Advisory Committee on the Microbiological Safety of Food

Second Report on *Campylobacter*

Advises the Food Standards Agency on the
Microbiological Safety of Food

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Second Report on *Campylobacter*: Memorandum of Research

Summary

Introduction

1. This is the Second Report of the Advisory Committee on the Microbiological Safety of Food (ACMSF) dealing with *Campylobacter*. The Committee issued an Interim Report in 1993.[1] This Second Report comes against the background of a Food Standards Agency (FSA) target of reducing the incidence of foodborne disease by 20% by April 2006.[2] *Campylobacter* is currently the biggest identified cause of bacterial infectious intestinal disease in the United Kingdom. A significant reduction in human campylobacteriosis would therefore make a very important contribution to achieving the Agency's target for reducing foodborne disease.

2. Our Interim Report[1] identified strong circumstantial evidence suggesting poultry as a major source of human *Campylobacter* infection, transmission being either directly through consumption of undercooked chicken or by cross-contamination of other foods in the kitchen. A 2001 FSA survey of raw fresh and frozen chicken purchased at retail in the UK found 50% of all samples tested were contaminated with *Campylobacter* (see Chapter 1).

3. We decided in 2000 to set up a Working Group to identify any important gaps and omissions in action taken since 1993 to reduce *Campylobacter* in food and food sources, and in the knowledge base. Our objective was to develop advice to help the FSA in evolving its strategy for reducing the incidence of foodborne *Campylobacter* infection in humans. Conscious that the FSA's foodborne disease target was time-bound, we decided to feed our advice into the Agency as and when it became available, rather than waiting until this Report had been finalised and adopted by the full Committee for submission to the FSA.

Basis for our approach and conclusions

4. As regards the structure of this Report, we have resisted the temptation to chronicle all of the relevant scientific and technical advances which have taken place since our Interim Report was published. Instead, we have pointed to key developments and where further information may be found.

5. The various assessments made and the conclusions we have reached reflect, in large measure, the evidence, oral and written, drawn from the scientific community and industry, and from the scientific literature. The recommendations reflect our conviction that there is an important association between poultry meat and human *Campylobacter* infection, and our judgement of the practical steps which we believe can be taken across the food chain to reduce the burden of human campylobacteriosis.

6. More than a million tonnes of chicken meat is produced in the UK annually. Some 96% of this is from intensive production systems. This is one reason why the advice in this Report on measures to prevent contamination of chicken meat in primary production is centred on intensively-reared birds. Another is that we believe that robust biosecurity regimes are more easily applied in the intensive production setting. However, we recognise that extensive production (free range and organic) is now a significant, albeit relatively minor, feature of the market and we touch upon this in Chapter 4 and Annex E.

What we have concluded about *Campylobacter*

7. The first tranche of advice which we sent the FSA, in September 2002, was about on-farm control measures against *Campylobacter* spp. in chickens.[3] An updated version of that advice constitutes Chapter 4 of this Report. We tackled the question of the control of *Campylobacter* in chickens first because, although we are not 100% certain of the extent of the association of poultry meat with human illness, we are satisfied that poultry plays a significant role in the causal chain of events leading to human foodborne illness. The human infectious dose is thought to be quite low and a single live chicken could potentially carry millions of human infectious doses of *Campylobacter*. Of course, the microbiological loading may be significantly reduced during slaughter and processing, but poultry meat will still pose a heavy challenge to hygienic measures along the supply chain right into kitchens, both in the home and in catering establishments.

8. Given the prevalence of *Campylobacter* in poultry, and knowing how easily pathogens can persist and spread in the domestic and catering environments, we believe that reducing the level of the organism in poultry meat is likely to make a significant contribution to the battle against human foodborne illness. Any reduction in the number of *Campylobacter* cells reaching the kitchen would enhance the effectiveness of normal hygiene measures. We recognise that there are other routes of infection and that both food and non-food exposure pathways are important. There is also the association between travel and human campylobacteriosis in the UK. We have given the non-poultry meat exposure pathways some attention in this Report. However, we believe that the existence of these pathways should not distract from the need to reduce carriage levels in broiler chickens which constitute a very popular food item.

