating to the main contributory factors to food poisoning outbreaks is of vital importance to public health departments as educational programmes can be geared towards highlighting these factors, including explanations on the chain of events that often lead to foodborne illness. The main contributory factors recorded are outlined in Tables 2.4.9-2.4.11.
Table 2.4.9  Factors Contributing to 566 Outbreaks of Salmonella Food Poisoning in England & Wales 1970-82

<table>
<thead>
<tr>
<th>Contributing Factor</th>
<th>Number of Outbreaks in which factors recorded (%)</th>
<th>1970-79 396 outbreaks</th>
<th>1980-82 170 outbreaks</th>
<th>Total 566 outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparation too far in advance of needs</td>
<td></td>
<td>173 (44)</td>
<td>67 (39)</td>
<td>240 (42)</td>
</tr>
<tr>
<td>Storage at ambient temperature (Room)</td>
<td></td>
<td>115 (29)</td>
<td>57 (34)</td>
<td>172 (30)</td>
</tr>
<tr>
<td>Undercooking</td>
<td></td>
<td>91 (23)</td>
<td>48 (28)</td>
<td>139 (25)</td>
</tr>
<tr>
<td>Inadequate cooling/Reheating</td>
<td></td>
<td>71 (18)</td>
<td>54 (32)</td>
<td>125 (22)</td>
</tr>
<tr>
<td>Contaminated processed food (excluding canned)</td>
<td></td>
<td>91 (23)</td>
<td>19 (11)</td>
<td>110 (19)</td>
</tr>
<tr>
<td>Raw Food consumed</td>
<td></td>
<td>55 (14)</td>
<td>29 (17)</td>
<td>84 (15)</td>
</tr>
<tr>
<td>Cross-contamination</td>
<td></td>
<td>57 (14)</td>
<td>27 (16)</td>
<td>84 (15)</td>
</tr>
<tr>
<td>Inadequate reheating</td>
<td></td>
<td>47 (12)</td>
<td>29 (17)</td>
<td>76 (13)</td>
</tr>
<tr>
<td>Inadequate thawing</td>
<td></td>
<td>42 (11)</td>
<td>19 (11)</td>
<td>61 (11)</td>
</tr>
<tr>
<td>Extra large quantities prepared</td>
<td></td>
<td>12 (3)</td>
<td>17 (10)</td>
<td>29 (5)</td>
</tr>
<tr>
<td>Use of leftovers</td>
<td></td>
<td>18 (5)</td>
<td>7 (4)</td>
<td>25 (4)</td>
</tr>
<tr>
<td>Improper warm holding</td>
<td></td>
<td>11 (3)</td>
<td>4 (2)</td>
<td>15 (3)</td>
</tr>
<tr>
<td>Infected food handlers</td>
<td></td>
<td>9 (2)</td>
<td>4 (2)</td>
<td>13 (2)</td>
</tr>
<tr>
<td>Contaminated canned foods</td>
<td></td>
<td>2 (0.5)</td>
<td>0</td>
<td>2 (0.4)</td>
</tr>
</tbody>
</table>

Table 2.4.10  Factors contributing to 660 outbreaks of foodborne disease in food service establishments - United States 1973-82

<table>
<thead>
<tr>
<th>Contributory factor</th>
<th>Number</th>
<th>Percent (a) *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improper cooling</td>
<td>366</td>
<td>55.8</td>
</tr>
<tr>
<td>Lapse of 12 or more hours between preparing and eating</td>
<td>203</td>
<td>30.8</td>
</tr>
<tr>
<td>Colonised person handles implicated food</td>
<td>150</td>
<td>24.2</td>
</tr>
<tr>
<td>Inadequate reheating</td>
<td>130</td>
<td>19.7</td>
</tr>
<tr>
<td>Improper hot holding</td>
<td>107</td>
<td>16.2</td>
</tr>
<tr>
<td>Contaminated raw food/ingredient</td>
<td>58</td>
<td>8.8</td>
</tr>
<tr>
<td>Obtaining food from unsafe source</td>
<td>42</td>
<td>6.4</td>
</tr>
<tr>
<td>Improper cleaning of equipment/utensils</td>
<td>38</td>
<td>5.8</td>
</tr>
<tr>
<td>Cross contamination</td>
<td>31</td>
<td>4.7</td>
</tr>
<tr>
<td>Use of left overs (b)*</td>
<td>31</td>
<td>4.7</td>
</tr>
<tr>
<td>Inadequate cooking</td>
<td>29</td>
<td>4.4</td>
</tr>
<tr>
<td>Toxic containers/pipelines</td>
<td>23</td>
<td>3.5</td>
</tr>
<tr>
<td>Intentional additives (e.g. MSG)</td>
<td>13</td>
<td>2.0</td>
</tr>
<tr>
<td>Inadequate/improper thawing</td>
<td>6</td>
<td>0.9</td>
</tr>
<tr>
<td>Contaminated water</td>
<td>2</td>
<td>0.3</td>
</tr>
<tr>
<td>Improper dish washing/contamination afterwards</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>Mistaken for food</td>
<td>1</td>
<td>0.2</td>
</tr>
<tr>
<td>Incidental additives</td>
<td>9</td>
<td>1.4</td>
</tr>
</tbody>
</table>

**Source:** (Bryan FL, 1981)

*a)  % exceeds 100 because multiple factors contribute to single outbreaks.

*b)  also lapse of 12 or more hours.
Table 2.4.11 Factors Contributing to 331 outbreaks of foodborne Salmonellosis - United States 1961-82

<table>
<thead>
<tr>
<th>Contributory Factor</th>
<th>Number</th>
<th>Percent (a)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Factors affecting contamination (b)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contaminated raw products</td>
<td>103</td>
<td>31.1</td>
</tr>
<tr>
<td>Cross contamination (c)</td>
<td>65</td>
<td>19.6</td>
</tr>
<tr>
<td>Colonised person handled implicated foods (d)</td>
<td>47</td>
<td>14.2</td>
</tr>
<tr>
<td>Improper cleaning of equipment/utensils</td>
<td>42</td>
<td>12.7</td>
</tr>
<tr>
<td>Contaminated water</td>
<td>2</td>
<td>0.6</td>
</tr>
<tr>
<td><strong>Factors affecting survival</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inadequate heating (cooking heat processing)</td>
<td>79</td>
<td>23.9</td>
</tr>
<tr>
<td>Inadequate reheating</td>
<td>38</td>
<td>11.5</td>
</tr>
<tr>
<td><strong>Factors affecting growth</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Improper cooling</td>
<td>144</td>
<td>42.5</td>
</tr>
<tr>
<td>Lapse of 12 or more hours between preparing and eating</td>
<td>63</td>
<td>19.0</td>
</tr>
<tr>
<td>Improper hot holding (e)</td>
<td>42</td>
<td>12.7</td>
</tr>
<tr>
<td>Use of leftovers (f)</td>
<td>13</td>
<td>3.9</td>
</tr>
<tr>
<td>Improper fermentations</td>
<td>2</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Source: (Bryan FL, 1988).

a) Percentage exceeds 100 because multiple factors contribute to single outbreak.

b) Raw produce of animal origin often the source of contamination.

c) From raw to cooked foods via hands, equipment utensils, cleaning cloths/sponges/towels to foods not subjected to further cooking.

d) Salmonella isolated from one or more food handlers during the investigation (unknown whether source or victim) or history of food handler touching food incriminated as the vehicle. Includes leaving foods at room or outside ambient temperature for several hours.

e) Holding foods at bacterial incubation temperatures for several hours.

f) Also 12 or more hours.
The above 3 tables outline in detail the major contributory factors to several outbreaks of foodborne illness, particularly salmonellosis, in both the US and England & Wales. A more direct comparison can be made between Tables 2.4.9 and 2.4.11 as both these refer specifically to outbreaks caused by *Salmonella sps.* However, as *Salmonella sps.* account for the vast majority of food poisoning outbreaks, it is probable that most of the outbreaks referred to in Table 2.4.10 were also caused by these pathogens.

**Discussion**

From a close scrutiny of all 3 tables, it is possible to comment on the significance of the main contributory factors, and to explain in practical terms how food poisoning incidents occur when fundamental food hygiene principles are not adhered to and observed. The main were factors were:-

1. **Improper/Inadequate Cooling of Foods**

   This relates to prolonged cooling of foods after cooking. This gives bacteria or, more particularly, their spores (e.g. *B. cereus*) that may have survived the cooking process, the opportunity to germinate and multiply. During the period of cooling, the temperature of the food passes through the 'danger zone' (10-50°C), with optimum growth at 37°C, enabling microorganisms to multiply at a fast rate, with toxin production in some cases. Improper or prolonged cooling of foods is very often a major contributory factor to outbreaks of food poisoning implicating beef stews and pies (*Cl. perfringens*), rice (*B. cereus*) and poultry (*Salmonella sps.*).
Rapid cooling of foods, followed by prompt refrigeration ensures that this contributory factor is eliminated. Improper cooling was recorded as a contributory factor in more than half of the 660 outbreaks referred to in Table 2.4.10.

2. Preparation of Food too far in advance of consumption (e.g. 12 hours or more).

As with prolonged cooling, this can also introduce a time factor that allows foodborne pathogens the opportunity to multiply on foods to such levels as constitute an infective dose (which can vary from person to person). Advanced cooking, even where initial cooking has been thorough, is potentially dangerous, particularly in situations where standards of personal and operational hygiene are not of the required level, or where refrigeration facilities are inadequate. Post-cooking contamination is likely to occur. Food Poisoning may then result from the consumption of such contaminated foods, either in their cold state (e.g. buffet foods), or following inadequate reheating.

Modern 'cook-chill' systems operate on the advanced cooking principle, with a recommendation that chilling be commenced as soon as possible after completion of cooking, and within 30 minutes of leaving the cooker. (There are specific recommendations for large joints). Such foods should then be cooled to between 0-3°C within a further period of 90 minutes.(FSAC(B), 1991).

Ideally, foods should be cooked as near to the time of service as possible. However, in large catering institutions, hotels, restaurants, etc., it is more practical to prepare foods well in advance, to refrigerate until required, and, if necessary, to reheat just before the service of meals. Caterers should be made aware of the inherent dangers associated with advanced cooking.
This is particularly important in situations where other potential contributory factors may also be present, such as inadequate refrigeration facilities, cross contamination, undercooking etc. This was the most important contributory factor recorded in England & Wales. (Table 2.4.9.)

3. Undercooking.

Heating foods to the correct core temperature so that foodborne pathogens are destroyed is one of the most fundamental principles of good food hygiene. However, this does not always happen, resulting in the survival of bacteria or their spores in the centre of the food (e.g. meat stew, rolled roast, turkey carcass) with subsequent multiplication of vegetative cells during prolonged cooling.

Another factor that goes hand in hand with 'undercooking' is the failure by foodworkers to defrost large joints of meat and poultry carcasses thoroughly, so that there is inadequate heat penetration to the centre of the joint/carcass. Microorganisms located in the centre survive the cooking process, with the result that meat served from near the centre of the joint/carcass may be a source of infection.

This was mentioned as a contributory factor in approximately a quarter of the outbreaks set out in Tables 2.4.9 and 2.4.11, although was much lower in the US study in Table 2.4.10.

Table 2.4.12 set out the core temperature/cooking time to destroy *Salmonella* and *Listeria sps.* in most foods. It can be seen that the time necessary to destroy these pathogens at the centre of foods is directly related to the amount of heat penetration.
4. Storage at Ambient Temperature

Most food poisoning bacteria multiply rapidly at ambient temperatures, particularly those temperatures that one would expect to find in a busy commercial kitchen. In fact, the optimum growth temperatures for these bacteria (34°C-40°C) has been recorded by this writer in hotel kitchens immediately adjacent to the cooking equipment, with foods being left 'to cool' in this environment. This contributory factor is very often associated with 'prolonged cooling', and the main preventative measure should be to serve the food as soon as possible after cooking, or to cool rapidly and refrigerate until required.

While this contributory factor is only specifically mentioned in Table 2.4.9, its importance is obviously disguised under the data for 'improper cooling' (Tables 2.4.10 and 2.4.11).

### Table 2.4.12
Equivalent Core Cooking Time/Temperature for the destruction of Salmonella and Listeria sps.

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Time Taken to destroy microorganisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>60°C</td>
<td>45 minutes</td>
</tr>
<tr>
<td>65°C</td>
<td>10 minutes</td>
</tr>
<tr>
<td>70°C</td>
<td>2 minutes</td>
</tr>
<tr>
<td>75°C</td>
<td>5 minutes</td>
</tr>
<tr>
<td>80°C</td>
<td>6 seconds</td>
</tr>
</tbody>
</table>

5. Inadequate Reheating

Foods that have been cooked well in advance of needs must be thoroughly reheated and not just given a quick warming up before consumption. Problems arise where food has been contaminated after initial cooking or where bacteria survive the initial cooking process and where such foods are subsequently stored for long periods at room temperature.

Where toxin production has taken place in the interval, the reheating temperatures, while sufficiently high enough to destroy vegetative cells, very often does not inactivate bacterial toxins, resulting in classical food poisoning such as that caused by *B. cereus* and *S. aureus*. Foodworkers need to be educated in the chain of events leading up to toxin production in foods.

While only recorded in 11.5% of the outbreaks set out in Table 2.4.11 and 13% in Table 2.4.9, it was a significant contributory factor (19.7%) in the outbreaks referred to in Table 2.4.10.

6. Inadequate/Improper Warm Holding of Foods

While this is recorded as a contributory factor in only 3% of outbreaks in the studies summarised in Table 2.4.9, Tables 2.4.10 and 2.4.11 record it in 16.2% and 12.7% respectively of outbreaks.

Warm holding of foods at the proper temperature (e.g. over 63°C) is very important. Vulnerable foods (e.g. poultry, other meats, stews etc.) should be stored or displayed under chilled conditions (below 5°C) or, if required hot, at temperatures above 63°C.
Where warm holding equipment is faulty, or where the doors are not fully closed, the temperature could fall close to the optimum temperature for bacterial growth. In situations where chickens are undercooked, or where cross contamination takes place from raw to cooked poultry carcasses, warm holding units can act as incubators for the growth and multiplication of bacteria, including pathogens such as Salmonella.

The use of warm holding equipment has grown very much in popularity in recent years, particularly in the retail and fast food catering sectors. Cooked chicken display units in particular have become very popular and are now almost standard equipment in retail butchers shops. This latter development is of concern given that retail butchers deal primarily in raw meat, with a consequent greater potential for cross contamination. It is very important, therefore, that butchers and their staff understand the principle of cross contamination, with a realisation that warm holding units can have serious implications for public health if not operated at the correct temperature.

7. Contaminated Food Handlers (Excretors/carriers etc.)

There was a large difference in the number of occasions foodworkers were implicated as a contributory factor in all three studies, ranging from a very low 2% in the UK study (Table 2.4.9) to a high of 24.2% in the US study (Table 2.4.10). This difference may be partly explained by the fact that Table 2.4.9 summaries the contributory factors to 'foodborne' outbreaks (i.e. all types), whereas Table 2.4.10 refers specifically to outbreaks caused by Salmonella sps. However, as Salmonella sps. account for the vast majority of food poisoning outbreaks, it is difficult to explain why there should be such a difference. The true explanation may lie in the extent and methods of collection and collation of epidemiological data in all three studies.
Nevertheless, it is well recognised that food handlers can be a source of infection, with the carrier state for *Salmonella sps.* being well recognised. Also, with the infective dose for human gastroenteritis viruses thought to be as low as one viable particle (Collins, 1994), it is vital that the highest standards in personal hygiene are observed at all times during food preparation.

Personal hygiene is of paramount importance therefore as, no matter how well designed or well equipped a food premises is, it only takes one person (excreter/carrier) engaging in unhygienic food handling practices to undo all the good work and high standards of personal hygiene practised by all other members of staff.

In Ireland, as in the UK, there is a legal responsibility on food workers to keep themselves clean and not to engage in any unhygienic practice in close proximity to food (Food Hygiene Regulations, 1950).

There is also a requirement on foodworkers to report gastrointestinal symptoms to management and the importance of doing this cannot be overestated. Such foodworkers should be excluded from working with food until cleared by a doctor, as they may be carriers of an infectious agent. Again, the role of hygiene education is very important if foodworkers are to get a clear understanding of the potential dangers associated with the overhandling of food etc. However, it is extremely difficult to reach out to all food personnel, given the fast turnover of staff in this industry. It is up to management in particular to be aware of their responsibilities and they should always be on the lookout for obvious or emerging unhygienic practices so that these can be arrested at an early stage.
8. Cross-Contamination

This is regarded as an important contributory factor in food poisoning outbreaks, as can be seen from the data in Table 2.4.11. However, interestingly enough, this was not borne out in the other two studies. In many catering situations, raw and cooked foods are very often handled and stored in close proximity increasing the opportunity for cross-contamination.

Poultry are a major reservoir for pathogens (including *Salmonella*, *Campylobacter*, *Listeria* and *Cl. perfringens*), and a source of cross-contamination, either by direct contact with other foods, or by carelessness in the handling and preparation of raw poultry products. Everyday examples of this include

(a) The storage of raw poultry directly above ready-to-eat foods in a cold room.

(b) Foodworkers directly handling cooked foods after handling raw poultry without thoroughly washing their hands beforehand.

(c) Putting cooked foods (e.g. chickens) directly onto a surface, which had been used to hold raw poultry previously - without adequate cleaning in the interval.

(d) Using the same knife for cutting or quartering raw chicken and cutting cooked meats (e.g. for making sandwiches) - without first thoroughly sterilising the knife.
There was a considerable variation in the number of occasions cross-contamination was recorded as a contributory factor, ranging from 4.7% (Table 2.4.10) to 19.6% (Table 2.4.11). However, as regards the two studies dealing specifically with outbreaks caused by *Salmonella sps.*, there was closer proximity in the recording of cross-contamination, with the UK study recording it as a factor in 15% of outbreaks and, as stated above, the US study recording it in 19.6% of outbreaks.

It is very important that there is clear epidemiological data available on the important contributory factors to food poisoning outbreaks. This information (for educational/control purposes) is vital to both the catering industry and public health authorities in their efforts to reduce the incidence of food poisoning. Further, hygiene education courses will benefit in that greater time and effort can be focused on the main factors that contribute to incidents of foodborne disease.

**Discussion**

This chapter has discussed the main contributory factors to several recorded food poisoning outbreaks in both the US and in England and Wales. It is obvious that many of these outbreaks could have been avoided if foodworkers had a basic understanding of some of the fundamental principles of food hygiene.

Inherent in this must be an understanding by foodworkers of the basic principles of microbiology, with reservoirs of infection, growth requirements of bacteria, the concept of cross-contamination, and the importance of personal hygiene being given priority.
Indeed, food poisoning incidents can be greatly reduced if four simple concepts are understood by foodworkers, i.e.

1) That raw foods, particularly those of animal origin, are likely to harbour pathogens, and therefore, should be treated/handled as such.

2) That thorough cooking/heat treatment throughout a food product will normally kill food poisoning bacteria (i.e. vegetative cells, not necessarily toxins and spores).

3) That foods should ideally be cooked as near as is possible to the time of service/consumption.

4) That all vulnerable foods (especially meat products) should be kept refrigerated ($<4^\circ\text{C}$) unless immediately required for use.

2.5 Summary

This thesis so far has highlighted the principal microorganisms in foodborne disease and their relative importance in the light of the most up-to-date surveillance information available at the time of writing. It has also looked at 'new' or emerging pathogens and the role that they are likely to play in the years ahead. However, exploring the microbiology of foodborne disease was insufficient in itself and it was, therefore, necessary to look more closely at:-

(a) The most frequently implicated foods in recorded outbreaks.

(b) The places where outbreaks most often occurred.
The major contributory factors as recorded in 3 important studies.

As stated at the outset, the epidemiological data that is collated and analysed to provide invaluable information on foodborne disease is only as good as the extent of surveillance programmes in any particular country.

Where surveillance is inadequate, as is the case in Ireland, there is very little solid statistical information available to depict trends, and consequently to put in place effective preventative programmes and measures.

The UK data, however, may be the most applicable to the Irish situation as the cultural behaviour, climatic conditions and eating patterns are more closely allied to our own. In addition, the data available from the UK covers a broad area and is of an extremely high standard.

It is now necessary to examine the different systems for the surveillance of gastro-intestinal disease generally and, in this context, it is proposed to discuss available data on foodborne disease in Ireland, and to compare this with data from the UK. The well established surveillance systems in the UK will again be discussed and available trends in Ireland, such has they are, will be analysed against the background of the UK models (i.e. Scotland, England & Wales and Northern Ireland).
The Surveillance of Food Poisoning and Foodborne Intestinal Tract Infections in Ireland

3.1 Introduction

This thesis so far has been concerned with the surveillance of foodborne infectious diseases and intoxications in Europe and North America. There has also been discussion on the significance of the main gastro-intestinal tract infectious agents in the UK (i.e. *Campylobacter* and *Salmonella sps*), as these are thought to be mainly transmitted through the food chain. In this section, it is proposed to discuss surveillance of food poisoning and gastro-intestinal disease from an Irish perspective, although for comparison purposes, the UK models will again be used as a reference.

There is great variation in the extent of the surveillance of foodborne disease throughout the world, and in countries where a formal surveillance programme has not been established, data published is likely to be incomplete and unreliable. Such is the case in Eire. The Department of Health in its 1984 'Memorandum on the Organisation of an Investigation Programme on Food Poisoning' (DoH, 1984), requested that full epidemiological and microbiological data be returned to the department when investigations into outbreaks have been completed. However, the failure to establish a formal surveillance unit for foodborne disease to date, together with the absence of a national surveillance centre for communicable diseases generally, as has been recommended in the Hickey report (Anon(D), 1990), means that there is no clear overall picture on the true incidence of foodborne disease in Ireland.
In 1979, WHO recommended the establishment of a surveillance programme in Europe for foodborne infections and intoxications that incorporated an early warning system for incidents affecting more than one country and a routine reporting system to record details of outbreaks of foodborne disease. The initial meeting convened by WHO took place in Geneva and 13 countries were represented to discuss the proposed programme.

Scotland were the first country to formally contribute to this programme. By 1992, with the publication of the 5th report (WHO, 1992), a total of 31 countries were contributing. The extent of data varies considerably, ranging from the supply of statistical data on the number of cases only, to full epidemiological information on foodborne disease outbreaks. This 5th report is the most comprehensive published so far, although 4 of the 31 participating countries (incl. Ireland) provided little or no worthwhile data. Indeed, on page 90 of the report, which refers to the Irish contribution to the programme it is stated that:

"General information has been taken from the 4th report of the WHO programme. Unfortunately, the Berlin centre has not received any reply from the contact point on foodborne disease in Ireland for the period 1985-89"

The dearth of statistics in relation to food poisoning in Ireland has also been referred to by other commentators such as Donnelly (1986) who drew attention to the unreliability of statistics that exist when stating that “few conclusions can be drawn from such (Irish) unreliable figures. If statistics are not available on which to base our preventive health programme, how are we to detect trends and decide which areas should receive priority”. It is a view with which this writer would certainly concur.
3.2 The Notification of Infectious Diseases in Ireland

There are currently 33 diseases notifiable in Ireland under the Infectious Diseases Regulations 1981. (Appendix 1). While some of these diseases can potentially be foodborne (e.g. B. dysentery, brucellosis, gastroenteritis, typhoid, paratyphoid, hepatitis A), the two diseases listed that are specifically associated with foodborne transmission are:

1. Food Poisoning (bacterial other than salmonellosis).
2. Salmonellosis (other than typhoid and paratyphoid).

While cases of salmonellosis are most often reported by hospital clinicians as a result of routine laboratory screening via the Directors of Community Care to the Department of Health, food poisoning is usually reported by general practitioners, based on clinical diagnosis and without bacteriological confirmation.