9. We have also afforded poultry particular attention – and made control in chickens a focus for early advice to the FSA[3] – because we believe that reducing *Campylobacter* carriage by housed broilers is now a practical proposition where previously many thought it impossible (see Chapter 4, paragraph 4.4). We do not minimise the difficulties involved, nor the serious pressures in what is a competitive and price-sensitive industry where increased import penetration is a continuing threat. But the evidence

collected by our *Campylobacter* Working Group during the course of its deliberations suggests that, on some farms, the problem is already being successfully addressed. It is also worth bearing in mind that this success has not been dependent on great scientific innovation but has come about through rigorous attention to detail, particularly in relation to robust biosecurity and through high standards of stockmanship. Moreover, we believe that these measures not only yield benefits in relation to *Campylobacter* but are also reflected in overall improvements in poultry health. As such, we believe that they are likely to be self-rewarding.

10. *Campylobacter* infection of chickens is not a problem peculiar to the UK. It is an issue of concern to a number of other countries, some of which were visited by the Working Group (see Chapter 5). The general approach in these countries to tackling the problem seems consistent with our own.

11. It bears restating that we firmly believe that practical on-farm measures are available now for controlling *Campylobacter* spp. in chickens. It is very important that industry grasps the nettle of controlling *Campylobacter* in primary production and processing because we do not regard it as reasonable to expect the problem only to be addressed further along the supply chain by consumers and commercial food handlers. Of course, consumers and commercial food handlers should take all possible steps through safe handling, thorough cooking and the avoidance of cross-contamination to eliminate any potential threat from *Campylobacter*. But hygiene measures should be supplemental to, and not a substitute for, addressing the *Campylobacter* problem at the beginning of the production, processing and supply chain.

12. When it surveyed levels of *Campylobacter* in retail chicken in the UK in 2001, the Food Standards Agency found that half of the product sampled was contaminated. We believe that tackling *Campylobacter* effectively on broiler farms, and improving hygiene standards at slaughter, could enable 2002 contamination levels in retail product to be reduced by at least 50% over the next 1-2 years.

13. Whilst this Report largely reflects the position in the UK, we note that there is significant trade in poultry meat within the European Union (EU), including in product from Third Country origins. We therefore hope that the FSA will explore the implications of the Report in an EU, as well as a UK, context, consulting the European Food Safety Authority if and as the Agency deems appropriate. We note that, in 2002, the UK imported over 350,000 tonnes of poultry meat (around 45,000 tonnes from outside the EU),[4] and similar measures to those designed to reduce *Campylobacter* in UK broiler production also need to be introduced into supply chains where the source material is outside the UK.

Research

14. In order for this Report to be as useful as possible to the Food Standards Agency in developing its *Campylobacter* reduction strategy, we have focused on short to medium-term practical options for tackling *Campylobacter*. We have not addressed those research opportunities and gaps which fall into a longer time frame. However, the Working Group will be returning to the question of research needs, and we say something more about this in Chapter 1.

The structure of this Report

15. This Report represents the output of the ACMSF *Campylobacter* Working Group's deliberations and also reflects the conclusions of our *Campylobacter* workshop (see Chapter 1).

Chapter 1

16. Chapter 1 describes in greater detail the background to our second consideration of *Campylobacter*, our 2002 workshop, and the link between our work and the FSA's strategy for reducing foodborne disease. Chapter 1 also explains our approach to research in this Report.

Chapter 2

17. Chapter 2 highlights areas of interest and developments in relation to the capacity of *Campylobacter* to cause disease in humans. We look at genome sequencing, at how *Campylobacter* responds to stress, and at the infectious dose. We also touch on the debate about the role and nature of the Viable Non-Cultural (VNC) form of *Campylobacter*. Other aspects covered in Chapter 2 are the organism's pathogenicity, human immunity, acute illness, and the long-term *sequelae* of *Campylobacter* infection.

Chapter 3

18. In Chapter 3, we look at the epidemiology of *Campylobacter* and focus, amongst other things, on the disease burden, seasonality, general outbreaks, risk factors, and on various epidemiological conundrums.

Chapter 4

19. The focus of Chapter 4 is the means of preventing *Campylobacter* contamination of chicken meat. It is clear to us that control of *Campylobacter* on-farm is now a practical proposition, at least where birds are housed, and that this is an area for priority attention.

Chapter 5

20. Chapter 5 reflects the Scandinavian approach to preventing *Campylobacter* contamination of chicken meat.

Chapter 6

21. In Chapter 6, we look briefly at the question of *Campylobacter* contamination in poultry other than chicken.

Chapter 7

22. Notwithstanding the important association between poultry meat and human *Campylobacter* infection, we have not overlooked the fact that there are numerous other sources and vehicles of infection. Chapter 7 addresses the means by which contamination of meat other than poultry meat can be prevented.

Chapter 8

23. In Chapter 8, we consider the ways in which *Campylobacter* can be tackled in the domestic and catering environments. We give particular attention to excluding *Campylobacter* from the domestic and catering environments, temperature abuse, effective cooking, manufacturers' cooking instructions, cross-contamination, hygiene advice, companion animals, and food handlers.

Chapter 9

24. Chapter 9 focuses on *Campylobacter* detection and the most appropriate methods to be used. We also look at the difficult area of *Campylobacter* typing and suggest one approach which we believe could improve epidemiological understanding of the organism in the next few years. We also look at the potential future typing opportunities which DNA microarrays may provide.

Chapter 10

25. Finally, in Chapter 10, we summarise, for ease of reference, the conclusions we have drawn in this Report and the recommendations we have made.

Annexes, Glossary and References

26. The Report also contains a number of detailed annexes, a glossary of technical terms, and a comprehensive reference section.

Prioritisation of recommendations

27. The Committee has endeavoured to prioritise its recommendations along broad lines, indicating those areas where: (a) action is required in the short-term, to assist the FSA in developing and implementing its *Campylobacter* strategy; (b) work should be started in the next year or so; (c) work can be put in hand as and when possible, and in the light of competing priorities. The degree of prioritisation allocated to each recommendation is shown in Chapter 10.

Chapter 1

Background

Human *Campylobacter* infections in the UK

1.1 *Campylobacter* has, in recent years, become the single biggest identified cause of bacterial infectious intestinal disease (IID) in the UK. Latest published data for laboratory reports of *Campylobacter* spp. in England and Wales are shown in Figure 3.1. These show a sustained upward trend throughout the 1980s and 1990s, peaking in 1998 when there were in excess of 58,000 laboratory-confirmed *Campylobacter* cases. Since then, there has been a significant reduction in laboratory reports which, by 2002, had fallen to just over 46,600. The Report recognises the importance of *C. jejuni* and *C. coli*, but uses the term *Campylobacter* generally throughout the document.

ACMSF Interim Report on Campylobacter

1.2 The Advisory Committee on the Microbiological Safety of Food (ACMSF) has had a long-standing interest in *Campylobacter*.

1.3 In 1993, the Committee published an Interim Report on *Campylobacter*.[1] Whilst noting that the sources and routes of transmission of *Campylobacter* infection were not yet fully understood, the Interim Report pointed to the strong circumstantial evidence suggesting one major source was poultry, transmission being either directly through consumption of undercooked chicken or by cross-contamination of other foods in the kitchen.

1.4 The Interim Report noted that a major difficulty with human campylobacteriosis was that most cases were sporadic and unconnected, making it very difficult to establish the role of different foods. A key thrust of the Interim Report was therefore the need for further research and surveillance on the organism, specifically in relation to typing; isolation and identification; disease-causing potential; prevalence; and transmission and infection – to fill gaps in the understanding and knowledge of *Campylobacter*. The Interim Report also reminded industry and consumers of the crucial importance of effective temperature control, thorough cooking, and the avoidance of cross-contamination; and of education and training. The Interim Report also recommended that all sectors of the food industry should adopt a HACCP-based approach to the control of potential microbiological hazards.