Legislation relating to infectious diseases in this country is covered under 'The Infectious Diseases Regulations 1981. Article 14 of these regulations, which deals with notifications, puts an onus on medical practitioners to notify in writing the local Medical Officer of Health a case, as soon as he becomes aware, or suspects that a person on whom he is in professional attendance, is suffering from a scheduled infectious disease. Therefore, the real incidence of infectious diseases (particularly those that are foodborne) in this country depends on:-

1. A person firstly seeking medical assistance.

2. The motivation of medical practitioners to formally notify cases of infectious diseases to the local Medical Officer of Health (Director of Community Care).
While the underreporting of all types of infectious diseases throughout the world is well recognised, the degree of underreporting is difficult to assess, so that the true incidence of infectious disease can only be vaguely estimated. For example, studies in North America (Roberts et al., 1989) have suggested that, with regard to salmonellosis, only between 1/29.5 and 1/145 of cases are actually reported. This 'submerged morbidity' has potential economic consequences and, from a public health point of view, means that for any given time, and particularly during outbreaks of salmonellosis, there are unrecognised excretors of *Salmonella* microorganisms in the community which are sources for the transmission of further infection. The consequences of this are particularly serious where such persons are engaged in catering work, or are working with high risk groups, such as young children (e.g. in playschools), the sick (hospitals) and the elderly (nursing homes).

Not all people seek medical assistance when suffering from infectious diseases, particularly where illness is less severe and of short duration which may include food poisoning. However, the failure of general practitioners to notify those cases that come to their notice, is a major contributory factor to the lack of reliable information on the incidence of foodborne infectious diseases in this country.

Research into the notification of foodborne disease confirms that there is great underreporting in Co. Kerry, with as little as two or three G.P.s making formal notifications each year to the Director of Community Care. There are other examples of such obvious underreporting of food poisoning in other parts of Eire as reflected in the statistics for counties Galway, Mayo, and Roscommon for 1991. During that year, it appears that not even one case of food poisoning was formally notified to the Department of Health from these counties.
Concern about the underreporting of food poisoning in Co. Kerry has led to the submission of a report (internal) to the Director of Community Care with a view to improving the situation. It was suggested that:

(a) G.P.s be reminded of their duty under the Infectious Disease Regulations 1981 regarding the notification of food poisoning.

(b) Where a suspected case of food poisoning comes to the attention of a G.P., he/she should first notify the Director of Community Care by telephone at the earliest opportunity.

(c) Written/formal notification should follow at a later date.

Early notification will result in more complete investigations into incidents of food poisoning by Environmental Health Officers (EHOs), with the retrieval of the suspected incriminated food for microbiological examination being of paramount importance. However, such foods are unfortunately most often disposed of by the time investigations normally begin. The early submission of stool specimens for microbiological examination is also important and indeed vital to the successful isolation and identification of many pathogens such as viral particles (e.g. Norwalk agent) as peak shedding of such viruses normally occurs within 24-48 hours of onset of symptoms (Appleton, 1990).

In summary, it can be stated that the obvious underreporting of food poisoning in Eire is a matter of concern and needs to be urgently addressed.
3.3 The Incidence of Food Poisoning in Ireland

As previously stated, the notification of food poisoning in Ireland (and indeed the UK) is normally based on clinical diagnosis, with occasionally a diagnosis being supported by laboratory confirmation. Notifications of *Salmonella sps.*, on the other hand, represent confirmed laboratory isolates and are usually reported by the attending doctors in hospitals.

It is likely that many of the 'food poisoning' cases notified will also include cases of salmonellosis, given the significance of *Salmonella sps.* in foodborne disease. However, when reporting a case of “food poisoning” it is not mandatory to suggest or confirm the causative agent with only seriously ill cases being normally asked to submit clinical specimens for microbiological confirmation. Specific causative agents of ‘food poisoning’ such as *Campylobacter sps.*, *S. aureus*, *Ct. welchi* and *B. cereus* are therefore not recorded. However, depending on the extent of epidemiological data gathered during investigations, it is probable that an EHO will be in a position to suggest the most likely source of infection including the causative agent where the latter has not been identified.

All cases of infectious disease, including salmonellosis and food poisoning notified to Directors of Community Care in Eire are also required to be returned to the Department of Health on a weekly basis. It is important to note that these figures represent total numbers only for each infectious disease, as it is not necessary to enclose epidemiological data with each case reported. However, with regard to actual outbreaks of food poisoning, full epidemiological data concerning the outbreak is required to be returned to the Department of Health (DOH, 1984).
Data on the incidence of food poisoning in Ireland is best discussed in the context of the reported incidence in the UK. Table 3.3.1 compares totals (and rates per 100,000 pop.) of statutory notifications of food poisoning in the U.K and Eire during the period 1983-93. As can be seen, there was a great difference between the reported incidence in Eire and the UK and this difference is more clearly depicted in Figure 3.3.1

Table 3.3.1 Comparison of Statutory Notifications of Food Poisoning in the UK and The Republic of Ireland.

<table>
<thead>
<tr>
<th>Year</th>
<th>Northern Ireland</th>
<th>Republic of Ireland</th>
<th>Scotland</th>
<th>England &amp; Wales</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Notifs</td>
<td>Rate</td>
<td>Stat. Notifs.</td>
<td>Rate/100,000 pop.</td>
</tr>
<tr>
<td>1983</td>
<td>128</td>
<td>8.3</td>
<td>83</td>
<td>2.4</td>
</tr>
<tr>
<td>1984</td>
<td>144</td>
<td>9.3</td>
<td>164</td>
<td>4.7</td>
</tr>
<tr>
<td>1985</td>
<td>158</td>
<td>10.1</td>
<td>98</td>
<td>2.8</td>
</tr>
<tr>
<td>1986</td>
<td>273</td>
<td>17.4</td>
<td>195</td>
<td>5.6</td>
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<tr>
<td>1987</td>
<td>423</td>
<td>26.9</td>
<td>88</td>
<td>2.5</td>
</tr>
<tr>
<td>1988</td>
<td>302</td>
<td>19.1</td>
<td>43</td>
<td>1.2</td>
</tr>
<tr>
<td>1989</td>
<td>501</td>
<td>31.6</td>
<td>64</td>
<td>1.8</td>
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<tr>
<td>1990</td>
<td>819</td>
<td>51.5</td>
<td>157</td>
<td>4.5</td>
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<tr>
<td>1991</td>
<td>636</td>
<td>39.7</td>
<td>83</td>
<td>2.4</td>
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<td>1992</td>
<td>915</td>
<td>56.5</td>
<td>46</td>
<td>1.3</td>
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<tr>
<td>1993</td>
<td>954</td>
<td>58.5</td>
<td>97</td>
<td>2.8</td>
</tr>
</tbody>
</table>

Source: Rep. of Irl., Scotland, N. Ireland, England & Wales

Health Statistics 1983-93. Dept of Health, Dublin
The Scottish Centre for Infection and Environmental Health, Glasgow.
Dept. of Health & Social Services (DHSS), Regional Information Branch, Belfast.
Office of Population Censuses & Surveys (OPCS) London
Figure: 3.3.1
Comparison of StatutoryNotifications of Food Poisoning in the U.K. and Republic of Ireland

References:
The different rates of food poisoning reported within the UK is difficult to interpret, but may possibly be explained by different reporting systems, including differences in the extent of surveillance within each region. Certainly, the rates in Northern Ireland were for many years much lower than in the rest of the UK, with, for example, the Scottish rates for 1989 being double those for Northern Ireland, and figures for England & Wales even showing a greater disparity for the same year (2.4 times). However, this gap closed considerably in subsequent years, with rates for N. Irl. and Scotland now being particularly close (See 1993 data). It can be seen that up until 1989 the reported incidence was highest in Scotland, when, for the first time, the incidence in England & Wales was reported (and continues) to be higher. The comparatively low rates recorded in Northern Ireland during the 1980s was probably a consequence of less comprehensive reporting but, even at that, the rate here was much higher than statutory notifications recorded in Eire. A direct comparison of rates between North and South clearly illustrates this.

It is difficult to accept that there could be such a huge difference in the incidence of food poisoning between Northern and Southern Ireland. With only two exceptions (1988 and 1991) rates in N. Irl. showed a sustained increase throughout the period, with three years of the most recent four year period (1990-93) showing rates in excess of 50/100,000 pop. A similar increase in incidence was not recorded in the south however. Indeed, on only one occasion, (1986) did the rate exceed 5/100,000 pop. Rates for 1987 and 1988 were almost exactly the same as those recorded for 1991 and 1992 respectively. Data for recent years showed no upward trend with, in fact, the rate recorded for 1993 being identical to that of 1985.
Again, it is difficult to interpret the enormous difference in the reported incidence of food poisoning North and South of the border (e.g. by a factor of 21 for 1993). The similarity of culture, eating patterns and standard of food hygiene would suggest a much closer incidence in both regions. However, as this difference is probably more a consequence of gross underreporting in the South, with increased awareness, and greater motivation amongst clinicians to notify cases in the future, it is likely that this gap will decrease in the years ahead.

Efforts to increase awareness with regard to foodborne disease and food safety in general should be spearheaded by the proposed new Food Unit and Food Safety Board to be established within the Department of Health as part of its new National Health Strategy published in April 1994 (DoH, 1994). This unit should in the future liaise closely with the new Public Health Departments also being established within each Health Board. Further, the recommendation for a 'National Surveillance Programme of Foodborne Diseases' by the Food Safety Advisory Committee (FSAC(A), 1994) which will presumably be co-ordinated by this unit, will greatly facilitate the collection, collation, analysis and dissemination of epidemiological data with regard to foodborne disease in Southern Ireland. As a result of this, it is hoped that clinicians will be motivated to a greater extent to contribute to the notification process.
3.4 Salmonellosis

The collation and reporting of data on salmonellosis in Ireland needs to be explained and understood in the context of the overall surveillance of intestinal tract infections. Data on the incidence of salmonellosis in Eire is based on laboratory reports communicated to the Department of Health via the local Directors of Community Care. These represent the statutory notifications published by the Department but, as with reports of "food poisoning", probably bear little resemblance to the true incidence of infection in the country. Table 3.4.1 sets out the total number of confirmed cases reported to the Department of Health during the period 1983-93. As can be seen, the highest number of cases during this period (484) was reported in 1991, representing an incidence of 13.8 per 100,000 population. Figure 3.4.1 demonstrates that from the perspective of the data provided via this system of surveillance, the incidence of salmonellosis clearly peaked during the period 1989-91 but has since returned to its presumed base level.

Table 3.4.1 Statutory Notifications of Salmonellosis to the Department of Health in Eire 1983-93.

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<tbody>
<tr>
<td></td>
<td>205</td>
<td>287</td>
<td>142</td>
<td>265</td>
<td>249</td>
<td>271</td>
<td>427</td>
<td>471</td>
<td>484</td>
<td>270</td>
<td>295</td>
</tr>
</tbody>
</table>

However, it is important to realise that data on salmonellosis published by the Department of Health through this system represents total reports of *Salmonella* isolates only. There is no reference to the serotype breakdown, although it is probable that initial reports to the Department from DCC’s may include this information.
Figure 3.4.1
Statutory Notifications of Salmonellosis to the Department of Health in Eire 1983-1993
To obtain a more detailed breakdown of trends in relation to the distribution of Salmonella serotypes, it is necessary to examine the two regional voluntary systems, which include surveillance data on Salmonella spp. in their overall surveillance programme for intestinal tract infectious agents. However, before proceeding to discuss the surveillance of intestinal tract infections in Ireland more fully, it is perhaps important to place these limited Irish regional systems in context by describing briefly the more established ‘national’ systems existing in the UK.

3.5 Centres for the Surveillance of Enteropathogens in the UK

Surveillance of Salmonella spp. (and other enteropathogens) in the UK, is operated and controlled by national surveillance centres/units (gastro-intestinal disease sections). These Communicable Disease Surveillance Centres collect, collate and report laboratory confirmed human intestinal infections in an informal voluntary basis (i.e. non-statutory) - in contrast to notifiable infections, and depend on a network of laboratories and the co-operation of staff who take a special interest in the epidemiology of specific infections such as Salmonella spp., Campylobacter spp., E.coli 0157, Shigellas, Pathogenic Protozoa etc. There are three separate surveillance systems operating within the UK.
1. England & Wales

The Communicable Disease Surveillance Centre (CDSC) of the Public Health Laboratory Service (PHLS).

This centre collects data on laboratory isolates causing gastro-intestinal tract infectious diseases from 52 PHLS laboratories and approx. 300 National Health Service (NHS) hospitals, and a few private laboratories. In general, while NHS laboratories deal with clinical specimens, the PHLS laboratories deal with specimens of public health significance (e.g. specimens resulting from screening, or illness in the community, or from the routine microbiological examination of food, water, milk). Almost all the 52 PHLS laboratories are located on the same sites as the major NHS hospital laboratories. The centre was established in 1977 (Cowden JM, CDSC,- personal communication).

This centre is supported by a national/international centre for the detailed identification of Salmonella sps., E. coli and other bacterial gastro-intestinal tract pathogens (i.e. The Division of Enteric Pathogens of the Central Public Health Laboratory). Further, the Royal College of G.P.s runs a sentinel practice scheme for a wide range of diseases, including infectious intestinal disease. The latter involves 60 G.P.s (425,000 patients approx.) who report on a weekly basis clinically diagnosed cases of various infections. (Anon(C), 1990).
2. Scotland

The Communicable Disease (Scotland) Unit - CD(S)U

This was established in 1969 and, together with the CDSC of the PHLS, comprises one of the most comprehensive and effective surveillance systems in the world. Since 1989, this laboratory reporting system has been strengthened by the identification of 30 formally designated reportable infections including bacterial meningitis, brucellosis, salmonellosis, botulism etc. (Appendix 2). These are separate and distinct from scheduled infectious diseases which must be formally (statutorily) notified.

As in England & Wales, many of the voluntary reports of intestinal infectious diseases may be food borne (e.g. salmonellosis, campylobacteriosis) but, unlike food poisoning, a mode of transmission/vehicle of infection does not have to be identified (many gastro-intestinal tract infections are spread directly from person to person). With this system, the identification of an isolate is the starting point for surveillance purposes (Sharp, CD(S)U - personal communication).

3. Northern Ireland

The Department of Health and Social Services (DHSS)

This system is equivalent to the systems in Scotland and England and Wales, and voluntary reports are received from 18 hospital laboratories. However, like England & Wales, but unlike Scotland, there is no formal list of reportable human infections (Dr. Mitchell, DHSS - personal communication).
A feature of all 3 surveillance systems for enteropathogens is the publication of weekly (Scotland, England & Wales) and monthly (N.Irl.) communicable disease reports on laboratory identified infectious diseases, which are distributed free of charge to interested parties in the field of community medicine, medical microbiology, and environmental health etc.

3.6 Surveillance Systems for Intestinal Tract Infections in Ireland

The preceding paragraphs gave a brief outline of the extent of gastro-intestinal disease surveillance in the UK, and data emerging from our own limited systems of surveillance need to be considered in this context.

The fact remains that there is no National Disease Surveillance centre in Ireland and the need for such a collation unit has been addressed in the Hickey report (Anon(D), 1990). The expert Committee, (chaired by Mr. Kieran Hickey, C.E.O., E.H.B.,) which published this report was established in 1988 and was asked 'to define the role of community medicine in the long term' (in Ireland). The report of this working party made many recommendations, including a recommendation for the establishment of a national disease surveillance unit to be given priority. While accepting that a small unit would be sufficient for surveillance purposes in Eire, the group suggested that the surveillance unit might confine its activities to communicable diseases in the first instance, but recommended an extension of its surveillance to include non communicable diseases as resources would permit.
The establishment of such a unit in Ireland would have enormous benefits for the public health service as we would be in a position to:-

(a) Detect our own specific changes in disease patterns.

(b) Evaluate disease control, enabling our health authorities to take early preventative action whenever necessary.

(c) Provide invaluable data and information to the Department of Health for better planning of the health services.

Hickey further recommends that "this surveillance unit should not be administered directly either by the Department of Health or the Health Boards, although the management board of the Disease Surveillance Unit should include representatives of these and other relevant bodies".

The Hickey report also made reference to the proposal for a single market. This has since been established (January 1st 1993) and now facilitates the freer movement of people, goods (incl. food) and services throughout Europe, with a consequent increase in the general rate of modern international travel in and out of Ireland. Because of this, it was felt important, if not essential, for Ireland to participate in international arrangements for the surveillance and control of communicable diseases and, again, the establishment of a national surveillance unit would greatly facilitate this.
The worthwhile objectives outlined above can be better understood by analysing the existing systems of surveillance in Ireland, and by comparing the trends noted with those reported by the UK centres for the surveillance of intestinal tract infections. *Salmonellas sps.* and *Campylobacters sps.*, the most important causative agents of foodborne disease, are particularly worth examining.

There are 3 different approaches to surveillance of intestinal tract infectious diseases in Ireland.

1. **Statutory Notifications (of scheduled infectious diseases)**

   By which medical practitioners attending patients under their care notify scheduled infectious diseases to the local Director of Community Care. This has already been referred to (3.4). These include confirmed cases of intestinal tract infections such as salmonellosis, typhoid, bacillary dysentery and gastroenteritis (in children under 2 years of age) as well as many more non-intestinal diseases. *(Appendix 1).* Such notifications are either made by GPs or the attending doctors in hospitals. The Director of Community Care in turn notifies the Department of Health on a regular basis. However, while total cases of 'Food Poisoning' and salmonellosis in Ireland are collated and published through this system, data on other foodborne pathogens such as *Campylobacter sps.* and *Listeria sps.* are not published as diseases caused by these pathogens are not notifiable. As full particulars regarding individual cases are not included with these reports, they are of little value from an epidemiological perspective.
2. Laboratory Reporting of Total Numbers of Salmonella Isolates to the Department to the Department of Health

This is an unreliable data collection system for the reporting of combined totals of *Salmonella* isolates from both human and veterinary sources. Reports are sent to the Department of Health on an informal basis from a number of veterinary and medical laboratories around the country. Data collected is not collated or interpreted in any meaningful way. It originally provided an alternative system for the surveillance of salmonellosis following the setting up of the Zoonoses Committee in 1970 by agreement between the Departments of Health and Agriculture.

3. Regional Laboratory Surveillance Systems

There are at present two well established regional systems in operation. One is based in Cork which covers the greater munster region, the other based in Dublin which collects and collates data from laboratories in Dublin city and county mainly, although areas ‘outside Dublin’ (mainly neighbouring counties of the EHB) may also be included for surveillance purposes. While these two units probably represent the forerunners of a national surveillance centre as recommended by Hickey (1990), they operate totally independently without any co-ordination or collation of data collected. A new unit has recently been set up in the North Western Health Board, with proposals for a 4th regional unit to be established by the Western Health Board. As the Cork & Dublin surveillance units are central to the context of this discussion, data collected and published to date will be discussed and compared, with further comparisons with data published by the more established ‘national’ centres in the UK.
(a) The Laboratory Surveillance System (LSS) in the Eastern Health Board.

This system started in 1989 with the goodwill and co-operation of a number of microbiologists and public health doctors and produces a quarterly report (I.D. BULLETIN), the purpose of which is "to inform professionals involved in the control of communicable diseases of the results of the surveillance system" (Anon. 1989). Table 3.6.1 shows the full list of diseases under surveillance at present, although individual infections may be added or removed as considered appropriate.

By 1994, all the major microbiology laboratories of the EHB were participating (11 Total), - a large increase from the 3 laboratories that initially reported in 1989.

Table 3.6.1 List of Infectious Diseases under Surveillance (LSS-EHB)

| Intestinal Tract Primarily: | Salmonella; Shigella; Camplyobacter; |
|                           | Listeria; Hepatitis A; Rotavirus; |
|                           | Cryptosporidium. |

| Others: | Meningococcus; Tuberculosis; Hepatitis B; Influenza |

(a) Southern Communicable Disease Surveillance (SCDS)

Reports of communicable diseases from six hospital laboratories are published in a quarterly report (‘INFOSCAN’ - Southern CDR), the goal of which is "to increase the awareness of infectious diseases in community and hospital practice, thereby optimising the investigation, diagnosis, therapy, control and prevention of such illnesses (Anon(e), 1991). Reporting began in 1991.

INFOSCAN is produced on a voluntary basis by a committee, and is distributed free of charge to all G.P.s, public health doctors, hospital consultants, contributing laboratories and infection control officers. Each quarterly issue also contains two brief articles of special local interest. While all the diseases under surveillance by LSS are also covered in the southern system, the latter appears to cover a wider range of infections as each quarterly issue of ‘Infoscan’ gives specific case numbers of various infectious diseases grouped as follows:-

1. Meningitis quarterly report: e.g. Neisseria meningitis types; Streptococcus pneumoniae; Viral meningitis; Listeria; S. aureus etc.

2. Bacteraemia Quarterly Report: e.g. Haemophilus influenzae; E. coli; S. aureus; Anerobes; Salmonella spp.; Streptococci spp. etc.

3. Mycobacteria Quarterly Report e.g. Mycobacterium tuberculosis; M. bovis; M. avium-intracellularure etc.

4. Gastroenteritis Quarterly Report: e.g. Camplyobacter spp.; Protozoa spp. Enteroviruses; Shigella; Enteropathogenic E.coli etc.
5. Salmonella Quarterly Report: e.g. Various Salmonella serotypes, including S. enteritidis; S. virchow; S. typhimurium; S. agona; S. bredeney; S. dublin etc.

6. Dermatophyte Quarterly Report e.g. Trichophyton rubrum; T. verrucosum; T. mentagrophytes; Candida sps.; Microsporum canis etc.

7. Sexually Transmitted Diseases: e.g. Trichomonas vaginalis; genital warts; chlamydia trachomatis etc.

While infectious diseases grouped in 4 and 5 are the only ones of concern to this discussion, it is important to realise that many of the infectious agents of the gastro-intestinal tract e.g. Salmonella sps.; E. coli; S. aureus; Listeria sps., etc. (which may, of course, be foodborne) also cause infections of a more serious nature such as meningitis, bacteraemia etc. in other parts of the body.

The value of establishing a national surveillance unit can be seen by taking a closer look at the data including trends already highlighted in published issues of both the 'I.D. BULLETIN' and 'INFOSCAN'. While one cannot make definitive statements from an analysis of data sets from the two systems given that these operate totally independently, it is nevertheless possible to depict certain trends. These can be discussed separately or compared with the more established surveillance patterns recorded by the UK centres. However, before going on to discuss published data from the surveillance of enteropathogens in Eire in some detail, it is firstly necessary to consider the factors that may influence the interpretation of this data.
3.7 Factors That Influence the Interpretation of Surveillance Data

A clear understanding of the nature and extent of surveillance is necessary in order that this data can be interpreted in its proper context. A number of factors may influence this interpretation and in relation specifically to the two Irish regional units may include:

1. **Geographical area covered by surveillance including the (a) nature and (b) size of population served.**

Both Irish systems are regional in focus, with the Cork based unit covering the greater munster area and the eastern health board unit reflecting trends in infectious diseases, primarily in the Dublin area. Published data from the UK, on the other hand, represent national totals for various enteropathogenic agents, although a breakdown of the incidence of infection in the various regions may also be obtained.

As regards the two Irish regional centres, it is probable that the Dublin unit reflects trends in a more urban population, whereas the southern system is likely to be more representative of both a rural and urban population. This is relevant in that urban-rural differences in the epidemiology of certain diseases is likely to emerge from time to time.

Such a difference in the epidemiology of campylobacteriosis for example, has been reported in Scotland (Narayan KMV, 1989). Differences in the incidence of infection amongst rural and urban populations was first observed in an earlier study, 1978-82 (including differences in seasonal peak, and slight differences in age-specific incidence rate).
These differences disappeared for the most part during the subsequent 5-year period and it is thought that this may have been due to the beneficial effects of legislation introduced in 1983, prohibiting the retail sale of unpasteurised milk in Scotland.

2. The number of contributing laboratories

9 of the 11 participating laboratories in the eastern region are Dublin based, whereas the 6 laboratories in the southern system are dispersed throughout Munster and include the large county/regional hospitals in Tralee, Limerick, Waterford as well as the Cork Regional Hospital and other Cork hospitals. Obviously, the more hospitals that contribute to a surveillance programme, the greater will be the extent of surveillance, thus reflecting more representative and population based data sets.

The comprehensive system of surveillance of enteropathogens and other infectious agents in England & Wales for example, is better understood when consideration is given to the vast network of contributing laboratories (52 PHLS and 300 NHS hospitals) situated throughout the country (Cowden JM; CDSC - personal communication).

3. The Number/Types of pathogens covered by Surveillance

The main intestinal tract infectious agents included for surveillance purposes by both Irish units are, Rotavirus, Campylobacter, Salmonella, Shigella and Cryptosporidium.
Surveillance in the southern region appears however to be more extensive, with reports on *Giardia, Yersinia, E. coli 0157* and other enteric pathogens included in their quarterly returns. Most laboratories routinely screen hospitalised cases of gastroenteritis for *Salmonella, Campylobacter* and *Shigella*.

However, screening for other intestinal pathogens such as *Giardia, Cryptosporidium, Enteropathogenic E. coli* and *Rotavirus* might only be undertaken if the patient is under a specific age (e.g. under 2-4 years for *E. coli, rotavirus* etc. or, perhaps, under 7-10 years in the case of *Cryptosporidium* - depending on the laboratory) with other criteria applying where there is evidence of recent travel abroad etc. An agreed criteria for screening amongst all participating laboratories is required as individual laboratories currently apply their own criteria. This would facilitate the discussion and interpretation of the epidemiology of gastrointestinal disease in different regions.

4. The degree of awareness and co-operation of G.P.s

While practitioners are statutorily bound to report clinical diagnoses of specific scheduled infectious diseases (including food poisoning) the surveillance of intestinal tract infectious agents is obviously dependent on laboratory confirmation of these agents.

G.P.s have an important role to play here by initiating the submission of clinical specimens, so that a more complete diagnosis can be achieved. While the more severe cases of gastroenteritis would be hospitalised and screened anyway, many more cases of gastro-intestinal tract infections go unrecorded as a result of specimens not being sent for laboratory examination. Further, the notification of gastroenteritis in the first instance is very haphazard.
Research carried out into this in Co. Kerry confirmed that, while a minority of G.P.s consistently report cases, the majority hardly ever comply with their statutory duty to do so. Lack of consistency throughout the country in the reporting of notifiable infections could result in the perception that some regions have a much higher incidence of specific diseases, when in fact this might be due to a greater contribution to the notification process.

The establishment of sentinel ('spotter') studies should be undertaken by the Irish College of General Practitioners (I.C.G.P.) in conjunction with the Health Boards, in an effort to ascertain the true incidence of intestinal tract infectious diseases in the community.

While such a scheme, 'spotter practice network' (SPN) was established in 1989 for the voluntary reporting of viral infections affecting children (measles, chicken pox, etc.), this method of surveillance could be extended to include food poisoning specifically and/or intestinal tract infections. The proposed new Public Health Departments presently being established within the Health Boards will have a central role to play here. One of their responsibilities will be the surveillance of communicable diseases and the incorporation of a GP Unit in each Department will facilitate this work.

It is now proposed to discuss the principal foodborne intestinal tract pathogens in Ireland, and to comment on emerging patterns and the significance of these from a public health perspective. To broaden the discussion, comparisons will be made with UK data as presented in Figures 2.4.4-2.4.6 in the previous section of this thesis.
Figure 2.4.6 is of particular interest as it compares total reports of the important intestinal tract infectious agents over a 12 year period (Engl. & Wales) with Figures 2.4.4 and 2.4.5, depicting the relative distribution of the three most significant enteropathogens for 1991 and 1992 recorded by the Communicable Disease Surveillance Centre (England & Wales) and the Communicable Disease (Scottish) Unit respectively. Figures 3.8.1 and 3.8.2 depict the relative distribution of the same pathogens reported by the two Irish regional units, as adapted by the writer for comparison and discussion purposes.

3.8 The Surveillance of Salmonella Sps.

At the outset, one has to be careful when discussing data from the Dublin based surveillance system (LSS), as reports can refer to isolates from residents within Dublin city and county only, with separate totals for residents 'outside Dublin'. The relevance of this can be seen from the fact that 93 (25%) out of a combined total of 364 reports of Salmonella sps. for both 1991 and 1992 were from cases living outside Dublin (Anon (C), 1993).

This obviously has implications when an epidemiological/population based analysis of data is made, as the 'outside Dublin' area included in the surveillance data to date has not been not clearly defined. However, from 1994 onwards, data will be reported for a population base of the EHB, as well as for Dublin (Anon, 1994). The same general point can be made regarding the Cork based surveillance unit, as reports do not usually define a specific population base ('Greater Muster area').
Distribution of Enteropathogens Reported within the LSS - Dublin and Outside Dublin.

1991 (N=970)

1992 (N=1027)

1993 (N=1145)

References:
Figure: 3.8.2

Distribution of Enteropathogens reported within the Southern Surveillance System, 1992 and 1993

References:
Data from the Dublin surveillance unit (LSS) for 1991-93 (Figure 3.8.1) confirms that *Salmonella sps.* were the 2nd and 3rd most frequently isolated intestinal tract infectious agents recorded during this period with its significance decreasing during this period. This was different to the Cork based surveillance unit in that *Salmonella sps.* on average occupied the 4th position over the 2 year period 1992-93 (Figure 3.8.2). Reports of total isolates of *Salmonella sps.* were understandably far greater in the Dublin system with a combined total of 381 cases for 1992 and 1993, compared to 238 recorded in Cork over the same period, as shown in Table 3.8.1.

### Table 3.8.1 Comparison of Total Isolates of Salmonella and Campylobacter sps. Dublin and Cork. 1992 and 1993

<table>
<thead>
<tr>
<th>Laboratory. Surveillance System (L.S.S.) Dubin (incl. 'outside Dublin')</th>
<th>Southern Communicable Disease Surveillance (Greater Muster Area)</th>
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<tbody>
<tr>
<td><strong>Salmonella Sps.</strong></td>
<td></td>
</tr>
<tr>
<td>1992</td>
<td>165</td>
</tr>
<tr>
<td>1993</td>
<td>216</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>381</strong></td>
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<td></td>
<td></td>
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<tr>
<td><strong>Campylobacter Sps.</strong></td>
<td></td>
</tr>
<tr>
<td>1992</td>
<td>146</td>
</tr>
<tr>
<td>1993</td>
<td>160</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>306</strong></td>
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</tbody>
</table>

Source: Adapted from various editions of I.D. Bulletin and Infoscan (1992-94)

However, when one examines the first population based epidemiological review of laboratory diagnosed *Salmonella sps.* in Ireland (1991 - Table 3.8.2) it was noted that a much higher incidence was recorded in Cork (by a factor of 2.5). In fact, the rate in Dublin for 1991 was much closer to that recorded in Northern Ireland, both of which were significantly lower than rates in England and Wales.
Table 3.8.2 Comparison of Incidence of Salmonellosis 1991 (rate per 100,000 pop.)

<table>
<thead>
<tr>
<th>Region</th>
<th>Rate</th>
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<tbody>
<tr>
<td>Dublin</td>
<td>14</td>
</tr>
<tr>
<td>Cork</td>
<td>34</td>
</tr>
<tr>
<td>Northern Ireland</td>
<td>10</td>
</tr>
<tr>
<td>England and Wales</td>
<td>44</td>
</tr>
</tbody>
</table>


The significant difference in recorded rates between the two Irish regional systems has not been explained, although it is known that the Cork region experienced an explosive increase in *Salmonella enteritidis* isolations in the summer of 1990 and to a lesser degree in the autumn of 1991 (Keane and Ryan 1992). We also know from surveillance that the Dublin region escaped this sharp increase in the incidence of salmonellosis. As to why the southern region should follow more closely the experience of the UK, where from 1988 onwards, cases of salmonellosis reached epidemic proportions, is not clear. The following discussion on trends observed in the Cork area may help to explain this. Indeed, an opportunity may have been lost whereby a full-scale epidemiological study could have been carried out to examine and explain the full extent of regional variation in the incidence of salmonellosis between Dublin and Cork and indeed other regions.

The existence of regional variations in the epidemiology of diseases reinforces the case already made for a National Surveillance Unit for infectious diseases in Ireland. This unit would automatically 'pick up' on regional differences in the reported incidence of gastrointestinal disease throughout the country, with consequential follow-up investigations to explain the reasons for this.
3.8.1 Salmonella enteritidis PT4

As discussed previously in this thesis, the epidemic of salmonellosis experienced in the UK in the late 1980s resulted from a sharp increase in the incidence of \textit{S. enteritidis} isolations, most of which were of phagetype 4 (PT4). Taking 1991 as an example, one can see a very similar pattern between Cork and England & Wales for the relative distribution of \textit{S. enteritidis} serotypes reported by these surveillance systems.

Dublin, on the other hand, reported a much lower proportion of \textit{S. enteritidis} isolates, identifying more closely with data from Northern Ireland. Figure 3.8.3 summarises this data recorded by these 4 surveillance centres/units for 1991.

While \textit{S. enteritidis} has clearly established itself as the most frequently isolated serotype in the UK, evidence from Cork suggests that, in the South at least, the problem has since been resolved with isolation rates (as a % of total isolations of \textit{Salmonella sps.}) being significantly reduced in 1992 and 1993. Figure 3.8.4 clearly demonstrates this. This is substantiated by the evidence presented in Figure 3.4.1 which presents data collated through the parallel system of statutory notifications.

The sharp increase in the incidence of \textit{S. enteritidis} in the UK and the Cork area has been associated epidemiologically, (and in some incidents confirmed bacteriologically) with the contamination of raw shell eggs, and to foods like custard cakes, mayonnaise, meringue etc., made from shell and liquid egg. (as previously discussed).

123
Comparison of S. enteriditis isolations with isolations of all other Salmonella spp within the Southern Surveillance System 1991-1993

References:
It has been concluded that contaminated liquid egg marketed in the Cork area was the source of the sharp increase in the incidence of salmonellosis in Cork in July & August 1990 (Keane and Ryan, 1992). Figure 3.8.4 confirms, however, that the incidence of \textit{S. enteritidis} has declined since then, with latest figures for 1993\(^1\) showing that there were only 36 cases reported in that year in the whole southern region. This represented 29\% of total \textit{Salmonella} isolates and compares favourably with the 25\% figure recorded for isolations of \textit{S. enteritidis} in Dublin for the same year (Anon, 1994).

Like Cork, Dublin also reported an increase in \textit{S. enteritidis} isolations during the 3rd quarter of 1990, (all PT4). However, it did not experience the outbreak situation recorded in the Cork area at that time. In fact, \textit{S. typhimurium} has clearly established itself as the predominant serotype reported in the Dublin area, accounting for 54 (40\%) out of a total of 134 \textit{Salmonella} organisms serotyped during 1991 (Anon(C), 1993) with more recent figures for 1992 & 1993 confirming this trend (Anon, 1994). However, less common serotypes are likely to appear from time to time, as was observed in 1990. During that year, \textit{S. bredeney} was the most frequently isolated serotype, with 48 (38\%) out of a total of 126 \textit{Salmonella} isolations. This sudden increase in isolations of \textit{S. bredeney} was noticed in the last quarter of 1990, with the epidemic curve suggesting a point source outbreak (Anon (E), 1990). In fact, a number of cases occurred in one institution although investigations failed to pinpoint the source.

Most recent data (1993) confirm that \textit{Salmonella sps.} account for approx. 7-10\% of total enteropathogens reported by both surveillance units (\textit{Figures 3.8.1-3.8.2}). However, what is of more importance is the actual incidence of salmonellosis in the community when expressed on a rate per population basis.

\(^1\) INFOSCAN 1993, Vol. 3, Nos. 1-4
The pattern in the UK suggests that the rate of isolations of *Salmonella sps.* reached its peak in both Scotland\(^1\) and England & Wales\(^2\) in 1992 (approx. 60/100,000 population) with Northern Ireland\(^3\) showing a much lower rate (approx. 10/100,000 population) for the same year, having peaked in 1987 (approx. 25/100,000 population). Interestingly, the rate in Dublin, (City & County) for 1992\(^4\) was almost identical to that recorded in Northern Ireland, so on the surface at least, the incidence of salmonellosis appears to be much lower in Ireland (North & South) at present than it is in the UK.

There is a bias in most countries including the UK and Ireland towards more complete investigations of gastroenteritis in children. However, data from the two Irish regional surveillance units, as reflected in the proportionally very high number of reports of *Rotavirus* infections, suggests that this bias may be particularly strong in this country.

As a consequence of this the number of isolations of *Salmonella sps.*, when expressed as a percentage of total isolates (of enteropathogens), will always appear to be quite low in Ireland when compared to UK data. However, as *Salmonella sps.* are by far the most common causative agents in foodborne disease outbreaks in the developed world, the active surveillance of this very important enteropathogen in Ireland is of prime public health concern, requiring each case to be individually followed up.

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Adapted from surveillance data on gastrointestinal tract infections

\(^1\) Scotland: CD(S)U
\(^2\) Eng. & Wales: CDSC
\(^3\) N. Irl.: DHSS
\(^4\) Dublin: LSS

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In recent years, *Salmonella sps.*, the once predominant enteropathogens, have been overtaken by *Campylobacter sps.* as the most frequently isolated intestinal tract infectious agents under surveillance in the UK.

This foodborne microorganism tends to cause sporadic illness rather than outbreaks of 'food poisoning' as is often the case with *Salmonella sps.*, although it is generally accepted that many real outbreaks of foodborne campylobacteriosis may go unrecognised.

While in most parts of the world, *C. jejuni* is the predominant species, accounting for 80-90% of infections (Skirrow, 1990), the species/serotype breakdown is not normally recorded in published surveillance data. Therefore, unlike *Salmonella sps.*, for which the serotype breakdown is more readily available, surveillance data in relation to *Campylobacter sps.* refers to 'total' isolates only. In the UK, it appears that the incidence in Scotland and England & Wales is quite similar with the incidence for 1989 for example being 60.5 and 64.3 per 100,000 population respectively, with Northern Ireland showing a much lower incidence (12.1/100,000 pop). This is further confirmed by the data presented in Table 3.9.1.

As with rates of isolation of *Salmonella sps.*, data for *Campylobacter sps.* suggests that both North and South of Ireland (as represented by Dublin data) compare more favourably than does the North with the rest of the UK. Again, rates in both Scotland and England & Wales were very close with Northern Ireland being much lower, although significantly higher than in 1989.
Table 3.9.1. Comparison of Incidence of Campylobacteriosis between Dublin & the UK, 1991.

<table>
<thead>
<tr>
<th>Region</th>
<th>Incidence per 100,000 population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dublin</td>
<td>7.9</td>
</tr>
<tr>
<td>Northern Ireland</td>
<td>19.3</td>
</tr>
<tr>
<td>Scotland</td>
<td>67</td>
</tr>
<tr>
<td>England &amp; Wales</td>
<td>65</td>
</tr>
</tbody>
</table>

Source: Adopted from surveillance data on gastrointestinal infections in each region, i.e. Northern Ireland (DHSS), Scotland (CD(S)U), England & Wales (CDSC), Dublin (Anon(F), 1991)

The much lower incidence recorded in Dublin may reflect a lower level of awareness/investigation, although an actual low level of infection in the community may, in fact, be the case.

While the Dublin region (including 'outside' areas) recorded a greater frequency of isolations of total *Salmonella* sps. in 1992 and 1993 (381 isolates) than the Cork surveillance unit (238 isolates), when one examines the data on *Campylobacter*, the situation is reversed. For these two years, there were 306 cases of campylobacteroisis in Dublin as opposed to 508 cases in Cork region - (60% higher) (Table 3.8.1). However, this data refers to ‘total’ isolates only - population based incidence cannot be calculated or compared as the population served by the regional systems is not clearly defined (Unlike data for Dublin city and county, which has a defined population). An increase in population based surveys is desirable. Future studies should then reflect the true incidence of both salmonellosis and campylobacteriosis in areas like Dublin city and county, Cork city and county, and, indeed, Galway city and county, should a unit be established for Galway and the West of Ireland generally.
In summary, while a greater frequency of isolations of *Campylobacter sps.* was recorded in the Cork region than in Dublin, when expressed as a percentage of total isolates there was very little difference between both sets of data. (Figures 3.8.1 and 3.8.2).

Finally, as *Campylobacter sps.* are clearly foodborne pathogens, isolations of these microorganisms should be reported and followed up. Sporadic cases may, in fact, be part of one large community outbreak with a common vehicle of infection, and it is only by vigilant surveillance, supported by follow up investigations, that a link with a food vehicle can be epidemiologically established.

Unlike salmonellosis, infections due to *Campylobacter sps.* are not statutorily notifiable, but with the dramatic emergence of this enteropathogen in the UK in recent years (albeit partly due to increased awareness), its significance from a public health point of view should not be underestimated in this country.

### 3.10 The Surveillance of Other Intestinal Tract Pathogens

The other main intestinal tract infectious agents under routine surveillance in this country include *Rotavirus, Shigella,* and *Cryptosporidium.* While published data from the Dublin region gives details of total isolates of these enteropathogens (together with totals for *Salmonella* and *Campylobacter sps.*), the Southern Surveillance unit has, since 1991, been publishing data on other intestinal tract pathogens, such as *Giardia,* enteropathogenic *E. coli,* *Yersinia enterocolitica,* *E. coli 0157,* *Adenovirus,* *Cl. difficile* etc. (Figure 3.8.2).
However, for the most part, these microorganisms cause infection in young children, normally from person to person via the faecal-oral route, without food being the vehicle of transmission.

While the surveillance of these microorganisms, unlike the surveillance of *Campylobacter* and *Salmonella* sps., does not strictly come within the scope of this discussion, it is nevertheless relevant to conclude this section with a brief comment on their significance in the overall context of the surveillance of gastrointestinal disease in Eire. The significance of the *Rotavirus* in particular is worth examining given its apparent predominance.

### 3.10.1 Rotavirus

This very common childhood intestinal pathogen appears to cause on average approx. half of all gastroenteritis cases reported in this country (**Figures 3.8.1 and 3.8.2**). *Rotavirus* causes diarrhoea in young children and, for this reason, assays are only carried out on samples from children under 2 years of age in Cork University Hospital (Cryan and Whyte, 1993) and, in many other medical laboratories. In both the developed and developing countries, *Rotavirus* is associated with about one-third of the hospitalised cases of diarrhoeal illness in infants and young children under 5 years of age. All children are infected in their first 3-4 years of life (Anon(D), 1993). As with other human gastroenteritis viruses, the *Rotavirus* is highly transmissible, with the infective dose believed to be as little as one viable particle (Collins, 1994).
Statistics from the UK confirms the lesser role played by the *Rotavirus* in their overall surveillance programmes for gastro-intestinal infections. For example, an examination of data from Scotland\(^1\) and England & Wales\(^2\) in recent years shows that, with the exception of 1992, *Rotavirus* has been the 3rd most frequently isolated enteropathogen. In that year, *Rotavirus* dropped to 4th place, having been overtaken by *Shigella* (Sonnei) in both Scotland and England & Wales. It would appear that the cyclical re-emergence of the latter pathogen - every 7 years or so in Scotland for example, (JCM Sharp, personal communication) was responsible for the dramatic increase in *Shigella sonnei* isolates in 1992 in the UK.

However, data from Ireland and the UK differs significantly in the relative importance of the *Rotavirus* in the different surveillance systems. In Ireland, (Cork & Dublin), the *Rotavirus* is clearly the mostly frequently isolated pathogen. This is probably due to the selective nature of investigating this common childhood infection with a greater bias towards the surveillance of gastroenteritis in children under 2 years of age in particular *(Appendix 1)*. In Scotland for example, where data is collated for 14 enteropathogens (incl. 4 different *Shigella sps.*), the *Rotavirus* appears to be the causative agents on average of approx. 14% of all gastrointestinal tract infections with 1538 (15%) cases being reported in 1990, 1631 (14%) in 1991, and 2340 (13.6%) in 1992. Very similar statistics were reported in England & Wales, with isolation rates of 15.5%, and 14.1% recorded for 1990 and 1991 respectively. *(Figs. 2.4.4 & 2.4.5)*

\(^1\) Scotland: CD(S)U - Adapted from Gastrointestinal disease surveillance data.
\(^2\) Eng. & Wales: CDSC - Adapted from Gastrointestinal disease surveillance data.
Even allowing for adjustments, whereby only the 5 main enteropathogens\(^1\) under surveillance in this country are included for comparison purposes, the proportion of *Rotavirus* isolations (as a % of total isolates) would read: 17.8%/1990; 16.8%/1991 and 15.5%/1992 (Scotland) For England & Wales the corresponding figures for 1990 and 1991 would be adjusted upwards to 17.5% and 15.7% respectively.

In Eire, *Rotavirus* infections are typically prevalent over the winter months. A review of the Dublin based laboratory surveillance system (LSS)\(^2\) over the 1991-93 period confirms this, with the last quarter (Oct-Dec) showing most infections. During these years, 484 infections occurred in this quarter compared to 140 in the 2nd quarter (April-June) when fewest infections were reported.

In Cork, a similar pattern is observed, although, to date, most cases have been reported in the Jan-Feb. period (Cryan and Whyte, 1993).

The predominance of *Rotavirus* as reported by the two regional surveillance units in Ireland clearly does not follow the pattern observed in the UK. This apparent discrepancy between the surveillance figures in both countries needs to be clarified. However, as both Irish units develop to include a more comprehensive screening programme for investigations into intestinal tract infections in the community, the overall significance of the *Rotavirus* as a proportion of all enteropathogens identified will probably decrease.

\(^1\) *Rotavirus, Salmonella, Camplyobacter, Shigella and Cryptosporidium*

3.10.2 Other Enteropathogens under Surveillance

The two other principal intestinal tract infectious agents included for surveillance purposes in both Irish regional units are *Shigella sps.* and the protozoan *Cryptosporidium*. While *Shigella* is normally spread directly from person to person via the faecal-oral route, water and milk may act as an indirect vehicles of infection. Two-thirds of cases occur in children under 10 years of age, and morbidity is greatest in the very young and old (Gannon, 1993).

The cyclical nature of the occurrence of shigellosis accounts for its re-emergence to significant proportions approx. every 7 years as previously mentioned.

On a line through 1991, one can see a very similar pattern in Scotland, England & Wales (Figures 2.4.4-2.4.5) and Dublin (Figure 3.8.1) in that *Shigella* accounted for 11-12% of all enteropathogens reported by the 3 surveillance units.

For 1992, the situation was somewhat similar, with isolation rates in Dublin of 15% (Figure 3.8.1), Cork 11% (Figure 3.8.2), England & Wales 15%, (Figure 2.4.4) with Scotland showing a higher rate of 22.7% - expressed as a % of total isolates. (Figure 2.4.5).

In Dublin¹, the least number of cases occurred during the last quarters of 1991-1993, with very little difference between reports in the other quarters of the year.

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¹ I.D. Bulletins 1991-1994
Cryptosporidium, the coccidian protozoal parasite was first implicated in human gastro-enteritis in 1976 (Cryan and Whyte 1992). In England & Wales, this was the 5th most frequently isolated enteropathogen in 1992 (Figure 2.4.4) with 5206 cases (provisional), with Scotland showing roughly similar trends (on a rate per population basis) with 954 cases.