1.5 Much of the Interim Report remains pertinent today. A significant amount of recommended research has been carried out, and there have been significant advances in what is known about *Campylobacter*. We point to some of these advances elsewhere in this Report.

FSA foodborne disease and chicken stategies

1.6 In 2000, the Food Standards Agency (FSA) set itself 2 targets in relation to foodborne disease, namely to reduce:

- the incidence of foodborne disease by 20% by April 2006,[2] and
- *Salmonella* contamination of UK-produced retail chicken by 50% by April 2005.[5]

1.7 The FSA noted, in its strategy for meeting its foodborne disease target,[6] that *Campylobacter* was the major cause of infectious intestinal disease in those consulting a doctor and the most common gastrointestinal pathogen reported by laboratories. The Agency concluded that a significant reduction in campylobacteriosis would therefore make a major contribution to achieving its foodborne disease target.

1.8 The FSA carried out a survey over the period April-June 2001 to establish baseline levels for the microbiological contamination of chicken (both UK and non-UK) on retail sale in the UK. Tables 1.1 and 1.2 show the levels of *Salmonella* and *Campylobacter* contamination respectively (reflecting the final survey results).[7]

Table 1.1: Percentage of *Salmonella*-contaminated raw fresh and frozen chicken purchased at retail in different parts of the United Kingdom

Category of chicken	England	Wales	Scotland	Northern Ireland	UK
Fresh	4.0	2.2	6.1	1.9	4.0
Frozen	9.8	6.6	16.7	16.2	10.4
All	5.5	3.4	8.8	5.5	5.7

Source: Food Standards Agency[7]

Table 1.2: Percentage of *Campylobacter*-contaminated raw fresh and frozen chicken purchased at retail in different parts of the United Kingdom

Category of chicken	England	Wales	Scotland	Northern Ireland	UK
Fresh	52	47	89	89	56
Frozen	30	29	35	40	31
All	46	42	75	77	50

Source: Food Standards Agency[7]

1.9 Having completed a preliminary analysis of the results from its survey, the FSA decided that, while the work on *Salmonella* reduction would continue, the reduction of *Campylobacter* in chickens should be afforded higher priority.[8]

1.10 As noted above, the long-standing view of the ACMSF has been that chickens are a major source of *Campylobacter*. The Committee believes that reducing the number of *Campylobacter*-positive chickens on retail sale and going into catering will make an important contribution to the reduction of human *Campylobacter* infections.

1.11 While the Food Standards Agency's foodborne disease strategy recognised poultry production as an obvious sector for attempting to control *Campylobacter*, it also drew attention to the possible contribution of raw meat as a source of organisms which cross-contaminate other foods. The foodborne disease strategy also highlighted the contribution which milk and dairy sector controls could make to the control of a range of pathogens including *Campylobacter*. Hygiene initiatives in the red meat and dairy sectors were seen as a useful means of reducing cases of campylobacteriosis, as were improving food hygiene in catering and in the home, focussing on thorough cooking and the prevention of cross-contamination.[9]

Campylobacter revisited by ACMSF

1.12 In 2000, the ACMSF decided to revisit *Campylobacter*, and to establish a formal Working Group for this purpose, with a view to identifying means of reducing the incidence of *Campylobacter* infections in humans. ***The Working Group's terms of reference were to identify any important gaps and omissions in action taken to reduce Campylobacter in food and food sources and in the knowledge base; and to develop advice which would assist the Food Standards Agency in evolving its strategy for reducing the incidence of foodborne Campylobacter infection in humans.*** Membership of the Working Group, and the full ACMSF, is shown in Annex A. Written and oral evidence was taken from a range of interested parties. Details are given at Annex B.

1.13 Given the aim of helping the FSA tackle the contribution made by *Campylobacter* to the burden of foodborne illness in the UK, and so that its advice would reach the FSA in timely fashion, the ACMSF decided to feed it in as and when it became available, rather than waiting until this final Report was ready. Advice was submitted to the FSA in September 2002 on control of *Campylobacter* in broiler flocks,[3] and a report was made to the Agency in January 2003 on the visit by members of the *Campylobacter* Working Group to Denmark and Norway (Annex C).