In Dublin, most cases are reported in the 2nd half of the year with a combined total of 55 cases being reported in the 1st and 2nd quarters of 1991-1993, as against 93 cases for the 3rd and 4th quarters - an increase of 70%. Data from Cork reflects similar trends in that for 1991 most cases were reported during July and August (i.e. 3rd quarter) (Cryan and Whyte 1992). However, spring peaks have also been reported.

Cryptosporidium occurs mainly in young children, although it is accepted that the age distribution may be biased in that some laboratories screen for Cryptosporidium in young children only, with adults being screened on request (e.g. in instances where contact with farm animals is suspected).

A large community outbreak of cryptosporidiosis was recorded in the Swindon and Oxford districts of England in 1989. This outbreak confirmed drinking water as the vehicle of infection. This however will be discussed more fully in the chapter dealing with water supplies.

As infections due to lesser recognised enteropathogens are likely to emerge from time to time, Irish surveillance systems should be prepared to broaden their screening base to include the surveillance of such intestinal tract pathogens.
Data from the Cork Unit suggests that such a programme already exists as their more recent issues of INFOSCAN have included data on such pathogens as *Giardia, Adenovirus, Yersinia enterocolitica, E. coli 0157, Cl. difficile* and others. It is hoped that the Dublin unit will also expand to include the surveillance of these infectious agents.

3.11 Summary

Comparability of data on intestinal tract infections can be difficult in the absence of background information into the various surveillance systems under discussion. To what extent increases observed in gastro-intestinal tract infections in recent years (most notably in the UK) are real, or are a consequence of greater awareness, more frequent submission of faecal samples to laboratories, increased microbiological and epidemiological investigations, new diagnostic technologies and/or enhanced reporting and surveillance, is difficult to interpret.

Increased travel abroad in latter years has contributed in part to increased isolations of some enteropathogens in the UK. However, the same general claim cannot be made for Irish surveillance data, given that the vast majority of intestinal infections (notably, *Rotavirus* infections) are reported in children under 5 years of age with lesser likelihood of an association with travel abroad in this age group.

Data from the surveillance of *foodborne* infectious diseases and intoxications while by definition implicating a food source, should not be discussed and interpreted in total isolation to the surveillance of gastrointestinal tract infections generally.
Surveillance of the latter does not necessarily include follow-up investigations aimed at confirming a mode of transmission/vehicle of infection, (except perhaps in outbreak situations). However when consideration is given to the fact that two most prevalent enteric pathogens reported in the UK (i.e. *Campylobacter* and *Salmonella sps.*) are mainly transmitted through the food chain, it is obvious that there should be an increased awareness of the role of food as a major source of infection in the community.

While the establishment of the two regional surveillance units in Eire has been a very welcome development, it is hoped that, as recommended by Hickey (1990) and others, a national surveillance centre will be set up in the near future to collate and analyse trends in the epidemiology of important infectious diseases on a national level. Again this should be facilitated with the establishment of the new Public Health Departments within the Health Boards.

Finally, while Eire’s contribution to the WHO surveillance programme for foodborne diseases and intoxications to date has been virtually non existent, the publication in 1994 of the Department of Health’s ‘Shaping a Healthier Future’ - a strategy for effective healthcare in the 1990s (DoH 1994), gives cause for optimism in that the development of a “national programme for the control of foodborne infection” is set out as one of its targets.

This target was based on the advice of the Food Safety Advisory Committee’s recommendation for a national surveillance programme of foodborne diseases published about the same time (Anon (A) 1994).

It is hoped that this unit will be given the resources to carry out its work effectively so that Eire’s future contributions to the WHO European Surveillance Programme will be both constructive and meaningful.
4.1 Introduction

The epidemic of foodborne salmonellosis in the UK in the latter half of the 1980s drew unprecedented attention to the food industry in general, with particular emphasis on the poultry/egg production and catering sectors, and to how these were being controlled from a food safety perspective.

The question of food safety had previously received heightened media attention with some reports raising public concern to a degree out of all proportion to the actual hazard existing. While *Salmonella* is probably best known to the average consumer as the 'germ' most popularly associated with 'food poisoning', in recent years the public have been bombarded with various food scares, implicating lesser well known but potentially very hazardous microbiological agents such as *Listeria, Cl. botulinum* and more recently *E.coli 0157*. The 'Listeria hysteria' of fairly recent times is one example of media hype which caused exaggerated public concern, and focused undue attention on the food industry, causing the industry and consumers to look with suspicion on the 'cold chain'. This was because this 'new' food poisoning agent was reported to survive and grow at temperatures that had always been accepted as safe for the storage of chilled foods. This was despite the fact that there are very few examples of confirmed incidents of foodborne Listeriosis. The very high mortality rate in vulnerable groups, no doubt, was a major contributory factor to concerns expressed
However, many food scares down through the years caused genuine public concern about food processing and handling procedures throughout the food industry. The *Cl. botulinum* incidents in the UK in 1978 (implicating imported canned salmon) and in 1992 (implicating hazelnut yoghurt) lead to greater awareness of this potentially deadly toxin producing bacterium (O’Mahony et al, 1990). These incidents and the Aberdeen typhoid outbreak of 1964 (Anon, 1964) focused Government attention on imported foods with a need for having strict controls such as sampling programmes etc.

However, by far the greatest number of food poisoning incidents have been associated with *Salmonella ssp*. The Stanley Royd Hospital outbreak of salmonellosis of 1984 (DHSS, 1986) which affected about 400 people, 19 of whom died (all patients) only galvanised previous concern in relation to hospital/institutional catering. The *Salmonella ealing* incident the following year affected 42 people, mostly infants (one of whom died) as a result of consuming dried milk powder (Rowe et al 1987). This caused panic in the parents of babies, and resulted in all products being recalled with production having to stop. A large outbreak of *S. typhimurium* food poisoning in 1989 in North Wales associated with cooked meats affected well over 500 people caused further public concern, and lead to a re-examination of existing arrangements for the handling of serious outbreaks of food poisoning in the UK (Anon (F) 1991).

However, despite the various reports of outbreaks of foodborne disease down through the years in the UK, the recent epidemic of ‘food poisoning’ caused by *Salmonella enteritidis PT4* especially throughout Europe and parts of North America was unprecedented. To this day, the control of this organism in particular remains the greatest challenge to Government Health Authorities, the State Veterinary Services and the poultry production industry.
Evidence from microbiological, epidemiological and veterinary sources confirmed that the epidemic was primarily due to infection in chickens, and not only from their contaminated carcasses but also from a new source - the contents of 'intact' hens eggs (PHLS, 1989). Eire, by and large, remained clear of this outbreak of salmonellosis, although localised infection was reported in the Cork area in particular in 1988 and 1989 (D. White, Cork Regional Hospital; H. Cowman, Food Hygiene Lab. Cork; M. Sheahan, Department of Agriculture - personal communications).

Poultry meat has long been recognised as a major source of salmonellosis in humans. However, with the emergence of \textit{S. enteritidis PT4} in the UK from about 1986, the poultry industry became the focus of greater attention. Once again, the safety of food became a major public health, political and media issue. On this occasion however, surveillance data from public health and veterinary sources confirmed the disease to be widespread in the community and of epidemic proportions.

Having regard to this latest epidemic and, from previous incidents of foodborne disease implicating poultry, it is very clear that the control of foodborne disease in humans is very firmly linked to the control of pathogens, particularly, \textit{Salmonella sps.} in poultry. Therefore, any discussion on the control of foodborne disease must consider at the outset control at primary level, while accepting that there are many other approaches to the control of foodborne disease, and many other well recognised factors that contribute to the incidence of 'food poisoning' in the community. In this, the final section of the thesis, it is therefore proposed to examine the various approaches to the control of foodborne disease in humans, and how best this can be achieved in the light of present knowledge in this area.
While not covering every conceivable food safety measure, it is, however, proposed to carry out an overview under the following headings:

4.2 The Control of Salmonella in Poultry.
4.3 Other Strategies for the Control of Foodborne Disease.
4.4 Food Irradiation.
4.5 The Hazards Analysis Critical Control Point (HACCP) Approach.
4.6 Measures for the Control of Foodborne Disease at Commercial Kitchen/Food Service Level.
4.7 Control at Retail Level - Temperature Control.
4.8 Hygiene Education.
4.9 Improved Surveillance of Foodborne Disease.
4.10 Water as a Source of Infection and Intoxication.

4.2 The control of Salmonella in Poultry

As far back as 1978, the thirty-first World Health Assembly recognised the need for co-ordinated strategies worldwide for the control of zoonoses\(^1\), including salmonellosis. A network of national control/surveillance centres was set up to co-operate with member states in the planning and implementation of their national programmes, and to provide essential technical backup to national health/veterinary authorities concerned with the control of zoonoses.

Foodborne zoonoses are the most important zoonoses in developed countries, with the foodborne pathogens *Campylobacter* and *Salmonella sps.* being clearly the two most prevalent enteropathogens causing gastro-enteritis/foodborne disease in the UK (Fig. 2.4.6).

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\(^1\) Zoonoses: Diseases transmissible between animals and humans.
Although both infectious agents have, in the past, been epidemiologically associated with common food sources (poultry and raw milk in particular), the association of *Salmonella* sps. with point-source outbreaks, as opposed to the sporadic nature of the occurrence of campylobacteriosis, has projected *Salmonella* as the main infective agent for monitoring and control. Further, it is only within the last 15-20 years that Campylobacter enteritis has been recognised, whereas *Salmonella* sps. have a much longer association with gastrointestinal disease.

Many of the measures recognised for the control of foodborne salmonellosis can also be adopted for the control of campylobacteriosis. However, with the increasing incidence of campylobacteriosis, it may be necessary to implement specific monitoring and control measures for this, the most common human enteropathogen in developed countries.

### 4.2.1 WHO Guidelines 1983

As poultry meat is the food most frequently identified in food poisoning outbreaks where a food source is identified, the control of *Salmonella* sps. in poultry is the most effective way of reducing infection in humans (Humphrey et al, 1988; Sharp, 1991). The World Health Organisation in its 1983 publication "Guidelines on the prevention and control of salmonellosis" (WHO 1983) sets out in great detail specific proposals for the control of this infectious disease.
The guidelines were a response to a recognition of the need for the monitoring and control of the disease, and the key areas where control can be exercised can be summed up as follows:-

(a) The establishment of a *Salmonella* free rearing/breeding environment for food animals. This relates to the design, layout, and construction of pig sties, intensive fattening units for calves, hatcheries and rearing/layer houses etc. for chicks, broilers and layers.

(b) Protection against environmental contamination, such as the elimination of access to housing units by birds, rodents, domestic pets, etc., and the provision of potable water supplies where possible.

(c) The availability of *Salmonella*-free feed by using raw materials of good bacteriological quality; the prevention of cross contamination at production factory, and the use of pelletized feed as one of the best ways for rendering the final product safe and free of *Salmonella sps*.

(d) The establishment of *Salmonella*-free breeding stock by, for example, the establishment of specific pathogen free (SPF) pig production; the isolation of cattle breeding stock from cattle fattened for slaughter; the importation of poultry grandparent stock free of *Salmonella* and the prevention of recontamination. This is a very important area of control and will receive more detailed discussion later.
(e) Transport of animals free from *Salmonella* contamination. This relates to the suitability of lorries for the transport of cattle and pigs in particular, i.e., use of readily cleanable internal surfaces, the proper cleaning and disinfection of these and other materials such as poultry crates etc. This is necessary as food animals are very often stressed while being transported with increased excretion of *Salmonella sps*.

While the above control measures can be applied to the 'clean' production of all food animals, additional internal management/control measures may be needed for the production of specific animals such as poultry, as recommended in the guidelines. Taking chick hatcheries as an example:-

(a) All-in all-out principle and mixing of age groups and species, i.e. the complete segregation of birds of different ages, including layers from broilers, the complete emptying and disinfection of houses, equipment etc. at the end of each rearing or laying cycle.

(b) The removal of dead and sick birds. These should be removed as soon as possible, and incinerated or buried as they may constitute a serious risk to the spread of infection in the flock. Commercial hauliers may introduce *Salmonella* to a farm, so dead birds to be disposed of in this manner should be brought to lorries at the entrance to the farm, rather than allowing the entry of such potential sources of infection.

(c) The disposal of litter. Poultry droppings/litter should be properly removed prior to cleaning and stored in a suitable bay at least 500 metres from poultry houses.
(d) Special hatchery hygiene measures - such as proper building design, e.g. to permit a one-way flow system for air through the hatchery; the use of the correct air filters at air inlets etc; the fumigation of all eggs on arrival at the hatchery, the use of flock codes to identify hatching eggs etc.

While most of the above precautions can be applied to the control of *Salmonella* in breeder, broiler, and layer units, they may each present their own particular control problems which need to be tackled on an individual basis. However, an important control measure common to all sectors of poultry/egg production is the availability of diagnostic/laboratory facilities and related expertise which will focus special attention on rises in chick mortality during the first few weeks of life, and also assist with the routine monitoring of *Salmonella* infection in flocks generally.

The production for slaughter of *Salmonella*-free poultry, as already mentioned, is the most effective way of ensuring *Salmonella*-free poultry meat, with a consequent reduction in the incidence of salmonellosis in humans. However, *Salmonella sps.* can be reintroduced at the slaughter house via cross contamination from contaminated flocks via water, feathers, equipment etc., and at catering/retail level, particularly from raw to cooked carcasses. WHO recognised this and, in the 1983 guidelines, discussed in great detail the various control measures that can be exercised at these levels. These guidelines should be followed closely by all key personnel involved in the production, processing, preparation and handling of poultry meat for human consumption as all have an important role to play in the ongoing struggle to reduce the incidence of foodborne salmonellosis, particularly that caused by *S. typhimurium* and *S. enteritidis PT4*. 
4.2.2 Zoonoses Orders

Surveillance data from the Public Health Laboratory Service (PHLS) in the UK confirms the continuing high reporting of infection due to *Salmonella sps.* in recent years. This is despite the fact that legislative control measures (zoonoses orders) introduced in 1989 during the recent epidemic of *S. enteritidis (PT4)* were specifically aimed at the control/reduction of *Salmonella* in food animals and, consequently, humans. Three zoonoses orders were introduced to control the epidemic (Anon (C) 1990) (Baird-Parker, 1990).

1. Zoonoses Order 1989 designated *Salmonella* (and *Brucella*) as risks to human health, with the mandatory reporting of microorganisms isolated from designated live animals, or carcasses, or from their products or surroundings.

2. Processed Animal Protein Order 1989 - Animal proteins used for animal feeds in the UK were required to be tested for *Salmonella sps.*

3. Poultry Breeding Flocks and Hatcheries (registration and testing) Order 1989 - introduced a registration and *Salmonella* testing system for laying flocks (with greater than 25 birds) and breeding flocks used for commercial layers and broilers.

(Note: Table 4.2.1 gives details of the total number of outbreaks of infection due to *Salmonella enteritidis* and *Salmonella typhimurium* in breeder and layer poultry flocks reported in the UK for the years 1991-1993).
The slaughter policy introduced as a result of the registration order for layers (Point 3) has since been discontinued on the advice of a subsequent independent advisory committee (The Dick Committee) on the control of *Salmonella sps.* in eggs (MAFF, 1993). Professor Dick, in her report, advised MAFF that the level of contamination in eggs when laid is very low, and suggested that controls should concentrate on preventing *Salmonella* multiplying during storage and use. The slaughter policy will, however, continue to apply to breeder flocks found to be contaminated with *S. enteritidis* or *S. typhimurium*.

### TABLE 4.2.1

Outbreaks of *S. enteritidis* and *S. typhimurium* in Poultry Flocks - England & Wales and Scotland 1991-93

<table>
<thead>
<tr>
<th>Type of Poultry</th>
<th><em>S. enteritidis</em></th>
<th><em>S. typhimurium</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Broiler Breeders</td>
<td>251</td>
<td>242</td>
</tr>
<tr>
<td>Layer Breeders</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>Commercial Layers (caged)</td>
<td>54</td>
<td>56</td>
</tr>
<tr>
<td>Commercial Layers (others)</td>
<td>34</td>
<td>18</td>
</tr>
<tr>
<td>Total No. of Flocks infected</td>
<td>355</td>
<td>324</td>
</tr>
</tbody>
</table>


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1. Poultry required to be tested excluding turkeys, ducks and geese. The figures for broiler breeders and layer breeders include reports of isolation from breeder flock & hatchery monitoring.
The control of *Salmonella* as effected by the introduction of Zoonoses Orders has been taken up by the E.E.C. who have since introduced a directive (92/117/EEC) requiring all member states to collect information on the incidence of specific zoonoses\(^1\) in animals and humans, and to report yearly trends and sources of infection to the E.C. Commission. Minimum standards are set for the monitoring of *Salmonella sps.* in poultry breeding flocks and compound feedstuffs intended for poultry.

The directive also permits the council to introduce specific control measures for other zoonoses including camplylobacterosis, echinocaccosis, listeriosis, rabies, toxoplasmosis and yersiniosis. It became effective in member states on January 1st 1994.

This directive is aimed at the eradication of *Salmonella sps.* from modern poultry production. When flock monitoring samples like faeces are found to be positive, a confirmative investigation has to be carried out by the sampling and examination of internal organs. Flocks are declared positive if zoonotic agents are isolated from internal organs other than those of the digestive tract. The isolation of *S. enteritidis* and *S. typhimurium* from internal organs of breeder flocks means that legal action to prohibit the further production of table or hatchery eggs has to be taken. Further, all hatching eggs in hatcheries found to have originated from positive flocks have to be destroyed.

This new directive will harmonise the rules governing the prevention/control of salmonellosis throughout the E.C. It is a co-ordinated approach to the control of salmonellosis in both food animals and humans, and therefore its introduction must be welcomed.

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\(^1\) Tuberculosis caused by *Mycobacterium bovis*, brucellosis, salmonellosis and trichinosis.
While the E.C. is now making serious attempts to control salmonellosis in humans, Nordic countries such as Sweden and Norway had previously introduced their own control measures which have proved to be extremely successful. This has been mainly achieved due to an excellent animal health status, and to strict import regulations for live animals, meat, and other foodstuffs of animal origin. Indeed, the Swedish Salmonella Control Programme (WHO, 1994) is a model upon which other national programmes could be based. Given the recognised success of this programme, it is important to briefly examine it as it is relevant to the context of this discussion.

4.2.3. Salmonella Control Programme in Sweden

Unlike England and Wales, where, for example, statistics for the years 1990-93 confirm that 88% of human *Salmonella* infections were acquired at home, (PHLS-SVS 1991-1994), in Sweden it is recognised that <20% of reported infections are domestic cases (De Jong and Ardersson, 1993). Like the UK, however, prior to the sharp increase in isolations of *S. enteritidis* from the mid 1980s onwards, the most frequently isolated *Salmonella sps.* from humans was *S. typhimurium*. However, as the number of Swedes who became infected abroad increased, this has since been replaced by *S. enteritidis* as the predominant serotype. This predominance of *S. enteritidis* reported by laboratories in Sweden in recent years has been due to the spread of this microorganism in eggs and chickens in Europe.

A fundamental principle to the control of *Salmonella* in food producing animals in Sweden is that meat products contaminated by any serovar of *Salmonella* are by law (under the Food Act 1971) declared to be unfit for human consumption.
While this might seem a rather harsh or strict interpretation of the definition of 'unfit food', it has proved effective in the control of zoonotic salmonellosis in Sweden. Further, due to this effective control of *Salmonella*, and despite the industrialisation of animal production, both red and white meat can today be claimed to be virtually free from *Salmonella sps.*, with less than than 1% of animals in production being positive for *Salmonella* (WHO, 1994). This is quite similar to contamination rates in the UK where the consensus of expert opinion seems to be that no more than 1% of beef and lamb carcasses, though a higher percentage of pig carcasses carry *Salmonella sps.* when they enter chill (Anon,(D) 1991).

Contamination rates of poultry carcasses is, on the other hand, much greater in the UK than in Sweden, with a recently published international survey\(^1\) showing a zero contamination rate in retail poultry surveyed in Sweden as compared to a 36% rate in the UK.

The following objective, concept and strategies for the control of *Salmonella* in food animals are formulated in Sweden.

**Objective:** Animal Product delivered for human consumption shall be free from *Salmonella*.

**Concept:** Animals delivered for slaughter shall be free from *Salmonella*.

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Strategies:

1. Prevent *Salmonella* contamination of all parts of the production chain.

2. Monitor the production chain at critical points to detect if *Salmonella* contamination occurs.

3. Undertake actions necessary to fulfil the objective of the control when *Salmonella* contamination is detected.

Some of the salient features of the Swedish Salmonella Control Programme in poultry are as follows:

(A) GrandParent/Parent Flocks

1. All imported poultry have to stay in quarantine until cleared by the state veterinary service.

2. Positive flocks, irrespective of *Salmonella* serotype are destroyed.

**Parent Flocks**

(1) Most companies send a substantial number of birds for autopsies/*Salmonella* examination regularly.

(2) Eggs for hatching as parent birds are fumigated, with the use of dirty eggs being prohibited.

**Hatcheries:** Control is based on the control of breeders, with eggs being fumigated before and during the time of brooding, and in the hatcheries.
(B) Control in Layers

Investigations undertaken during 1988-89 due to the international problem with *S. enteritidis* indicated that *Salmonella* was not disseminated by layers in the egg production sector but, however, commercial layers were contaminated to some extent by serotypes other than *S. enteritidis*.

Unlike broiler production, in which the testing for *Salmonella sps.* is mandatory prior to slaughter, control for layers which began in 1990, is voluntary, and involves the bacteriological examination of pooled faecal samples from layers. Within this system of control, 44 flocks have been found to be contaminated up to June 1993 with the predominating serotype found to be *S. livingstone*, (26 flocks).

(C) Control in Broilers

1. All flocks are now compulsorily tested for *Salmonella sps.* at a maximum of two weeks before slaughter.

2. If any *Salmonella sps.* is detected the flock is destroyed.

3. Intensive cleaning/disinfection is undertaken after a broiler house is emptied and particularly after a production unit is found to be contaminated with *Salmonella sps*.

4. The mandatory use of competitive exclusion (CE) in two consecutive flocks raised in a unit, after a *Salmonella* contaminated flock has been identified and destroyed.
Feed: Control is also exercised by the exhaustive testing of imported animal feed, although it is accepted that the import of meat and bone meal still presents a potential pathway for introducing *Salmonella sps.* in Swedish feed factories. Heat treatment/pelleting feed is an effective method of controlling *Salmonella* in feed, but time/temperature ratios during manufacture needs to be monitored carefully.

The Swedish Salmonella Control Programme has been extremely effective, resulting in the complete elimination of *Salmonella sps.* in both grandparent and parent flocks as well as hatcheries concerning broilers.

4.2.4 WHO Consultation Document

The success of the Swedish Salmonella Control Programme has been acknowledged by WHO in its Consultation Document (WHO(A), 1992) on the control of *Salmonella sps.* in poultry. In this is was suggested that the Swedish experience could be put to good effect in its plan for the training of appropriate personnel, teachers etc. to effect Salmonella control on an international basis.

This document based its various recommendations on the principal conclusion of an International Seminar on ‘Salmonella and Salmonellosis’, held in Ploufragan, France in September 1992 in that "a significant reduction of invasive zoonotic *Salmonella* in agriculture is possible, which may effect the source of up to 80% of human salmonellosis, and 50% of all foodborne infections and intoxications". One of its recommendations was that the overall strategy for *Salmonella* control should follow a 'top-down' approach, starting with primary breeding (grandparent) flocks and their inputs.
This document which includes discussion on the experience of other countries such as Canada and Norway in the control of *Salmonella* provided valuable information on modern strategies for the control of *Salmonella* and should be studied by all appropriate personnel involved in control programmes.

4.2.5 **Flair Research Programme into the Control of Zoonoses in Poultry**

Research into the control of zoonoses (Mulder and Kan, 1991) and, in particular, to the control of potentially pathogenic microorganisms in poultry and poultry meat, (Mulder, 1991) has had significant financial backing from the EU since 1989 as part of a programme to reduce the incidence of foodborne infectious diseases.

This research and technological development programme in the field of food science and technology - 'Flair' (Food-linked-agro-industrial research) has, as one of its major objectives, the improvement in food safety and food quality for the consumer (Mulder and Kan, 1991). The aim of the research programme on 'colonisation control' is to disseminate results of research carried out in different institutes in participating countries in this particular field (Mulder, 1991).

Examples of the nature of the research undertaken include:-

1. An examination of the difference in colonisation resistance between experimental and commercial breeds of poultry towards *Campylobacter* bacteria - with, for example, experimental breeds showing greater resistance against colonisation than 4 commercial breeds tested.

2. Trials with spray application of anaerobic bacteria in the hatchery. This particular technique of competitive exclusion (CE) to reduce *Salmonella* shedding in broilers, involved the direct spraying of an oral challenge of anaerobic bacteria.
The trials suggested that this procedure was particularly effective against *S. enteritidis*, although looking at all serotypes generally, no difference in *Salmonella* content was observed by comparing the results from treated and control chickens.

3. An examination of the use of acid treatment of feed for the control of *Salmonella* infections in poultry. Results showed that the addition of formic and proprionic acids to hydrated feeds contaminated with *Salmonella sps.* reduced the incidence of *Salmonella* infections in birds consuming the feed. Similar beneficial effects were not, however, seen in birds consuming dry feed.

Results and conclusions of the research work are presented in a series of volumes published by the 'Centre for poultry research and information services'; Beekbergen, The Netherlands, (Dr. R.W. Mulder - personal communication) and includes research into:-

(a) The attachment of bacteria to the gut.
(b) The role of antibiotics in the control of foodborne pathogens.
(c) Probiotics and pathogenicity.
(d) Hygienic aspects of processed poultry meat.
(e) Detection methods and sampling plans for pathogens in poultry.

There are many sources of *Salmonella sps.* in live poultry, including the parent stock, feed, rearing environment, other animals, and farm personnel. Effective control measures necessitate a thorough analysis of these various sources through which *Salmonella sps.*, and indeed other pathogens such as *Campylobacter sps.* can be introduced.
Controls should include the establishment of a plan of action involving all stages in the poultry production chain, to reduce as far as possible, carriage rates in both the live and carcass birds. The "top down" philosophy of control works on the basis that, if infection is cleared out from the top end, and good hygiene standards are maintained throughout the industry, then infection will be progressively cleared from the whole of the national stock.

However, poultry clear of *Salmonella* sps. entering a slaughter house or processing unit, may be recontaminated via cross-contamination within these premises, so it is essential that product protection procedures are recognised and put in place as an integral part of the overall control system. One vital control procedure suggested for poultry slaughter and processing and introduced in many countries, including the United States, is the application of the Hazard Analysis Critical Control Point (HACCP) approach, the goal of which is "to minimise microbial contamination, and to control bacterial recontamination and/or outgrowth" (USDA(A) 1990). The approach recommended for US slaughter and processing plants covers the principles of HACCP in great detail, including the application of critical control points (CCP), and is to be recommended as a valuable tool in the control of all pathogens in poultry.

The successful utilisation of all HACCP systems is of course dependent on factors such as employee training, records monitoring, quick reaction to non-compliant findings, and periodic verification to ensure proper system operation.

The final line of defence in the control of foodborne infectious disease is the commercial/domestic kitchen. Control at this, the last stage in the food chain, is most often dependant on the extent of awareness by foodworkers in food hygiene, including then having an understanding of the chain of events that can lead to food poisoning. This aspect of control will be discussed at a later stage.
While the incidence of salmonellosis in Ireland appears on the surface to be very low in comparison to the UK, the localised outbreak of *S. enteritidis* 'food poisoning' in the Cork area in 1988/89 suggests that Eire was not totally immune from trends observed in other countries. Many European countries have experienced an increased incidence of infection due to this pathogen from the mid-1980s onwards, and the comparatively very low rates of infection in both poultry and human sources in Ireland says something about our own systems of control at primary level, particularly in the area of poultry production.

Ireland's strict import policy has been one very effective way for controlling *Salmonella* in poultry. Broiler grand parent stock are imported from the UK, with parent 'layers' being introduced from N. Ireland. Quarantine and monitoring measures have been successful in that no breeder flocks have been found to be positive for either *Salmonella enteriditis* or *Salmonella typhimurium* in recent years (M. Sheehan, V.O. Department of Agriculture - personal communication).

The sharp rise in the incidence of infections due to *S. enteritidis* during the 1988/89 period in the Cork area was, however, suspected of being associated with a few local breeder flocks. It is thought that hatching eggs may have accidentally reached the consumer market through confectionary products (via liquid egg) in particular.

Layer flocks have never been found to be positive for *Salmonella enteritidis* or *Salmonella typhimurium* in this country although a total of 5 fresh (table) and liquid egg samples examined in the Food Hygiene Laboratory in Cork in late 1988 were found to be positive.
(Two liquid egg samples were positive for *Salmonella enteriditis*, the other 3 fresh egg samples being positive for either *Salmonella enteriditis* or *Salmonella typhimurium* (Helen Cowman, Senior Lab. Technician, Food Hygiene Lab. Cork - Personal Communication).

The epidemic of *S. enteritidis* infection in the UK, with the subsequent identification of poultry and eggs as the source of infection, prompted a rapid response by the Irish Dept. of Agriculture with the voluntary co-operation of the poultry production industry. The comprehensive "Code of Practice for the Poultry Industry", (Dept. of Agriculture & Food, 1988) announced by the Minister for Agriculture, Mr. Michael O'Kennedy in December 1988, was a response to evidence of increased incidence of human salmonellosis in both the UK and Ireland - the latter being observed in the last quarter of 1988 in the Cork area. This was followed up by the "Salmonella Monitoring Programme", which outlined in a comprehensive fashion a set of guidelines to be followed for the control of *S. enteritidis* and *S. typhimurium* in poultry. This put in place a system of monitoring to ensure compliance with the new EEC zoonoses directive (92/117/EEC).

The Irish Salmonella Monitoring Programme, like the very strict programme operating in Sweden, has been very effective in containing the explosive increase in incidence of human salmonellosis experienced in the UK. It is a blueprint for the Irish poultry industry in their efforts to eradicate or, at least, control *Salmonella sps.* in poultry, and consequently in humans, and contains specific provisions which must be applied by the industry at all levels including:-

- Poultry houses.
- Breeding farms.
- Hatcheries.
• Grower and layer rearing farms.
• Commercial layer farms.
• Egg producers/packing.
• Feed mills.

An obvious consequence of the success of the Salmonella Control Programme in Ireland is the comparatively low levels of *Salmonella sps.* in retail poultry carcasses. Information obtained from both the Dublin and Cork Veterinary laboratories suggest that approx. 10% of retail chickens are contaminated with *Salmonella* organisms. This is confirmed by the international survey\(^1\) into contamination rates in retail chickens which placed Eire 6th best of 14 countries surveyed with a contamination rate of 13%.

The achievements of the voluntarily adopted system here in Ireland of a "seek find, and remove" policy (i.e. *Salmonella* positive flocks) has been acknowledged by the Food Safety Advisory Committee in their report on foodborne salmonellosis (FSAC,(C) 1991).

This committee, which undertook a comprehensive analysis of the whole area of foodborne salmonellosis, its surveillance, epidemiology etc., with particular emphasis on the Irish poultry production sector, suggested 8 control measures to reduce foodborne salmonellosis. The main controls in relation to the poultry industry, such as the prevention of entry of *Salmonella sps.* to commercial flocks, breeding stock etc. have previously been referred to, and other recommendations which relate to commercial catering, the need for hygiene education for food workers etc. will be reviewed at a later stage.

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The committee, however, did recommend legislative control for the poultry industry in the long term, as it feared that complacency might eventually undermine the whole voluntary code that has been so effective.

The committee finally identified proposed legislation in 3 areas which have subsequently been introduced, and which now form an integral part of the overall Salmonella Control Programme viz.:


2. The E.C. Directive (89/437/EEC) on the control of liquid egg/egg products for further use in food manufacture whereby these are now required to be pasteurised before use (Irish Legislation-S.I. 419 of 1992).


This author has identified the control/eradication of *Salmonella sps.* in poultry from the "top down" as the key mechanism to the control of foodborne infectious disease in humans. However, in practice it is very difficult to achieve this ideal in large-scale poultry production, although the success of Nordic countries such as Sweden (WHO, 1994) and indeed Norway (Bredal and Langeland, 1994) in this regard is worth noting. It is, therefore, necessary to examine other strategies and approaches where the control of foodborne diseases can be effectively exercised and it is now proposed to discuss these.
4.3 Other Strategies for the Control of Foodborne Diseases

There have been many discussions down through the years on the most effective way to tackle foodborne disease. Wilson, in the first half of the 1930s suggested an approach to the control of milkborne gastro-enteritis, a very common source of infection at that time (Mossel and Strvijk, 1993).

His strategy for the control of milkborne enteric disease consisted of 3 steps ('Wilson's triad') and could have been equally applied to non-dairy foods of animal origin (e.g. poultry) and consisted of:-

1. Strict hygiene of raw material (To minimise pathogenic load).

2. Thorough pasteurisation ('processing' to kill pathogens).

3. Prevention of adverse post-processing events until ingestion by control of contamination and colonisation (e.g. prevention of cross contamination from raw milk, dirty equipment etc.)

WHO has also identified 3 lines of defence in the fight against foodborne disease (WHO (A), 1989).

1. The rearing of pathogen-free animals for food production - 1st line of defence (already discussed).

2. The use of processing methods such as heat application, freezing, chemical preservation, and irradiation (2nd line).
3. Information and education for both food handlers and consumers (3rd line).

There is no doubting the effectiveness of all the above general approaches to the control of foodborne infectious disease. Unfortunately, surveillance data throughout the world suggests that, despite our improved knowledge on the sources and chain of events that lead to foodborne disease and, despite greater awareness of the importance of food hygiene generally, these approaches, with the exception of milk pasteurisation, have not been successful, as foodborne disease continues to increase.

From the processing perspective, the pasteurisation of milk represented the most significant control measure in the reduction of foodborne disease. Milkborne infectious diseases are very rarely reported in countries where pasteurisation has been introduced, with countries like the Netherlands reporting no outbreaks since the second World War (WHO(A), 1989). The experience of Scotland is significant in that there has been a dramatic reduction in milkborne salmonellosis and campylobacteriosis after the introduction of legislation in 1983\(^1\) effectively banning the sale of raw milk for retail sale (Sharp, 1989).

In Ireland, the production of raw milk for retail sale is insignificant and this writer is unaware of outbreaks being reported in latter times. There may, however, be areas of the country where the retail sale of unpasteurised milk is still permitted which poses a potential risk to public health. In Co. Kerry, for example, the introduction of legislation in 1981\(^2\) prohibited the sale of unpasteurised milk in the 3 urban areas only (Tralee, Killarney & Listowel).


\(^2\) Special Designation Orders 1981 - Made under the Milk and Dairies Act, 1935 (Sec. 33; Subsection 1)
A similar effort to ban the sale of raw milk in the rest of the county was defeated. However, by 1995 there was only one remaining producer selling raw milk in the county and, as this producer has been constantly advised of the potential health risks associated with the consumption of unpasteurised milk, it is expected that production will cease in the very near future (J. D. Pierse, Co. Vet. Officer - personal communication).

Many other food preservation/processing methods such as freezing, drying, canning, use of preservatives etc. have been applied for prolonging the shelf life of foods, as well as providing unfavourable conditions for the survival and multiplication of foodborne pathogens. As these are well recognised processes, it is not necessary to discuss them further here.

However, before discussing other approaches to the control of foodborne disease, it is worth to briefly reflect at this juncture, on a new, if perhaps controversial approach (process) to food safety currently under discussion, the use of ionising radiation (food irradiation). This is an example of a 2nd line of defense.

4.4 **Food Irradiation.**

Two main reasons are put forward for treating foods with ionising radiation:-

(a) To reduce post-harvest losses, e.g. the prevention of sprouting of potatoes and onions, the inhibition of ripening processes, and the killing of endemic insect pests such as the fruit fly in tropical countries.

(b) To reduce the incidence of foodborne diseases by the destruction of pathogenic microorganisms.
The kinds of ionising radiation approved for the irradiation of foods commercially include gamma-rays, normally from a radioactive cobalt 60 source, x-rays with energies up to 5 MeV, and electrons up to 10 MeV, with the former being more penetrating and therefore more effective for the irradiation of thicker food samples, such as whole chicken carcasses (Moseley, 1990).

The case for the irradiation of certain foods like poultry and spices can be argued on both public health and commercial grounds. The real benefits of this process need to be communicated clearly and unambiguously to consumers, who, can then make an informed choice. Several expert committees including JECFI\(^1\) and ACINF\(^2\) have looked at the benefits and safety aspect of the irradiation of foodstuffs and the the views of such committees, together with a commentary on the overall current thinking on the subject, have been reviewed by Moseley (1990).

It can generally be concluded that the irradiation of foods up to an overall average dose of 10Kgy is safe, and presents no toxicological, nutritional or microbiological problems. The UK Government has in fact now implemented the advice of its own advisory Committee (ACINF/1986) and after many years of debate, the Food Safety Act 1990 finally made food irradiation legal in Britain.

The WHO Consultative Group on food irradiation in their 1989 report (WHO(B), 1989) concluded that irradiation was the most appropriate technology to give added assurance of safety of red meats and poultry processed by good manufacture practice (GMP).

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1. JECFI: Joint Expert Committee on Food Irradiation.
2. ACINF: Advisory Committee on Irradiated & Novel Foods.
As regards molluscs, the report stated that preliminary (but unconfirmed) data suggested that irradiation treatments may also reduce significantly the activity of contaminating viruses (which cause most confirmed outbreaks - Table 2.4.1). However, the report specifically singles out poultry as a prime candidate for treatment by irradiation.

A previous WHO Task Force (WHO, 1987) concluded that, as there appeared to be no technology that could guarantee the production of raw foods of animal origin, particularly poultry and pork, free of pathogenic microorganisms such as *Salmonella, Campylobacter, Toxoplasma, & Trichinella*, the irradiation of such foods must be seriously considered. As such, the Task Force recommended that Governments should be encouraged to accept the Codex General Standard for Irradiated Foods, and to implement them towards a broad clearance of irradiated foods. This group further recommended however the preparation of technological guidelines for irradiation in co-operation with experts in food technology, food safety, and radiation processing.

The Food Safety Advisory Committee in Ireland in its 1989 report (FSAC(B), 1989) accepted the potential usefulness of irradiation in reducing foodborne disease caused by *Salmonella, Campylobacter* and *Listeria sps*. They quote the EC draft directive (88/336/EC) which stipulated that irradiated foodstuffs may only be marketed if the package/container bears the information "irradiated" or "treated with Ionising Radiation", and advised that all irradiated ingredients should be shown on the list of ingredients, as the consumer must not be misinformed in relation to this process. The committee finally recommended that:

1. The draft directive (88/336/EC) should be accepted in principle.
2. There should be strict regulation of food irradiation treatments, including the setting up of a licensing authority for irradiation units. In a subsequent report (FSAC(B), 1994), it was recommended that this task should be given to the Radiological Protection Institute of Ireland.

3. Responsibility for statutory controls should fall on the particular government department for the foodstuff in question.

4. For imported irradiated foods, there should be an onus of responsibility on importers to inform the Department of Health of the introduction to this country of such foods, with failure to notify being made a criminal offence.

This report was followed up by a second report in 1994 (FSAC(B), 1994), and again the Committee reviewed expert opinion on the subject of the irradiation of foodstuffs. In this the committee reiterated its previous opinion that the process of irradiation is acceptable and safe when carried out in accordance with Codex Alementarious Commission general standards for irradiated food and the Codex Code of Practice for the operation of radiation facilities in international trade.

The irradiation of certain foodstuffs such as poultry, according to current expert opinion, is the most effective mechanism for the control of foodborne disease. Given the emotive nature of such a process of food decontamination and preservation, its routine introduction to any country will involve patient debate, clarification, and consultation between all relevant parties - most significantly the consumer.
4.5 The Hazard Analysis Critical Control Point (HACCP) Approach to Food Safety

The HACCP approach to food safety has gained considerable approval in recent years. While visual inspections and product analysis are useful food safety measures, what is needed is a method that will ensure that food processes and operations are designed to be safe. Potential hazards are identified so that control measures can be put in place at critical points in a food process. The system has been introduced in different countries since 1970 when food processing specialists in the US adopted the essence of systems which had been developed since the 1930s.

The HACCP system has been studied by several expert groups including the Internation Commission on Microbiological Specifications for Foods (ICMSF 1988) and The Codex Alimentarious Commission (1991) both of whom recommended the system as a method for ensuring the microbiological safety of food. Mitchell (1992) referred to HACCP as both a philosophy and a tool, and describes the concept as a “systematic way of analysing the potential hazards in a food operation, identifying the points in the operation where hazards may occur, and deciding which are critical to consumer safety. (critical control points - CCPs). The CCPs are then monitored and remedial action, specified in advance, is taken if conditions at any CCP are not within safe limits”.

Majewski (1992) sets out the advantages HACCP has over the traditional approach to food safety viz.

(1) HACCP is proactive and preventative - prescribed remedial action can be taken quickly before problems occur.
(2) It may identify hazards that have not been experienced, it it therefore particularly useful when setting up new operations.

(3) It applies to all parts of the process, rather than samples selected for testing (mostly end-product).

(4) It allows resources to be concentrated on critical control points rather than being spread thinly across the whole process.

(5) It involves all levels of staff, not just technical personnel, and it is controlled by those involved directly in production rather than microbiologists in remote laboratories.

The Committee on the Microbiological Safety of Food in the UK in their first report (Anon(C), 1990) took the view that the most effective way of ensuring the microbiological safety of food is to design controls and monitoring requirements into the operation. It recommends that “all food premises should be designed on HACCP principles and operated by properly trained staff using validated control programmes in premises with appropriate hygienic facilities”.

The 3rd World Congress on Foodborne Infections and Intoxications in June 1992 discussed the HACCP approach repeatedly in its various sessions. In its final report it called for the stringent application of HACCP during processing along the food chain with particular reference to high risk foods of animal origin (Anon (E), 1993).
The draft EC proposal for a 'Council Directive on the hygiene of foodstuffs' puts an onus on food business operators to identify any steps in their activities which are critical to ensuring food safety, and to ensuring that adequate safety procedures are identified, implemented, maintained and reviewed using the principles underlining the system of HACCP. These are outlined in the directive as:-

- analysing the potential food hazards in a food business operation.
- identifying the points in those operations where food hazards may occur.
- deciding which of the points identified are critical to food safety (i.e. CCPs).
- identifying and implementing effective control and monitoring procedures at those critical points.
- reviewing the analysis of food hazards, the critical control points and the control and monitoring procedures periodically and whenever the food business operations change.

Devine (1990) in a paper on HACCP referred to a previous WHO conference on "Food Safety in Europe in the 1990s" which examined in particular the potential role of HACCP as an effective tool for food safety control in the future. This Conference recommended that not only should HACCP be considered as a means of improving the efficiency of food inspection services, but also its concept should be provided for in national and international food legislation.

No doubt the draft EC Directive on the hygiene of foodstuffs is a response to this recommendation.
It further looked at the practical application of HACCP, and finally considered that the training of food inspectors (e.g. Environmental Health Officers) and Food Technologists in the concepts and application of HACCP programmes warranted high priority within the WHO/Euro safety programme.

Arguments for the application of HACCP in the food processing and catering industries are substantial. HACCP principles have been applied to two small scale food manufacturing units in North Kerry to assess their value from a food safety perspective. These are:

(a) The manufacture of Farmhouse Cheese.
(b) The manufacture of Lasagne.

These two examples are discussed in detail in Appendix 3 and Appendix 4.

4.6 Measures for the Control of Foodborne Illness at Catering/Food Service Level

"The Kitchen - the final line of defence"

The elimination of pathogens at primary production level is the first line of defense for controlling foodborne disease in humans. While this ideal is very difficult to achieve, the development of modern methods of food processing have, at least, ensured that almost all prepacked processed foods on retail sale nowadays are virtually free of pathogens (2nd line of defense). However, it is mainly in relation to the unhygienic handling and preparation of raw foods at commercial kitchen level that results in the large point-source outbreaks of food poisoning that most often attract media and public attention.
Lack of awareness by foodworkers at this level particularly with regard to the safe handling, storage, preparation, reheating etc. of foods such as poultry, beef products, rice etc. has major public health implications and needs to be addressed. It is now proposed to discuss various aspects of commercial catering where lack of awareness in relation to food hygiene is most often manifested. This will be discussed from the perspective of the major food poisoning organisms under two headings:

4.6.1 Foodborne Intoxications.
4.6.2 Foodborne Infections.

4.6.1 Foodborne Intoxications

Starting with the 3 most common food poisoning (i.e. toxin producing) microorganisms, Clostridium perfringens, Staphylococcus aureus and Bacillus cereus, it is clear that outbreaks occur principally due to a failure on the part of food operators to understand the basic principles underlying the survival and growth (with toxin production) of these pathogenic bacteria or their spores in food.

There have been several documented reports of outbreaks implicating these microorganisms, by the PHLS and the CD(S)U down through the years, and in the UK during the 1980s they were responsible for an average of about 80 outbreaks (and 1600 cases) per year (Galbraith, 1990).

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1 Note: Cl. botulinum, the bacterium that causes botulism, is very seldom, if ever, found to cause food poisoning in routine catering situations. As it is more often associated with food manufacture, including canning and bottling processes, it will not be discussed here.

2 PHLS: Public Health Laboratory Service, Communicable Disease Surveillance Centre, Communicable Disease Reports (weekly).

3 CD(S)U: Communicable Disease (Scotland) Unit. Communicable Disease & Environmental Health in Scotland. (Weekly reports).
1. **Clostridium perfringens**

While this microorganism is often discussed in the context of a "true" food poisoning agent, it is worth noting that, unlike *S. aureus* and *Bacillus sps.*, actual toxin production does not take place in contaminated food, but in the small intestine.

Reports of outbreaks from the PHLS suggest a tendency for this microorganism to have an association with institutional catering, implicating in particular beef joints, beef stews, beef pies, poultry etc. However, reports implicating pork (Dale et al, 1985) and ham (Bakhshi, 1985) have also been described. About 28% of poultry-associated outbreaks have been attributed to *Cl. perfringens* (Anon(C), 1991) which, after *Salmonella sps.*, accounts for most reported outbreaks of food poisoning in England and Wales (Table 2.3.1).

This bacterium should immediately be suspected as a causative agent in outbreaks in which large joints of meat or stews, (especially beef), are incriminated, and stool specimens from patients and leftover food (if available) should be matched for similar serotype, and examined for enterotoxin production.

The most significant contributory factors in outbreaks involving this microorganism include:-

(a) The use of extra large joints of meat.

(b) Inadequate thawing of joints and poultry carcasses.

(c) Inadequate heat penetration to the centre, allowing the survival of bacterial spores.
(d) Prolonged cooling of the joint/carcass/stew at room temperatures, allowing the spores to germinate to the vegetative state.

(e) Inadequate reheating of the meat, allowing the survival of bacterial cells. (A core temperature of at least 70°C must be held for 2 minutes to kill vegetative cells) (Smith et al, 1993).

Specific Control Measures

(a) Ideally use smaller joints.