ACMSF *Campylobacter* workshop

1.14 As a precursor to the first meeting of the ACMSF's *Campylobacter* Working Group, the Committee held a *Campylobacter* workshop at the Britannia International Hotel in London Docklands in February 2002. This was attended by ACMSF Members and by invited experts from the UK and Europe. The workshop had 3 broad objectives, namely to:

- take stock of research findings both in the UK and elsewhere in the World;
- identify any major gaps in knowledge justifying on-going research; and
- decide, on the basis of current knowledge, whether there were food chain interventions which would reduce consumer exposure to *Campylobacter* and suggestions which would assist the FSA to achieve its foodborne disease and chicken meat microbiological contamination reduction targets.

1.15 The workshop proved very useful in helping identify a number of lines of thought and enquiry which participants felt would benefit the work of the *Campylobacter* Working Group. The following conclusions were drawn from the workshop discussion:

- **typing and detection:** there was a need for detection and typing methods to be adopted which were applicable across clinical, veterinary, environmental and food isolates. Progress was being made but differences remained which were hindering investigations;
- **the immune status of the population:** significant uncertainties remained on human vulnerability and immunity to *Campylobacter* infection;
- **pathogenesis:** it remained unclear whether all campylobacters were pathogenic to humans. There was a need for a robust animal model with surrogate markers for virulence. Investigation of pathogenesis was regarded as very important

and it was considered that, for this purpose, identification of non-pathogenic strains would be very useful;

- **origins and transmission:** there was a need to clarify origins of infection and routes of transmission. Targeted case-control studies would be essential in examining the importance of water and "travel" exposure pathways;

- **genomics:** the use of genomics was thought likely to yield general benefits in the longer-term. It was felt, however, that the exploitation of genome sequence data using microarrays might be useful for targeted research in the shorter-term;

- **seasonality:** the importance of seasonality needed to be assessed. The apparent seasonal increase in *Campylobacter* might reflect a combination of factors, eg. general environmental loading and the response to higher temperatures; animals being turned out to pasture; etc;

- **freezing:** it was noted that freezing of chicken carcasses served to reduce campylobacters on poultry meat by orders of magnitude. This contrasted with the situation in relation to *Salmonella* which was noted as being less sensitive to freezing;

- **irradiation:** irradiation of carcasses was an option available for reducing *Campylobacter* contamination of poultry meat but there was likely to be a high level of consumer resistance;

- **risk assessment:** the contribution of quantitative risk assessment needed to be borne in mind;

- **food and water exposure pathways:** there was strong evidence that poultry meat was an important source of human campylobacteriosis. However, the importance of foods other than poultry meat needed to be examined, as did the prevalence of the organism in finished foods and its response to processing. Given the organism's ubiquity in the environment, there was a need to focus on the contribution to human infection made by salads, sewage sludge and other organic wastes used in agriculture, and irrigation water. It would also be important to clarify the importance of human exposure through public water supplies;

- **food chain interventions:** it was considered essential to be able to identify intervention points to reduce the prevalence of *Campylobacter* at various stages of the food chain. This, it was felt, would help get the organism out of raw food

materials and thus ensure that measures taken to reduce the risk in the kitchen (eg. avoiding cross-contamination, appropriate storage and handling, thorough cooking) were incremental. By focusing on good and bad practices in poultry meat production, it should be possible to identify key, reproducible interventions. The role of general health in predisposing birds to colonisation needed to be investigated. It was noted that intensive and extensive production systems were likely to pose different problems in terms of control. However, a number of measures like pre-slaughter testing, and the scheduling of slaughter to reflect the disease status of birds, were likely to be helpful in both production settings.