(b) Meat should be thawed thoroughly before cooking.

(c) Meat should be checked by kitchen staff to make sure that all parts of joints reach at least 70°C or over. The use of digital thermometers are recommended for this purpose, with staff being trained in their proper use.

(d) Meat should be cooled rapidly if not required for immediate use, and refrigerated until required. For stews, the use of shallow trays are recommended, with large joints being halved/quartered. For rapid cooling, blast chillers, or in smaller food establishments, oscillating fans are suggested.

(e) Meats to be reheated must be served piping hot, and not just given a quick warming up.
2. Staphylococcus aureus

This microorganism causes a minority of reported food poisoning outbreaks, accounting for less than 2% of outbreaks in England and Wales during the 1983-87 period (Tranter, 1990).

Outbreaks involving *S. aureus* have implicated various foods, and it is thought that many outbreaks are due to contamination by foodworkers. 20-50% of healthy individuals carry *S. aureus*, with the nose being the main site for multiplication (Tranter, 1990).

Cooked high-protein foods in which staphylococci face no competition from other organisms present the highest risk (e.g. cooked meats). Outbreaks have been described implicating various foods including lasagne (Anon(A) 1985); rice salad (Hayward, 1987) and egg/meat sandwiches (Ritchie, 1984), the former outbreak necessitated invoking the EEC hot line with reported outbreaks in the UK, Luxembourg and Italy. Indeed the importance of effective surveillance for the control of foodborne infectious diseases was well demonstrated by this incident, as the early warning facilitated prompt investigation and early recall of lasagne products in England and Wales (Anon(A), 1985).

**Specific Control Measures**

Hygiene education/awareness on the part of foodworkers, particularly in the area of personal hygiene, is essential for the control of this type of food poisoning. To be more specific:

(a) Foodworkers should not cough or sneeze in the vicinity of pre-cooked foods in particular.
(b) Septic sores on hands should be covered with waterproof dressings.

(c) Foodworkers should not work with food if suffering from colds and flu.

(d) Hair should be kept neatly tied back and covered.

Others:

(e) As milk from mastitic cows is also a potential source of this microorganism, all milk should be pasteurised before consumption, or before being used in a food process (e.g. the manufacture of farmhouse cheese).

(f) As toxin production does not take place at temperatures below $10^\circ$C, vulnerable foods should be stored below this temperature (below $4^\circ$C preferably)

(g) As with controls generally, foods should be prepared as near to the time of consumption as possible.

**Specific Recommendation**

All foodworkers ideally, but specifically those 'key' workers such as chefs, cooks, and catering supervisors should receive instruction on personal hygiene, and on all aspects of the principles of food hygiene. A "Certificate of Competence in Food Hygiene" should be a normal requirement for applications to positions such as these.
The importance of adopting high standards of personal hygiene on the part of all foodworkers is a fundamental prerequisite in the control of food poisoning caused by \textit{S. aureus}.

3. \textbf{Bacillus cereus}

Unlike \textit{S. aureus}, there was an increase in the incidence of food poisoning due to \textit{B. cereus} in terms of both numbers of outbreaks and numbers of cases during the 1980s (Galbraith, 1990). This would appear to be due to an increase in the consumption of ethnic foods in particular, with rice being the most commonly implicated food. Several outbreaks have been described, (Anon(B), 1985); (Dawkins et al, 1984); (Lewis, 1991), with the former report indicating a particular association with Chinese restaurants. Two 'syndromes' of \textit{B. cereus} food poisoning are described - the emetic form, and the diarrhoeal type, with the former being particularly associated with rice dishes.

The main contributory factors to this type of food poisoning include:

1. The pre-cooking of large batches of rice, with the survival of bacterial spores in this starch rich environment.

2. The holding of rice for long periods at room temperature, allowing the germination and multiplication of vegetative cells with the production of a heat stable toxin. (The organism grows readily between 28°C-35°C).

3. The reheating of rice at a later stage, with the preformed toxin being unaffected by this process.
Specific Control Measures

Persons engaged in oriental/ethnic cooking in particular should be given instruction on the risks associated with their traditional way of cooking and holding rice. Such advice should emphasise:

(a) Cooking rice in smaller batches and on the same day of use.

(b) The holding of boiled rice at temperatures outside the danger zone (5°C-63°C).

Pre-cooked rice is often stored at ambient temperatures. This practice should be discouraged as it is essential that the rice should be held above 63°C in a bain-marie or such similar equipment. The aim must be to reduce or eliminate the opportunity for toxin production in the rice, by preventing the germination of spores which usually survive initial cooking.

4.6.2. Foodborne Infections

(1) - Campylobacteriosis

(2) - Salmonellosis

(1) Campylobacteriosis: The control of the two main causes of foodborne infectious diseases in humans, salmonellosis and campylobacteriosis, is very much interlinked, given the particular association between these microorganisms and poultry carriage and the consumption of raw milk.
The control of salmonellosis via the elimination of *Salmonella* sps. in poultry has already been well documented in this thesis.

While research to eliminate *Campylobacter* sps. infection in poultry has not been studied or undertaken with the same vigour and enthusiasm, it is inevitable that such research will take place in the future, given that *Campylobacter* sps. are now the most frequently isolated enteropathogens in the UK (Fig. 2.4.6). Indeed, ongoing research into the control of pathogens generally in poultry (e.g. Mulder, 1991) will ultimately have to take a serious look at the high carriage rate of this pathogen, given that there appears to be a definite relationship between increased chicken consumption (particularly fresh chickens) and increased human camplylobacteriosis in England and Wales. This is demonstrated in Fig. 2.4.9. While it can be argued that the sharp increase in laboratory reports were a result of better awareness and the development of better testing techniques, it is generally accepted that this increase is real (Skirrow, 1990).

Epidemiological data from many countries has confirmed poultry as an important source of infection, (Bolton and Jones, 1989) and according to Skirrow (1990), the single most effective measure to control Campylobacter enteritis is to control infection in broiler chickens. The carriage rate of *Campylobacter* sps. in poultry is thought to be very high. In Eire, 92% of poultry flocks were reported to be positive in one survey, (FSAC,(A)1991, although a recent international survey revealed a contamination rate of 29% in retail carcasses. This compared to a 1% rate in Norway, 10% in Sweden and 41% in the UK.

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Campylobacter enteritis has also been associated with the consumption of raw milk (Sharp, 1989). The Scottish experience confirms the success of the 1993 legislation\(^1\) requiring that milk for retail sale be pasteurised beforehand. In England and Wales, unpasteurised milk has been the food item most commonly reported in outbreaks caused by *Campylobacter sps*. (Skirrow and Benjamin, 1980).

*Campylobacter sps*, unlike *Salmonella sps*. do not multiply on foods because they only grow in a reduced oxygen tension, and at temperatures above 29°C (Anon(C), 1992). This is unusual in that storage of food at ambient temperatures, (unlike all other well recognised 'food poisoning' bacteria), cannot be considered to be a major contributory factor in incidents of foodborne Campylobacter enteritis. In general, the following preventative measures can be applied to control infection due to *Campylobacter sps*.

1. The prevention or reduction of colonisation of broilers.

2. A complete ban on the sale of raw milk, and avoiding the drinking of raw milk.

3. Thorough cooking of poultry, having firstly ensured complete thawing.

4. Avoidance of cross-contamination from poultry carcasses to cooked foods or foods to be eaten without further processing.

5. As birds and domestic pets such as cats and dogs harbour *Campylobacter sps*., it is essential that these animals be kept out of food preparation facilities.

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6. Water supplies to food premises, institutions etc. should be protected against contamination from birds in particular. While supplies to kitchens should be direct from the public main, this may not always be the case.

7. Hygiene education for food handlers should emphasise the risks associated with the handling/preparation of raw poultry in particular.

(2) Salmonellosis: As the epidemiology and control of foodborne salmonellosis has been discussed already in this thesis, it is not necessary to elaborate much further here.

The prevention of foodborne salmonellosis at catering level however is very much related to foodworkers having a clear understanding of the contributory factors to outbreaks of foodborne disease outlined in Tables 2.4.9 - 2.4.11. This is because the vast majority of recorded outbreaks of 'food poisoning' are caused by *Salmonella sps.* and as such, most of these contributory factors would have been recorded following investigations into outbreaks of salmonellosis.

The main control measures can be summarised as follows:-

Foods, particularly poultry should be:

- thoroughly defrosted prior to cooking.
- thoroughly cooked right through to the centre.
- cooked as near to the time of service as possible.
- cooled rapidly and refrigerated if required at a later stage.
- thoroughly reheated and served piping hot.
• during storage and preparation, great care should be taken to avoid cross-contamination from raw carcasses to other foods.

Further:

• all milk should be pasteurised prior to consumption.
• eggs and egg based dishes should be thoroughly heat treated. Where this is not possible, pasteurised egg should be used.
• foodworkers suffering from the symptoms of gastroenteritis should not handle food and should seek medical advice in case they might be excretors of *Salmonella sps*.

• Finally, foodworkers should be made understand that all raw foods of animal origin, particularly poultry, are potential sources of infection and, as such, should be stored, handled, prepared and cooked with this in mind. Therefore, the importance of hygiene education for food workers at all levels of catering cannot be overstressed.

4.7 Control at Retail level - Temperature Control

4.7.1 Retail Chilled Display: The prolonged chilled storage of ready-to-eat foods, from manufacture through distribution to retail display, has attracted increased attention in recent years from a food safety perspective. This is in light of increased knowledge of the survival and growth of foodborne psychotrophs, particularly *Listeria monocytogenes* and *Yersinia enterocolitica*. These pathogens can grow at temperatures previously thought to be very safe for the chilled storage of many high risk foods such as cooked meats, pate, soft cheese, coleslaw, etc.
The relatively long-term chilled storage of many of these foods introduces a time factor, whereby pathogenic psychrotrophs can grow and multiply to potentially infective dose levels for some vulnerable groups (e.g. pregnant women, old people, immunocompromised people, or alcoholics in the case of Listeriosis) (FSAC(A), 1989).

*Y. enterocolitica* can grow slowly between 0-2°C and, in one study a few hundred organisms in raw pork held at 7°C grew to more than 10⁹ cells/ g. within 10 days (Doyle, 1990). *L. monocytogenes* can multiply at temperatures as low as 1°C, and at 4°C, generation times of between 12 and 19 hours have been reported. (Beckers et al, 1989). If one takes a generation time of 18 hours as an example, and a food that originally contained 10 *L. monocytogenes* organisms per gram - the number could increase to 50/g after storage at 4°C for two days, to 150/g (3 days) and 1000/g (5 days) (Lund, 1990). The Food Safety Advisory Committee in their report on Listeria recommended that chilled foods be kept at a maximum temperature of 3°C (FSAC(A), 1989). However, in a survey¹ carried out in North Kerry, only 6(10%) of 61 chilled units displaying ready-to-eat foods were in compliance with this recommendation.

This survey, the results of which are summarised in Table 4.7.1, was carried out between March and November 1992 and involved a total of 41 food premises. Temperature readings were taken in 61 chilled units, the majority being 'serve-overs'. 26 of the premises were groceries, with most of the remainder being cafes, delicatessens, and restaurants.

¹ Survey carried out by the writer, 1992.
Table 4.7.1  Survey of Display Temperatures of Chilled Foods

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<tr>
<th>Temperature storage range</th>
<th>No. of units</th>
<th>% of Total(61)</th>
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<tr>
<td>0°C to ≤5°C</td>
<td>19</td>
<td>31%</td>
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<tr>
<td>&gt;5°C to ≤10°C</td>
<td>31</td>
<td>51%</td>
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<tr>
<td>&gt;10°C to ≤15°C</td>
<td>9</td>
<td>14.8%</td>
</tr>
<tr>
<td>&gt;15°C</td>
<td>2</td>
<td>3.3%</td>
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</table>

Observations/Conclusions

1. Only 10% of units complied with the recommendation of the FSAC for the storage of 'chilled foods' - i.e. ≤3°C.

2. Many of the units did not have a thermometer gauge.

3. Many of the food operators did not know the recommended storage temperatures for the foods displayed, nor knew how to adjust the temperature control button.

4. Where units had a visual display thermometer, the readings were very often lower than that recorded by the digital probe thermometer used in this survey. This is because the actual display temperatures of such units refer to the temperature at the terminal which is located most often underneath the base of the units. This is enclosed, and represents the coldest part of the unit. In other words, the visual display temperature very often did not reflect the real storage temperatures of the foods.
5. Many units exhibited a variation in the display temperature, with the warmest parts being at a point furthest from the outlet for the cold stream. For example, a 5°C differential was noted in one open-top large unit in a modern supermarket.

6. The highest temperature (21.9°C) was recorded in an elaborate shallow-welled carvery/display unit. Many similar-type, highly finished (e.g. marble top, stainless steel and brass finishes) units do not in actual fact provide proper chilled display, and the appearance of such units can be very deceptive. Many of these units are 'chilled' via a stainless steel plate located at the base of the well, or by the upward movement of 'cold' air through a perforated display tray. These would be better served by a cross movement of cold air from one side of the display well to the opposite side, creating a horizontal chill air current.

Recommendations

1. That all chilled units both for commercial and domestic use be provided with built-in thermometers. To facilitate an easy understanding of temperature control, a coding system would be most effective (e.g. the 'blue zone' = 0 to 3°C - correct storage temperature for ready to eat foods like cooked meats, pate, coleslaw, soft cheese).

2. Thermometer gauges should be located so that they are clearly visible.

3. Control buttons should be easily accessible with clear instructions as to how they can be adjusted.
4. Consideration should be given to legislative controls such as the setting of mandatory maximum storage temperatures for certain foods - for example as set out in the Food Hygiene (Amendment) Regulations 1990 in the UK. These regulations introduced a 5°C and an 8°C limit for the chilled storage of 'relevant foods'.

4.7.2 Salad Bars: Salad bars have become very popular in recent years. These are a novel form of the display/self service of salad foods, and have given rise to a certain degree of concern with regard to their suitability from a food safety perspective, viz:

(a) Susceptibility to potential contamination from customers etc.
(b) Effectiveness of temperature control during prolonged periods of display.

With regard to display temperatures, a survey was carried out on 1st June 1994 to ascertain if there was a significant variation in the holding temperatures of 4 different salads over a 7 hour period (Fig. 4.7.1). The salads had been stored in the supermarket coldroom overnight at 3°C and put out on display at approx. 9am. It was noted that all 4 salads increased in temperature during the course of the day, with coleslaw showing the greatest increase (5.2°C/9.30 am - 11.4°C/4.15 pm). The average ambient temperature in the vicinity of the Salad Bar was 16°C. The gradual increase in salad temperatures is of significance in that generation times for *L. monocytogenes* are greatly reduced with an increase in food temperatures. This may have implications where such foods had high initial bacterial loads and were consumed by vulnerable groups.

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1 Food Hygiene (Amendment) Regulations 1990 (S.I. 1431 of 1990)
Variations in the display temperatures of salad bar foods over the course of a day
Recommendations

It is therefore important that salad bars should be properly controlled and such control will involve:

- the covering of display units with a proper sneeze barrier to minimise as far as possible risk of contamination from customers etc.
- the monitoring of display temperatures on a regular basis with a probe thermometer, followed by maintenance of the unit where proper temperature control cannot be achieved.
- salad rotation, whereby old stock is placed on top when replenishing trays.
- the provision of disposable gloves and long handled spoons for customer use.
- in large supermarkets, it would be advisable to assign special responsibility for salad bars to one operator in particular.

Discussion

Temperature control is a vital control mechanism in the transmission of foodborne infectious disease, being particularly important for foods that are sold in a ready-to-eat state. For many 'food poisoning' microorganisms, such as *Listeria monocytogenes*, the infective dose is not known but is believed to vary for different individuals. It is, therefore, most important that initial numbers of the organism in foods are not given the opportunity to multiply to levels that are particularly harmful to vulnerable groups.
However, temperature control should not be regarded as the exclusive responsibility of food producers, retailers etc. as with many householders nowadays doing their major shopping on a weekly basis, the role of the domestic refrigerator has an increasing part to play in the control of pathogens in food. It is therefore essential that the final consumer be included in an overall education programme aimed at reducing the incidence of foodborne disease (See Section on Hygiene Education).

In their report on 'Listeria', the Food Safety Advisory Committee (FSAC(A), 1989) referred to the WHO (1988) report which stated that “total elimination (of *Listeria* sps.) from all food is impractical if not impossible. The critical point is not how to prevent its presence but how to control its survival and growth in order to minimise amounts in food”. It is obvious that proper chilled storage of all foods, but particularly certain high risk foods, is a major critical control point in the control of foodborne disease.

**4.8 Hygiene Education**

It is very important for foodworkers to have a basic understanding of the fundamental principles of food hygiene, including an understanding of the major factors that contribute to incidents of foodborne disease. Human error and ignorance all too often results in food becoming contaminated, and conditions being presented that allow the multiplication of food poisoning bacteria to infective dose levels in food. Such ignorance is highlighted in the discussion with practical examples and recommendations outlined in Section 4.6. It is only through proper education in food hygiene that this ignorance can be dispelled.
An adequately planned and implemented educational programme in food hygiene is a vital component in any co-ordinated and comprehensive campaign for the prevention and control of foodborne disease.

Referring to the control of salmonellosis specifically at all levels of the food production chain, WHO, (1983) recommends the inclusion of the following main elements in any educational programme:

(a) Definition of educational objectives.
(b) Planning of, and evaluation of systems.
(c) Programme preparation, choice of teaching media and methods.
(d) Implementation of the educational programme.
(e) Evaluation.

In these guidelines, WHO recommends a broad based campaign to reach all types of personnel that might have an influence on the control of salmonellosis. This would include farmers producing food animals, animal feed producers, slaughterhouse personnel, laboratory and field staff, and foodworkers in catering establishments etc.

The Environmental Health Officers Association (EHOA) in Ireland has already set about targeting the catering sector with the introduction of its fairly recent 'Basic and Intermediate Courses in Food Hygiene'. These EHOA Certificate Courses deal with various aspects of food hygiene from discussions on the importance of food hygiene, to hygienic procedures for the preparation and storage of foods and related matters such as personal hygiene, temperature control, cleaning procedures, layout/facilities for food premises etc. An introduction to HACCP is also covered, as these courses provide a comprehensive hygiene education programme aimed at all those involved in the handling and preparation of food.
It is hoped that all food businesses will eventually avail of this service. While mandatory training in food hygiene of all those working in the catering sector may be a long way off yet, it is inevitable that this will be a requirement in the future. In fact, such training has already been recommended by the Dick Committee in the UK (MAFF, 1993).

However, it is imperative that all those at supervisory level at least, should have some basic training in food hygiene and when mandatory training is eventually introduced, this group should be targetted first. A Certificate in Proficiency in Food Hygiene should be a standard requirement for advancement to Head Chef and Catering Supervisor levels.

Hygiene education, however, needs to be broadened to include the school system and consumers in the home. Evidence from the UK confirms that the majority of foodborne disease outbreaks actually occur in the home, so it is incumbent on Health Authorities to direct an education programme specifically at this area.

While the Department of Health in Ireland has, in the past, issued advice leaflets targetted at the home, (e.g. on turkey preparation, Listeria etc.), this has been organised for the most part on an 'ad hoc' basis, and not as part of a co-ordinated food hygiene education strategy. This could be rectified by targeting the consumer in a more structured way by highlighting the importance of food hygiene via information leaflets, newspaper articles, t.v. programmes, seminars etc.

An example of an appropriate ‘leaflet’ is that issued by the US Department of Agriculture in 1990 (USDA(B), 1990).
This particular publication was specifically aimed at vulnerable/high risk groups such as senior citizens, pregnant women, and people suffering from immunocompromised illnesses such as AIDS, liver disease etc. It advises on a safe approach to food handling and preparation in simple easy to read manner. Those preparing and handling food in this country should be given priority for training in food hygiene.

A booklet recently issued by the Food and Drink Federation in the UK\(^1\) is another example of the type of concise, easily understood food safety material which could be directed to the consumer in Ireland. The idea of a food safety week has also been tried in the UK since 1993, but the success or otherwise of this initiative has yet to be assessed. However, it is another approach worth considering.

Hygiene receives little attention in our school education programmes, with perhaps the exception of a passing mention in home economics classes. This is particularly disappointing when consideration is given to the fact that a 1970 report on education by the then Food Hygiene Advisory Committee (FHAC, 1970) specifically recommended that training in food hygiene and other aspects of hygiene be taught in all schools.

There should be a much greater effort to introduce the concept of hygiene generally (including food, personal and 'environmental' hygiene) in our schools as part of a broader approach to education. This should also include a programme on the environment aimed at giving students an appreciation for maintaining a clean environment which is vital for good health.

\(^1\) The A-Z of Food Safety
EHOs have been involved with hygiene education in schools for many years on an informal (invitation) basis. However, with a more structured approach to health education in schools, and resources permitting, EHOs could play a more integral role in future education programmes. Writing in another context Galbraith, (1990) wrote that “every infectious disease has an environmental component, most notably in foodborne and waterborne disease and many environmental hazards are related to infection”. This relationship between environment and health can best be appreciated if introduced at an early age through the school system.

4.9 Improved Surveillance of Foodborne Disease

The importance of establishing an active surveillance programme for the collection and collation of vital epidemiological data in relation to foodborne disease has been well highlighted in this thesis so it is not necessary to elaborate much further here. The fact remains that the true incidence of foodborne disease is not known in this country so it is not possible to put into action effective control measures and education programmes aimed at reducing infection in the community.

General practitioners need to be encouraged and indeed reminded of their statutory duty to notify the local Department of Public Health of suspected cases of food poisoning that come to their notice, and this needs to be directed from the top down, i.e. from the Department of Health via local Health Boards (Directors of Public Health). Notification needs to be prompt and complete, so that investigations can be put into action at the earliest opportunity. The early retrieval of food samples and clinical specimens is essential if the source of infection/intoxication is to be confirmed.
The more data available to the Health Authorities on the epidemiology of foodborne disease, the better they are equipped to deal with a sudden rise in incidences in the community as detected via routine surveillance, or from reports of point-source outbreaks. Isolated sporadic cases that come before medical practitioners over a wide geographical area might in fact be part of one outbreak with a single, identifiable source of infection.

One example of this occurred in the UK in 1984 implicating lasagne made from pasta contaminated with *S. aureus* which confirmed the value of effective surveillance (Anon(A), 1985).

The experience of the UK in recent years, specifically in relation to the epidemic of *S. enteritidis PT4* infection, is a constant reminder to all that foodborne disease can have serious public health and economic implications, and, therefore, should not be taken lightly. An improved notification system, together with active surveillance to report on trends in relation to gastrointestinal disease generally and foodborne disease specifically are invaluable tools (control measures) in tackling this potentially serious health problem. Recent proposals by the Food Safety Advisory Committee (FSAC(A), 1994) for the establishment of such a programme is therefore a welcome development.

### 4.10 Water as a source of infection and intoxication

Given that water is a basic ingredient in nearly all food processes, discussion on the control of foodborne diseases would not be fully complete without considering water as a potential source of infection and intoxication.
Many reports and papers have been written confirming water as the source of infection of various gastrointestinal tract infectious agents. These include *Cryptosporidia* (Dick, 1989); *Campylobacter sps.* (Melby *et al.*, 1991); *Giardia* (Neringer *et al.*, 1987); *Giardia* and *Entamoeba histolitica* (Andersson and Jong, 1989) and *Shigella sonnei* (Benton *et al.*, 1989). One outbreak of waterborne gastro-enteritis in Sweden in 1988 effected 41% of the population of a large town which corresponded to almost 11,000 people (Andersson, 1991).

While a report on waterborne disease in Scotland for the 1945-87 period confirmed 'chemical poisoning' as the most frequent causative agent, microbiological agents accounted for 35% of outbreaks and 83% of total cases during this period (Benton *et al.* 1989). This was primarily due to a lack of adequate disinfection, or breakdown in chlorination in public supplies.

A review of outbreaks of waterborne disease in Sweden between 1975-84 confirmed that there were 32 outbreaks affecting 12,000 people during this period (Andersson and Stenstrom 1987). This was somewhat similar to Scotland, in that about 40% of outbreaks (35% Scotland) implicated microbiological agents, with *Campylobacter sps.* (5 outbreaks/2120 cases) and the *Rota virus* (1 outbreak/3200 cases) being responsible for the vast majority of cases where a causative agent was confirmed. Most of these outbreaks were caused by technical hitches, such as back-siphonage of waste water along drainage pipes, broken sewerage systems or sudden pollution of raw water intakes coinciding with malfunction of chlorination.

Probably the largest reported outbreak of waterborne gastroenteritis in this country was the 'Naas incident' in 1991 (Moore, 1992). In this, a public water supply serving approximately 1,500 households in the town of Naas, with an estimated population in excess of 5,000, became grossly contaminated by human sewage.
26 people, the majority of whom were children, were hospitalised, and approximately 4,000 people became ill with symptoms of gastroenteritis. A bored-well used to augment the main regional supply was confirmed to be the source of infection.

The vast majority of public supplies in Ireland are now chlorinated, although there are a significant number of individual householders (including Guest Houses) in rural areas using a private (unchlorinated) supply - usually a bored well.

While the chlorination of water supplies is generally regarded as the most effective measure to ensure a potable supply, recent evidence suggests that chlorination is not effective against some microbiological agents, such as *Cryptosporidia sps*. In 1993 this intestinal parasite was responsible for the biggest outbreak of waterborne illness ever recorded in the US, affecting over 400,000 people in Milwaukee, Wisconsin with over 4,400 needing hospital treatment (Anon, 1994).

A previous outbreak of human cryptosporidiosis in the Oxford and Swindon districts of England in early 1989, subsequently confirmed to be waterborne, resulted in the publishing of two reports. The Dick report (Dick, 1989) examined the events leading up to the outbreak, including a review of the Thames Water Authorities handling of the situation, and the Badenoch report (Anon(F), 1990) looked at the whole area of *Cryptosporidium* in Water Supplies, including the epidemiology of waterborne cryptosporidiosis and an assessment of current water treatment measures, testing techniques etc.
The following is a summary of the main points highlighted in these two reports:-

1. The outbreak highlighted the importance of routine surveillance of gastrointestinal pathogens as the outbreak only came to light after increased isolations of cryptosporidia were identified in a localised area. This coincided with the treated water supply from one particular reservoir serving the Oxford/Swindon area.

2. The standard method for disinfecting treated water supplies by chlorination is ineffective against cryptosporidial oocysts.

3. While the direct examination of water supplies for cryptosporidial oocysts on a routine basis is not necessary, water sources in areas of intensive grazing may be subject to contamination by oocysts after periods of heavy rain. From both a treatment and public health point of view, this needs to be kept in mind.

   It is necessary, therefore, for water authorities (and indeed health authorities) to keep an index of all sources of supply in their area, showing details of treatment, distribution etc. Particular emphasis should be given to the monitoring of susceptible supplies after periods of heavy rainfall. Full information with regard to all supplies should be made available to the local Medical Officer of Health (writer’s comments).

4. Local authorities should be aware of, and have access to facilities for the examination of water for cryptosporidial oocysts, rather than waiting for an outbreak to occur to react.
5. A combination of good flocculation and sand filtration rather than chlorination, provides the most effective barrier so far known for preventing *Cryptosporidium* entering the final treated water.

The microbiological quality of water from the reservoir implicated in the Oxford/Swindon outbreak was satisfactory as measured by normal indicators prior to the outbreak. This calls into question the reliability of these indicators in assessing the potability of water from a public health point of view.

In fact, two fairly recent papers from the US (Anon(G), 1990) indicated that microorganisms such as *Coliforms* and *Legionella* can survive in chlorinated water as they are protected by protozoa into which they have entered. It was suggested that microorganisms within the cysts of protozoa can survive up to 50 mg/l of free chlorine, and that with unexplained bacteriological failures of water supplies, a search should be made for the presence of protozoan cysts.

Studies into human enteroviruses in water also call into question the reliability of the coliform group as indicators of water quality. One study (Slade, 1985) found these viruses in water from a chalk well with a history of excellent bacteriological and organoleptic quality. It was concluded that viruses may occur in underground sources which have consistently proved to be satisfactory in routine monitoring tests for bacteriological indicators of faecal pollution.

The emergence of 'new' foodborne pathogens is an inevitable consequence of the strict surveillance of foodborne diseases. Likewise, new microbiological agents are likely to emerge from time to time to pose a threat to public health. The incident in Swindon and Oxford in England in 1989 is one such example of this.
Recent evidence from the UK suggests that another hazard - that caused by the presence of a toxin associated with cyanobacteria (blue-green algae) in fresh water may be emerging to pose another potential public health problem unless corrective action is taken. Blue-green algae are true bacteria belonging to the class photobacteria and grow rapidly in still waters high in phosphates above 20°C, causing algal blooms (Hunter, 1993). In 1990, the National Rivers Authority in the UK published a report on 'Toxic Blue-Green Algae' which listed outbreaks of cyanobacterial poisoning associated with both recreational and mains water supplies.

The main source of the phosphate is discharge from sewage works and agriculture fertilisers. Deaths of sheep and dogs have been reported in the UK, with similar reports of dog deaths in Ireland (Co. Kerry) - the latter incidents leading investigators to suspect blue-green algae in a local lake as the cause of these deaths (J.D. Pierse, Co. Vet. Officer Kerry - personal communication).

In Kerry, it has been suggested that a major cause of algal bloom may be the overgrazing of sheep, whose numbers have escalated rapidly in recent years with the introduction of EC headage/ewe premium payments (Table 4.10.1).

In a report by Pierse, (Table 4.10.1), this vast increase in the number of sheep in County Kerry has caused a number of problems including upland vegetation erosion. This results in the supplementary feeding of sheep with grain, hay and silage - with the consequent increase in the volume and nutrient content of faeces being washed into lakes and streams. This will also have the effect of increasing the total bacteriological load, including potentially increased levels of *Campylobacter sps.*, *Salmonella sps.* and *Cryptosporidia*. As some of these upland surface lakes may be sources of municipal water supplies careful monitoring is required. In fact, there is anectodal evidence to suggest that one such source in County Kerry was affected by cyanobacteria in the past.
4.10.1 Increase in Sheep Numbers in Kerry 1930-1992

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<td>163,976</td>
<td>166,035</td>
<td>170,487</td>
<td>222,351</td>
<td>217,300</td>
<td>257,000</td>
<td>329,903</td>
<td>455,485</td>
<td>469,385</td>
</tr>
</tbody>
</table>

Source: 'Overgrazing of Sheep in Kerry' - J.D. Pierse, Co. Veterinary Officer (Kerry) - personal communication, 1993.

The effect of toxic algal blooms on drinking water supplies remains to be seen in the future. The Department of the Environment in Eire was sufficiently concerned about the problem that it issued an Advice Circular\(^1\) to all Local Authorities advising them of the situation.

Direct responsibility for the provision of safe water supplies in Eire rests with the Local Authorities. Legislation covering this ‘The European Communities (Quality of water intended for human consumption) Regulations 1988’ has given formal effect in Irish Law to the EC Drinking Water Directive (80/778/EEC). Information on the quality of water supplies throughout the country is now published on a yearly basis by the Department of the Environment. Reports for 1989 and 1990 have already been published with the latter report (DoE, 1992) showing, for example, that over 700 samples taken in that year were contaminated with faecal microorganisms.

The implementation of the new Regulations (1988), with their stricter sampling programmes etc., together with the continued publication of Annual Reports by the Dept. of the Environment, must be considered to be significant control measures to ensure that Irish water supplies are clean and safe to drink.

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\(^1\) Dept. of Environment, Dublin (1993), “Algal Blooms” - Circular No 10/93
Pilot schemes set up in 1991 in various Health Regions of the UK to improve information on waterborne disease nationally have proved to be very useful. These schemes indicated the value of a standardised approach for obtaining data on incidents (including non-infectious incidents) related to water. In Scotland, all 15 Health Boards participated in a monthly reporting scheme. Questionnaires were circulated from the Environmental Health (Scotland) Unit (EH(S)U) to Consultants in Public Health Medicine (CPHM) and Directors of Environmental Health (DEH). A report by Campbell (1992) covering the first four months of the scheme (Sept.-Dec. 1991) stated that 38 incidents were reported during the period, these being grouped into 'Consumption Incidents'; 'Contamination Incidents'; and 'Recreational Incidents'. The scheme has received a very favourable response from participants and is continuing with some modifications. It was felt that the scheme will enhance the close working relationships of all professionals interested in the prevention of public health effects of water contamination.

Epidemiological evidence linking waterborne disease with water consumption is very weak when compared to extensive data available on foodborne disease. However, given the vast network of the water distribution system in developed countries, and the increasing threat to the environment from agricultural and industrial sources in particular, it is inevitable that water courses will continue to become contaminated, with a consequent threat to public health.

More needs to be known about the epidemiology of waterborne disease and water-related hazards. Active surveillance is a vital tool in the provision of this information.
4.11. Summary

The control of foodborne diseases in humans would be a much easier task if pathogens, particularly *Salmonella* *sps.*, could be controlled at primary breeding level, particularly in relation to poultry production. However, as this is very difficult to achieve, other measures need to be taken to eliminate, or at least, reduce the chances of food poisoning microorganisms reaching the consumer.

These strategies have been discussed in some detail in this thesis. Some of these have been described as 2nd and 3rd lines of defence, and include food processing (eg milk pasteurisation, food irradiation etc) and hygiene education for food workers. In fact, it has been pointed out that lack of education or awareness on the part of food workers with regard to the basic principles of food hygiene accounts for most food poisoning incidents that occur at catering/food service level. The value of temperature control during food preparation at commercial kitchen level and during chilled storage in retail food outlets was further demonstrated. According to many expert committees, the HACCP approach is to be recommended as an important food safety 'tool' throughout the food industry. This has been demonstrated with the use of 2 practical examples. Given the fact that water is a basic ingredient in many food processes, it was also relevant to discuss the potential risk to health from this source, either from direct consumption or from contaminated water used in food processes.

Finally, measures for the control of foodborne disease would be greatly supported in countries where reliable microbiological and epidemiological date is available to health authorities. This is because control measures could be better directed and targeted at areas where they can be most effective.
Such important epidemiological data is not available in Eire, and it is recommended that a surveillance programme for foodborne diseases should be formally established in this country. The recent proposal for the establishment of a food surveillance unit within the Department of Health is therefore a welcome development.
Summary of Principal Conclusions and Recommendations

1. At present reliable microbiological and epidemiological data in relation to foodborne disease is not readily available in Eire. The writer therefore, endorses the recommendation of the Food Safety Advisory Committee for a national surveillance programme of foodborne diseases for this country.

2. In the absence of reliable data in relation to foodborne disease in Eire it may be appropriate to rely on trends emerging from the UK, given the similarity of culture, eating patterns etc. However, as has been demonstrated, trends in the epidemiology of foodborne disease can vary from country to country.

3. Epidemiological and statistical data on foodborne disease in Scotland, published annually by the Communicable Disease (Scotland) Unit, is recommended as the model upon which the proposed Irish Surveillance Unit should be based.

4. The vast majority of reported outbreaks of food poisoning throughout Europe in recent years have been caused by *Salmonella sps*. In the UK *Salmonella enteritidis* (in particular phage type 4) is now the most frequently isolated serotype reported by all 3 national surveillances centres.

5. ‘New’ foodborne pathogens are likely to emerge from time to time (eg *L. monocytogenes* and *E. coli 0157* in recent years). As such, surveillance authorities should be ever alert to changes in disease patterns. Further, clinical microbiology and food hygiene laboratories should keep abreast of new diagnostic/isolation techniques.
6. While *Salmonella sps.* are clearly responsible for the vast majority of point-source outbreaks of food poisoning, *Campylobacter sps.* have emerged in recent years to become the most frequently isolated enteropathogens in the UK.

7. All cases of Campylobacteriosis should be informally notified to local health authorities in Eire for follow-up investigations by EHOs. This is desirable so that a clearer picture of the epidemiology of this infection in Eire can be obtained.

8. The main reservoir of infection of *Salmonella* and *Campylobacter sps.* is poultry. As such, the most effective measure to reduce foodborne disease in humans is to control these pathogens in poultry.

9. Contamination levels of *Salmonella sps.* in retail chicken carcasses appears to be much lower in Eire than in the UK. This may account for the apparent lower incidence of human Salmonellosis in Eire.

10. The writer acknowledges the success of the Salmonella Control Programme by the Department of Agriculture and Food in Eire.

11. The writer endorses the recommendations of the Hickey report for a National Surveillance Centre for infectious diseases in Eire.

12. Regional/National variations in the reporting of gastrointestinal infectious diseases may emerge which may not be real. This may be due to an absence of agreed criteria between medical laboratories, with stricter criteria being adopted in some laboratories. While this may be directly related to staffing levels etc., it is recommended that an agreed criteria for the investigation of intestinal tract infections be adopted in Eire.

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13. The writer wishes to acknowledge the work of the Dublin and Cork Surveillance units in the provision of invaluable data in relation to gastrointestinal and other infectious diseases. It is recommended that similar units be established in other regions of the country as these will provide the basis for a national surveillance centre, as recommended by Hickey.

14. *Rotavirus* is clearly the most frequently isolated enteropathogen recorded by the Dublin and Cork surveillance units. This is very different to the UK where *Campylobacter sps.*, followed by *Salmonella sps.* are the predominant infectious agents. This difference may reflect a greater bias in Eire towards more complete investigation of gastroenteritis in children, as reflected by the comparatively high reporting of *Rotavirus* infections (i.e. as a % of total isolates of all enteropathogens). The writer recommends that this difference be examined by those engaged in the surveillance of gastrointestinal infections in Eire.

15. The reporting of food poisoning is greatly under reported in Eire. Medical practitioners need to be reminded of their statutory duty to notify cases of clinical food poisoning (and confirmed Salmonellosis) to their local Health Board. This should be spearheaded at local level by the recently established Public Health Departments within each Health Board.

16. While the control of pathogens in poultry is the most effective way of reducing foodborne disease in humans, in practice this is very difficult to achieve. Other measures (i.e. 2nd and 3rd lines of defence) need to be taken. These include processing controls (eg milk pasteurisation and food irradiation) as well as the implementation of hygiene education programmes for all those engaged in the handling and preparation of food.
17. Certification of proficiency in food hygiene should be a compulsory requirement for all those seeking advancement to supervisory level in the catering sector i.e. head chef, catering supervisor etc. Ideally all foodworkers should be required to attend courses in Food Hygiene. Courses should be specifically directed at all those working with vulnerable groups (e.g. in hospitals, nursing homes, etc.) The home also needs to be targeted.

18. The study of all aspects of 'hygiene' should be introduced in schools. This could be part of a broader approach to education which would include environmental studies.

19. The writer endorses the application of HACCP as an effective food safety measure. All those responsible for quality control, (food technologist etc) and those with a statutory duty in the area of food control (EHOs) should be given adequate training in the practical application of HACCP to food preparation/processing.

20. There should be clear liaison between local authorities and Health Boards in relation to environmental hazards, given the fact that pollution of the environment eventually impinges on public health. This is particularly relevant to water pollution. Local authority laboratories should keep abreast of up to date isolation techniques for microbiological agents such as Cryptosporidia.

21. Active surveillance at regional and national level is the key to the provision of vital statistical and epidemiological data necessary for the effective control of foodborne diseases.
APPENDIX 1

NOTIFIABLE INFECTIOUS DISEASES IN EIRE

Acute Anterior Poliomyelitis
Acute Encephalitis
Acute Viral Meningitis
Anthrax
Bacillary Dysentery
Bacterial Meningitis (including meningococcal septicaemia)
Brucellosis
Cholera
Diphtheria
Food Poisoning (bacterial other than salmonella)
Gastro Enteritis (when contracted by children under 2 years of age)
Infectious Mononucleosis
Infectious Parotitis (mumps)
Influenzal Pneumonia
Legionnaires Disease
Leptospirosis
Malaria
Measles
Ornithosis
Plague
Rabies
Rubella
Salmonellosis (other than typhoid or paratyphoid)
Tetanus
Tuberculosis
Typhoid & paratyphoid
Typhus
Viral Haemorrhagic Diseases
Viral Hepatitis type A
Viral Hepatitis type B
Viral Hepatitis unspecified
Whooping cough
Yellow fever
# APPENDIX 2

## REPORTABLE INFECTIONS IN SCOTLAND

<table>
<thead>
<tr>
<th>Infection Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actinomycoses</td>
</tr>
<tr>
<td>Amoebic Infection</td>
</tr>
<tr>
<td>Atypical Mycobacterial Infection</td>
</tr>
<tr>
<td>Babesiosis</td>
</tr>
<tr>
<td>Bacterial Meningitis (CSF)</td>
</tr>
<tr>
<td>Botulism</td>
</tr>
<tr>
<td>Brucellosis</td>
</tr>
<tr>
<td>Campylobacter Infection</td>
</tr>
<tr>
<td>Chlamydia Psittaci</td>
</tr>
<tr>
<td>Clostridium Infection</td>
</tr>
<tr>
<td>Coxsackie Infection (not CSF)</td>
</tr>
<tr>
<td>Cryptosporidiosis</td>
</tr>
<tr>
<td>Cytomegalovirus</td>
</tr>
<tr>
<td>Echovirus Infection (not CSF)</td>
</tr>
<tr>
<td>Esch. Coli O 157</td>
</tr>
<tr>
<td>Giardiasis</td>
</tr>
<tr>
<td>Human Parvovirus Infection</td>
</tr>
<tr>
<td>Hydatid Disease</td>
</tr>
<tr>
<td>Leprosy</td>
</tr>
<tr>
<td>Listeriosis</td>
</tr>
<tr>
<td>Methicillin-Resistant S.aureus</td>
</tr>
<tr>
<td>Q Fever</td>
</tr>
<tr>
<td>Rotavirus Infection</td>
</tr>
<tr>
<td>Salmonellosis</td>
</tr>
<tr>
<td>Tapeworm Infection</td>
</tr>
<tr>
<td>Toxocariasis</td>
</tr>
<tr>
<td>Viral Meningitis (CSF)</td>
</tr>
<tr>
<td>Yersiniosis</td>
</tr>
</tbody>
</table>
APPENDIX 3

THE APPLICATION OF HACCP TO THE PRODUCTION OF FARMHOUSE CHEESE

1. **Assemble Team**

   No “team” as such, given that this is a small family run business. However, the cheesemaker, her assistant, and the author (EHO) worked together, which for the purposes of this example comprised the “team”.

2. **Describe the product**

   A semi-hard gouda-type cheese, made from unpasteurised cows milk. Varieties include, plain, garlic, nettle and chive-flavoured etc.

3. **Identify intended use of product**

   Wrapped and unwrapped ready-to-eat product to the supplied to retail and catering food premises.

4. **Construct a flow-diagram (See diagram)**

5. **Verification of flow-diagram**

   Discussed at length with cheesemaker. Procedure well established. Deviations from the standard process might include:

   (1) Additions of different flavours if required.
   (2) The cutting and vacuum packing of cheese into smaller (retail) portions.

6. **Listing of Hazards/controls at each step (Table A)**

7. **Application of HACCP decision tree to each step**

   The purpose of this was to identify the critical control points (CCPs) in the process. These are the steps or stages which are critical to the safety of the product or where control can be exercised. The determination of CCPs is fundamental to the whole concept. These need to be carefully defined as it is important to realise that a STEP is not a CCP if it is not possible to affect control at that point. The HACCP decision tree is arrived at by asking a series of questions related to the exercising of preventative or control measures at each “step” in the process (As per table A)
Having listed all the potential hazards associated with the process, and establishing the CCP's where actual control of hazards can be exercised (e.g. microbial contamination) by applying the HACCP decision tree at each step, it is necessary to complete the logic sequence for the application of the HACCP concept (8-12 below).

8. **Established Critical/Target limits for each CCP**

   e.g. - temperature of water added to vat
   - establishment of proper acidity level
   - visual appearance, texture and consistency of curd
   (based on experience of operator)
   - temperature of cold storage

9. **Establish a monitoring system for each CCP**

   In the absence of a “team” as one would have in a large manufacturing operation, all the “monitoring” was carried out by the cheese maker herself. This included making observations and checking the texture/consistency of the curd and final produce - an expertise that can only be acquired with experience. Other “recordable monitoring procedures included the measurement of the acidity level of the curd mix and the temperature of same.

10. **Establish corrective actions**

    Again this is based on the experience of the cheesemaker who is readily able to detect deviations from the normal process. Such defects might include

    (a) Excessive acidity - starter too active
    (b) Acidity too low - Inactive/Inhibited starter
    (c) Bitterness - Starter related/milk composition
    (d) Early gas - poor hygiene. Early in the ripening coliforms convert lactose to gas.
    (e) Late gas - Clostridium sps., later in ripening, convert lactic acid to gas plus undesirable acids.
    (f) Mould spoilage - on surface.
    (g) Excessive/inadequate growth of mould in mould ripened cheese - incorrect flavour body.

    With experienced cheesemakers and a strict monitoring procedure, corrective actions will be taken before the process gets out of control.
Verification

In small home-based operations such as this, very often auditing and reviewing control in the process are only carried out if there is a breakdown in the process, or if a potential hazard arises, e.g. based on a complaint, or where test samples show that undesirable contamination has been introduced into the product.

In this case, the writer undertook an examination of the process by taking samples at different stages in the manufacture of the cheese, which will be discussed in the next section. See TABLE B for results.

Establish record keeping and documentation

As the practical application of the HACCP system was never previously applied to this small home-based operation, detailed records relating to different stages and aspects of the process, e.g. temperature, acidity, whey loss, etc. are not normally recorded.

However, other essential records are kept which relate more to the everyday running of the business, including:

(a) Ingredients used
(b) Date of production
(c) Labelling details
(d) Ripening/cold storage periods, etc.
<table>
<thead>
<tr>
<th>Raw materials:</th>
<th>Raw Milk, Water, Salt, Starter Culture, Rennet</th>
</tr>
</thead>
</table>

**Raw Milk:**
Own cows, 100 gls used. Morning milk only. Milk piped directly to transport tank, and hand-pushed to cheese house approx. 70 metres away.

**Water:**
Private well located in field approx. 100-150m away from farm dwellings, piped directly to sink in cheese house. Not treated.

**Salt:**

**Starter culture:**
Mesophilic aromatic lactic culture (product of Denmark)

**Rennet:**
Vegetable Rennet
<table>
<thead>
<tr>
<th>STEP</th>
<th>COMMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Production of Raw Material</td>
<td>Milk Production (at milk parlour)</td>
</tr>
<tr>
<td>2. Transport to Cheese House</td>
<td>Milk pumped to curd vat (from hand pushed transport tank)</td>
</tr>
<tr>
<td>3. Water added at approx. 70°C</td>
<td>Brings temperature of milk to approx. 30°C</td>
</tr>
<tr>
<td>4. Add &amp; Mix Starter</td>
<td>(Conversion of lactose to lactic acid)</td>
</tr>
<tr>
<td>5. Add &amp; Mix Rennet</td>
<td>(Containing Enzyme “Rennin”)</td>
</tr>
<tr>
<td>6. Setting of “mix”</td>
<td>30 mins. approx. Enzyme “Rennin” coagulates milk with the milk protein casein being “thrown down” to form curd. A rise in acidity. 1st stage of setting.</td>
</tr>
<tr>
<td>7. Cutting the curd</td>
<td>Use of “cutters” - “mix” allowed to stand for 5 mins. approx.</td>
</tr>
<tr>
<td>8. Stirring and “Cooking” of curd</td>
<td>Heating “mix” in double skinned curd vat via water circulation (30 mins). Also mix agitated with whey loss.</td>
</tr>
<tr>
<td>9. “Washing” the curd. Lost whey replaced with water</td>
<td>Water added at 30°C approx. This corresponds to approx. one-third the volume of the tank and replaces the whey loss. (Note: whey affects the acidity of the curd).</td>
</tr>
<tr>
<td>10. ”Milling” the curd</td>
<td>Agitation with further whey loss. Whey pumped to storage tank. Allowed to set.</td>
</tr>
</tbody>
</table>
11. Addition of flavours (if required) e.g. Garlic, chives, nettle
12. "Handling" the curd Pushing or pressing down curd with hands to consolidate, with further whey loss
13. Removal to curd moulds Curds removed to moulds of various sizes, usually with hands
14. Pressing the curd Consolidation of curd in moulds using weights. 1 hour approx.
15. Wrapping the curd/cheese Wrapped in Muslin cloths
16. Pressing the cheese Replacing in moulds and pressing for 24 hours
17. Drying the cheese Removal from moulds and allowed to dry for approx. 1 hour
18. Salting Cheese placed in Brine overnight
20. Ripening Cold room at 12°C/2 weeks.
21. Cold storage Cold room at 7°C until sold.
22. Vacuum Packing Cutting and vac packing if necessary
23. Transportation of product to retail outlets Insulated delivery vehicle used
# TABLE [A]

## LISTING OF HAZARDS AND CONTROLS AT EACH STEP

<table>
<thead>
<tr>
<th>STEP</th>
<th>HAZARDS</th>
<th>PREVENTIVE MEASURES</th>
</tr>
</thead>
</table>
| 1. Contamination of udders with pathogenic microorganisms, e.g. S.aureus, E.colis, in particular | 1. Effective cleaning of teats and udders.  
2. Milking equipment in good working order.  
3. Clean milking environment.  
4. Clean storage of clusters.  
5. Testing for Somatic Cell Count | |
| 2. Contamination of milking equipment, pipelines, etc. | 1. Clean milking environment  
2. Proper use of pipeline rinse and sterilizing solutions.  
3. Proper cleaning/storage of clusters, etc. | |
| 2. Transport of milk to cheese house | Contamination of “clean” milk with microorganisms particularly of the coliform group, but also possibly pathogens | Thorough washing and disinfection of transport tank after each use. |
| 3. Addition of Water to Vat | Possible introduction of pathogens if the private supply used has been contaminated | 1. Test water regularly  
2. Check out sources of possible contamination |
| 6. Setting of mix | Failure to achieve normal acidity level for this stage in the process | Check source/quality of starter.  
Check source/quality of rennet.  
Check quantity of both applied |
<table>
<thead>
<tr>
<th>Step</th>
<th>Task</th>
<th>Precautions/Checks</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.</td>
<td>Washing the curd</td>
<td>As for step 3 above</td>
</tr>
<tr>
<td>12.</td>
<td>Handling the curd</td>
<td>Contamination of curd with pathogens e.g. S. aureus</td>
</tr>
<tr>
<td>13.</td>
<td>Curd removal to moulds</td>
<td>Contamination with S. aureus as direct hand contact often applied</td>
</tr>
<tr>
<td>15.</td>
<td>Wrapping the curd</td>
<td>Contamination with S. aureus from direct hand contact</td>
</tr>
<tr>
<td>16.</td>
<td>Pressing the cheese</td>
<td>Contamination of curd from dirty mould and press</td>
</tr>
<tr>
<td>20.</td>
<td>Ripening the cheese</td>
<td>Survival/growth of pathogens if temperature too high</td>
</tr>
<tr>
<td>21.</td>
<td>Cold Storage</td>
<td>Survival/growth of pathogens if present (e.g. Listeria sps.)</td>
</tr>
<tr>
<td>22.</td>
<td>Cutting/Vacuum packing</td>
<td>Contamination of cheese from hands, knives, work surface, etc.</td>
</tr>
<tr>
<td>23.</td>
<td>Transportation</td>
<td>Survival/growth of pathogens if present - particularly over long journeys during the summer period.</td>
</tr>
</tbody>
</table>

**Water quality**
- Check acidity also (whey loss)

**Thorough washing of hands beforehand. Use of sterile gloves.**
- Wash hands thoroughly. Use of sterile gloves or a sterile appliance.

**Contamination of curd with pathogens e.g. S. aureus**
- Contamination with S. aureus as direct hand contact often applied
- Contamination with S. aureus from direct hand contact

**Wash hand thoroughly. Use of sterile gloves.**
- Wash hands thoroughly. Use of sterile gloves.

**Clean and sterilize moulds and wooden presses after each use.**
- Clean and sterilize moulds and wooden presses after each use.

**Check and adjust temperature of cold room if necessary**
- Check and adjust temperature of cold room if necessary
- Check and adjust temp. if too high

**Survival/growth of pathogens if present (e.g. Listeria sps.)**
- Survival/growth of pathogens if present (e.g. Listeria sps.)

**Wash hands thoroughly. Sterilize knife before use. Clean/sterilize cutting surface. Use of insulated containers. Use of refrigerated vehicle. Provide thermometer in vehicle.**
<table>
<thead>
<tr>
<th>DATE</th>
<th>TIME</th>
<th>SAMPLE</th>
<th>STAGE/STEP</th>
<th>E. COLI</th>
<th>RESULTS</th>
<th>SALMONELLA/</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>30.3.93</td>
<td>9.00 am</td>
<td>Fresh mornings milk</td>
<td>1</td>
<td>0/3 Plate Count 100/g</td>
<td>Neg.</td>
<td>Neg.</td>
<td>Taken direct from Milking pipeline. Milk from 6 cows used for cheesemaking.</td>
</tr>
<tr>
<td></td>
<td>10.15 am</td>
<td>Curd(SampleA)</td>
<td>6</td>
<td>170/g</td>
<td>0</td>
<td>Neg.</td>
<td>1st stage of setting.</td>
</tr>
<tr>
<td></td>
<td>2.00 pm</td>
<td>Curd(SampleB)</td>
<td>after S10</td>
<td>20/g</td>
<td>Pos.</td>
<td>Neg.</td>
<td>Curd Cottage cheese consistency.</td>
</tr>
<tr>
<td></td>
<td>10.00 am</td>
<td>Water</td>
<td>3</td>
<td>0 (Coliform 1/g)</td>
<td>-</td>
<td>Neg.</td>
<td>Private well. Water untreated.</td>
</tr>
<tr>
<td>27.4.93</td>
<td>4.00 pm</td>
<td>Cheese</td>
<td>One sample only tested for E. coli All 3 samples tested for pathogens</td>
<td>unct/g (S.A.)</td>
<td>S.A. 3200/g S.B. 4000/g S.C. 4500/g</td>
<td>All Neg.</td>
<td>Finished product taken from cold room at 7°C. Same batch. Production date: 30.3.93</td>
</tr>
<tr>
<td>29.6.93</td>
<td>4.00 pm</td>
<td>Cheese</td>
<td>Finished Product</td>
<td>S.1. 160/g S.2. 80/g S.3. 80/g S.4. 30/g S.5. 160/g</td>
<td>All o/g</td>
<td>All Neg.</td>
<td>Same batch. Cold Room at 7°C Production date: 30.3.93</td>
</tr>
</tbody>
</table>
COMMENT

1. All samples were taken the day before testing, and kept at 5°C overnight.

2. **E. coli** readings in the curd samples taken on 30/3/93 were surprising as one would have expected a much higher reading in sample B given the holding temperature of mix (35°C approx.) and the time lapse between the taking of the samples (approx. 4 hours). However this may possibly be explained by:
   (a) Bacteria not being homogeneously distributed in batches of foods, leading to a variation in counts in different samples.
   (b) **E. coli** being inhibited and dying off with time due to competition from other bacteria, including starter.

3. Only one sample of milk was taken, which did not contain **E. coli**. 100 gls. of milk were used in the process, and given the uncountable reading for **E. coli** in a sample of cheese taken on 27th April, 1993, it is likely that more extensive sampling of the raw milk would have revealed contamination with **E. coli**. However, **E. coli** may have been introduced from environmental contamination of equipment, utensils, handling procedures, etc.

4. Only one of the two curd samples taken on 30th March, 1993 was positive for **S. aureus** (Sample B). Again this may have more to do with the heterogeneous distribution of bacteria within the same batch of a food, or their numbers were not at sufficiently high levels to be detectable in the first curd sample.

5. All samples of cheese taken on 27th April, 1993, showed high levels of **S. aureus**, as obviously growth/multiplication of this potentially food poisoning microorganism had taken place throughout the batch. **S. aureus** could easily have been introduced in the cheese house, e.g. by the operator during the manufacturing process (hands, equipment, etc.) Alternatively it may have been present in the original milk used, although a milk sample taken on 30th March, 1993 did not reveal contamination with this microorganism.

6. Although only one sample of cheese was examined for **E. coli** on the 27th April, 1993 the result revealed alarmingly high levels (unct/g). The five samples taken on the 29th June, 1993, while all showing **E. coli**, the counts were much lower that the previous sample, confirming a significant "die-off" rate during cold storage.

7. Similarly with **S. aureus**, none of the five samples taken on the 29th June, 1993 revealed contamination with the microorganism, although all three of the previous samples (27/4/93) showed heavy contamination levels. Again, this confirmed that, as was the case with **E. coli**, there was a significant, and eventually complete "die-off" of these potential pathogens during the storage of this high acid cheese.
8. The water used in the process on this occasion was free of *E. coli*, although a subsequent sample (29/6/93) showed a count of 36/g in the water taken direct from the cheesehouse tap. However a second sample taken on the latter date was found to be negative after being heated in the usual manner before being used in the cheese manufacturing process. This indicated that for this particular process, the heating of the water to the correct temperature is a CCP for this operation and requires continued monitoring.

9. While raw milk is normally used in most farmhouse cheese manufacturing processes it is important to realise that from a public health point of view, this present a potential hazard, not so much with the type of cheese outlined and discussed in this example, but in the high moisture/low acid varieties such as Camembert and brie. It is also well to remember the recommendation of the Food Safety Advisory Committee (FSAC, 1990) in their report on farmhouse cheese manufacturing. In the interest of public health, this expert committee recommend that milk used in the manufacture of farmhouse cheese should be pasteurised beforehand. However, given the claims made for the demand for farmhouse cheeses, and because of the availability of imported farmhouse cheeses made from raw milk, they concluded that consideration should be given to allowing the use of unpasteurised milk, provided there was a strict adherence to the code of hygiene practice for soft and fresh cheeses recommended by the IDF (International Dairy Federation) in 1939.

For sampling purposes, the FSAC recommended the use of the “3-sample plan”, and it is worth noting that under this sampling procedure the results of the samples taken for bacteriological examination on 29th June, 1993 would be deemed to be satisfactory as only 2 samples exceeded the “m” figure of 100 *E. coli*/g, with no pathogens being detected (Note: Criteria based on IDF code of practice for manufacture of soft cheese using raw milk). It can be argued that too many CCPs can overcomplicate a system or process, and therefore one should concentrate on the more important (critical) areas where control can be exercised. For example in the specific process discussed here, it could firstly be said that such low moisture-high acid cheeses are not high risk foods, and the HACCP approach would be better applied to the manufacture of soft (high risk) cheeses. This would be to misunderstand the concept - the HACCP system is proactive in that it seeks to identify and minimize hazards before they are likely to arise, and therefore its concept should be applied to all food manufacturing operations as a means of reducing if not eliminating the risk to public health. It is important to remember also that while microbiological hazards normally present the greatest danger in food operations, other hazards such as those of a physical or chemical nature are also likely to arise in many food manufacturing operations, and therefore need to be taken into account.

Simplification of the HACCP system would entail taking a closer look at the CCPs, and deciding on which steps in a process are critical to the safe manufacturing or preparation of food. For example two types of CCPs are recognised.

1. **A CCP** - assures control of a hazard i.e. a hazard can be completely controlled, e.g. deciding on the pasteurisation of milk (correct time/temperature) before being used for the manufacture of farm house cheese.
(2) A CCP2 - will minimize but cannot assure control of a hazard (i.e. hazard more difficult to control) e.g. storage and distribution of cheese.

In this example, many of the CCPs are actually CCP2s - maximum control could be exercised if the milk was pasteurized in the first instance. However establishing CCP2's would also be necessary as contamination could also be introduced during other steps in the process where control would need to be exercised. While the acidity/moisture levels would always present an inhibitory factor to microorganisms in semi-hard or hard cheeses, this safety factor would not be available in the manufacture of soft cheeses.
APPENDIX 4

THE APPLICATION OF HACCP TO THE PRODUCTION OF LASAGNE

1. **Assemble Team**:
   "Team comprised of 3 foodworkers (including proprietor) and the environmental health officer (Author). This is a small scale food business, operated from an "advance" unit rented from a government agency.

2. **Describe Product**:
   "Homemade Lasagne", made with minced beef, pasta, tomato puree, carrots, onions, mushrooms, flour, margarine, milk, cheese, herbs and spices.

3. **Identify intended use of product**.
   Pre-cooked product sold in cellophane wrapped trays of approx. 200g weight. to be reheated in a microwave (2/3 mins.) or in a preheated oven (200°C/20 mins.)

4. **Construct a flow diagram** - See diagram

5. **Verification of flow diagram**
   Every stage in the process discussed with and verified by the proprietor.

6. **Listing of hazards and controls at each step**.
   See table C outlining these.

7. **Application of HACCP decision tree to each step**.
   Principles as per example 1 - HACCP/Cheese manufacture. CCPs determined by close examination of TABLE C.

8. **Establish critical/target limits for each step**.
   - Cooking of minced meat to proper core temperature
     (red meat turns brown at approx. 70°C)
   - Temperature of cold room set at 2-3°C.
   - Temperature of insulated delivery vehicle to be regularly monitored. Target < 10°C (Note: shorthaul deliveries only).

9. **Establish a monitoring system for each CCP**.
   In this very small scale processing unit, there was very little formal monitoring as such. Experience of proprietor (with advice from EHO) sufficient to determine acceptability of process. However, a digital probe thermometer available to occasionally monitor temperature of:-
   (a) Cold Room
   (b) Core temperature of food during processing, or finished product when required.
   (c) Delivery vehicle.
10. **Establish corrective actions.**
   In this process, this mainly involved contacting cold storage service personnel immediately where unacceptable fluctuations in cold room temperature occurred. This was a very rare occurrence.

11. **Verification.**
   Process including corrective systems discussed with EHO (author) during routine inspections. Advice given where necessary. Process reviewed where high bacterial counts/pathogens isolated - usually as a result of end product sampling at retail level.

12. **Establish record keeping documentation.**
   In this operation, records normally kept include:
   (a) Date of production/"use by" date.
   (b) EHO advised to also record temperature of cold room and delivery vehicle more frequently, being particularly vigilant during the summer months.
Ingredients used in production of Lasagne

Meat sauce: Minced beef, onions, mushrooms, carrots, tomato puree.

White sauce: Flour, margarine, milk

“Lasagne”: Pasta (lasagne) sheets, water

Cheese: Grated - purchased in sealed plastic bags from outside source.

Parsley: Chopped sprigs
Step 1
Cook lasagne “sheets” (pasta)

Step 2
Allow to cool 2 hours

Step 3
Cook minced meat for approx. 10-15 mins.

Step 4
Preparation of vegetables (onions, carrots, mushrooms) add and mix

Step 5
Add tomato puree, water and cornflour and mix

Step 6
Simmer for 25 mins.

Step 7
Transfer to individual trays (24)

Step 8
Preparation of white sauce (flour, margarine, milk) and add.

Step 9
Top with shredded cheese

Step 10
Sprinkle with sprigs of parsley.

Step 11
Allow to cool in open trays for 45-60 mins.

Step 12
Wrapping of trays with cellophane

Step 13
Storage in cold room overnight at <4°C

Step 14
Delivery to retail outlets via insulated delivery van
<table>
<thead>
<tr>
<th>STEP</th>
<th>HAZARD</th>
<th>PREVENTATIVE MEASURES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Cooking of Lasagne</td>
<td>Survival of bacterial enterotoxins (e.g. S.aureus) and/or bacterial spores (e.g. B.cereus).</td>
<td>Thorough cooking of lasagne “sheets”.</td>
</tr>
<tr>
<td>2. Cooling of Lasagne</td>
<td>Germination of bacterial spores with multiplication of vegetative cells.</td>
<td>Avoid a prolonged cooling period.</td>
</tr>
<tr>
<td>3. Cooking of minced meat</td>
<td>Survival of bacterial spores (e.g. Cl.perfringens), or in the case of poor heat penetration, E. coli 0157.</td>
<td>Cook thoroughly throughout.</td>
</tr>
<tr>
<td>5. Addition of water, tomato puree and cornflour.</td>
<td>Cornflour/tomato puree: unlikely hazard. Water: Introduction of pathogens via contaminated source (e.g. sewage/slurry).</td>
<td>Use chlorinated supply. Ensure that supply is direct. Have tested occasionally.</td>
</tr>
<tr>
<td>7. Transfer to trays.</td>
<td>Contamination introduced from environmental sources and from food operators.</td>
<td>- Strict personal hygiene e.g. use of hair nets disposable gloves, etc.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Use of clean utensils only.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Proper storage of trays.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Pest-proofing of premises.</td>
</tr>
<tr>
<td>STEP</td>
<td>HAZARD</td>
<td>PREVENTIVE MEASURES</td>
</tr>
<tr>
<td>------</td>
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</tr>
<tr>
<td>8. Preparation and addition of white sauce.</td>
<td>Introduction of milkborne pathogens, e.g. salmonella sps, campylobacter sps. and S.aureus.</td>
<td>Use pasteurised milk only.</td>
</tr>
<tr>
<td>11. Cooling at ambient temperature for 1 hour.</td>
<td>Multiplication of CL.perfringens as a result of germination of spores during prolonged cooling. Multiplication of pathogens e.g. L.monocytogenes introduced from environmental/personal sources. Multiplication of other food poisoning bacteria introduced post processing (e.g. from the cheese/parsley).</td>
<td>Blast chilling of trays very soon after filling.</td>
</tr>
<tr>
<td>12. Wrapping with celophane.</td>
<td>Introduction of contamination via dirty celophane and from hands of food operators.</td>
<td>Avoid contamination of exposed celophane film from work surfaces, hands, etc. Care to be taken not to tear film when wrapping.</td>
</tr>
<tr>
<td>13. Cold storage.</td>
<td>Multiplication of L.monocytogenes</td>
<td>Store 0-3°C</td>
</tr>
<tr>
<td>TIME</td>
<td>SAMPLE</td>
<td>STEP</td>
</tr>
<tr>
<td>----------</td>
<td>---------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>11.00 am</td>
<td>Raw minced Beef</td>
<td>-</td>
</tr>
<tr>
<td>11.30 am</td>
<td>Meat Sauce (Cooked)</td>
<td>3-6</td>
</tr>
<tr>
<td>11.30 am</td>
<td>White sauce (cooked)</td>
<td>8</td>
</tr>
<tr>
<td>11.50 am</td>
<td>Shredded Processed Cheese</td>
<td>9</td>
</tr>
<tr>
<td>11.50 am</td>
<td>Parsley (washed)</td>
<td>10</td>
</tr>
<tr>
<td>11.40 am</td>
<td>Lasagne sheets/pasta (Cooked)</td>
<td>1-2</td>
</tr>
<tr>
<td>11.55 am</td>
<td>Finished lasagne (without cheese and parsley topping)</td>
<td>After S.8</td>
</tr>
<tr>
<td>11.55 am</td>
<td>Finished lasagne</td>
<td>After S.12</td>
</tr>
<tr>
<td>11.00 am</td>
<td>Can of tomato puree</td>
<td>/</td>
</tr>
<tr>
<td>11.30 am</td>
<td>Water supply</td>
<td>During process</td>
</tr>
</tbody>
</table>
COMMENT

This process took approx. one hour to complete, although advanced cooking/cooling of the lasagne sheets in a large pot had taken place before preparation of the meat sauce. The vegetables had also been prepared in advance.

(1) The main “steps” where control could be exercised revolved around the cooking of the meat sauce (CCPI’s). Minced meat is always likely to harbour potentially harmful bacteria such as L. monocytogenes (as in this example), Clostridium perfringens, Staphylococcus aureus, with E. coli 0157 being increasingly reported in recent years in incidents of food poisoning implicating minced/burger meat.

Thorough cooking of the meat sauce will destroy these pathogens, although the spores of Clostridium perfringens may survive the cooking process (large batches/inadequate heat penetration) with multiplication of vegetative microorganisms during a prolonged cooling period.

(2) The other main components of the product, lasagne “sheets” (pasta) and white sauce should not present any source of contamination, given the nature of these “ingredients” and the preparation involved. Food preparation at these “steps” however needs to be considered from a HACCP perspective as all aspects of the process should be examined for control purposes.

As can be seen from the results, the white sauce was found to be bacteriologically sterile, although the lasagne sheets did show an “uncountable” plate count at 30°C.

(3) Given the process as outlined, the most likely sources of contamination in the final product could in effect occur at the post processing stage i.e. from the addition of:-

(a) Parsley:- Raw vegetables and herbs introduced post processing to a food product (as in this case) should be regarded as a potential source of contamination from both natural soil bacteria and bacteria which might be present as a result of the application of natural fertilizer. While the results showed the absence of pathogens, the very high faecal coliform count did suggest that pathogens could be introduced via this “ingredient” (garnish). This was despite pre-washing of the herb. (Interestingly a separate sample from the same “batch” examined after immersion in a hypochlorite solution (Milton) according to manufacturers instructions, was found to be bacteriologically sterile, suggesting that this is a method the proprietor could employ to minimise contamination in the finished product.)

(b) Shredded Cheese:- Bought in from outside source in sealed plastic bags. In the sample taken, there was evidence of mild contamination with faecal coliforms, with the plate counts being excessively high. A previous sample of this cheese revealed very high levels of faecal coliform which necessitated follow-up investigations of hygiene standards in this particular plant.
Despite indications that processing methods employed in this unit could give rise to contamination in the post processing stage, the bacteriological quality of the finished lasagne (after step 10) was very good. In fact, the quality of another sample taken before Step 9 (i.e. without parsley and cheese) was almost as good. However, previous samples of this product taken at retail level revealed excessive levels of faecal coliforms, and the presence of L. monocytogenes.

The most surprising result was in fact was the presence of E. coli contamination in the water supply in the Unit (5/100 mls.), as the supply came from a public/chlorinated source. It was initially suspected that the supply to the unit might have been indirect (i.e. via the storage tank in the attic), but follow-up investigations by the writer and a plumber confirmed that the mains supply came direct to the sinks. Other samples taken in the town about that time proved to be negative. A follow-up sample taken at the unit also proved to be satisfactory.

The can of tomato puree, as would be expected was bacteriologically sterile.

In summary, the key aspects of this particular process where microbiological hazards are likely to exist, and where control can be exercised include:

(a) The preparation of the meat sauce - thorough heat penetration.
(b) The filling of the tray - care to be taken to avoid the introduction of contamination at this stage.
(c) Cooling of the trays - use of blast chiller instead of prolonged cooling at ambient temperatures.
(d) Topping with shredded cheese - use of disposable sterile gloves.
(e) Thorough washing of the parsley (use of hypochlorite if necessary).
(f) Temperature control: i.e. cold room and delivery vehicle.

However in determining CPPIs and CCP2s, it is likely that those steps that are absolutely critical, or where control of the main microbiological hazards can be assured revolve around the preparation of the meat sauce. (Steps 3-6)
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