1.16 Details of participants in the workshop, and of the presentations made, are also included in Annex A.

Scientific progress and research

1.17 More is known about *Campylobacter* now than at the time our Interim Report[1] was published in 1993. Much of this increased knowledge reflects the results of research undertaken in response to the recommendations in our Interim Report. We have not attempted, in our latest Report, to chronicle these advances in detail. Rather, we have endeavoured to signpost developments and where information about them can be found.

1.18 The purpose of this Report is to assist the FSA in its strategy for combating *Campylobacter* infections in humans and hence is focused on the short to medium-term. The importance of getting our Report to the FSA as early as possible has been a constant feature of our deliberations. We have thus not dealt with research opportunities and needs where there are significant gaps in knowledge, such as those discussed at our workshop.

1.19 Despite the scientific advances made, *Campylobacter* remains a poorly characterised microorganism and this impacts on its epidemiology and control. There is therefore a need for continued fundamental research, especially in the area of functional genomics. As noted earlier, the focus of this Report has been on the practical measures which will help the FSA develop its strategy for tackling *Campylobacter* in the shorter-term. However, the ACMSF *Campylobacter* Working Group will meet again with the aim of identifying where research outputs, had they been available, would have contributed to progressing the objectives identified as desirable in this Report more quickly. It should be recognised that any research requirements identified through this planned review can only yield results in the medium to longer-term, given the time lag involved between identifying research and being able to apply practical outputs.

Acknowledgements

1.20 The Committee wishes to express its appreciation for the views and assistance it has received from all of the organisations detailed in Annex B.

Chapter 2

The Organism, Human Immune Response and Pathogenesis

Introduction

2.1 There has been an immense amount of work on these aspects since the publication of our Interim Report on *Campylobacter*.[1] This has led to a much greater understanding of the organism, how it grows and survives, and how it causes illness. However, there are still issues to be resolved, and it is clear that further work may be required to fully understand the threat the organism poses to the foodchain. It is not intended that this Chapter should provide a definitive text on issues such as *Campylobacter* genetics, physiology and pathogenesis. Rather, it is intended to summarise the current state of knowledge and highlight areas of interest.

Genome sequencing

2.2 The publication of the genome sequence of *Campylobacter jejuni* NCTC11168 in 2000[10] was a major breakthrough in *Campylobacter* research. This strain of *C. jejuni* has a chromosome of 1,641,481 base pairs, which is relatively small compared to other bacteria, and is predicted to encode for 1,654 proteins and 54 stable RNA species. With 94.3% of the genome coding for proteins, this is one of the densest bacterial genomes (if not the densest) sequenced to date. It is unusual in that there are very few insertion or phage-associated sequences and an almost complete lack of repetitive DNA sequences (only 4 repeated sequences within the entire genome). The discovery of hypervariable sequences, commonly found in genes encoding the biosynthesis or modification of surface structures, is important. A surprising feature is that, with few exceptions, there seems to be minimal organisation of genes into operons or clusters.

2.3 The sequencing information has helped to identify the location and function of a variety of *C. jejuni* genes and has started to provide an insight into the metabolism of the organism, the virulence factors important in its pathogenesis, and its survival strategies. The size of the chromosome means that the number of genes is limited (around 1,600 compared to over 5,000 in *Salmonella)* and this might explain why *Campylobacter* has fastidious growth requirements. Further, the high rate of variation in the hypervariable sequences could explain how genetic traits are altered in *C. jejuni* populations and also how the organism is able to survive in changing environmental conditions.

2.4 The genome sequence of *C. jejuni* NCTC11168 may lack important genes. Whilst it may thus be considered as a valuable reference point for post-genomic analysis, there is a need to examine the DNA sequences of additional strains in order to approach a definitive view of the *Campylobacter* genome. Already work on other strains of *C. jejuni* has shown striking differences in the content, position and arrangement of mapped genes.[11]

Responses to stress

2.5 There is debate as to the extent to which *Campylobacter* is sensitive to environmental stresses. A long-held view is that the organism is unusually sensitive, and there is no doubt that *Campylobacter* appears to lack many adaptive responses which can be correlated with resistance in other bacteria. For example, analysis of the genome indicates that the organism does not possess the RpoS global regulator[10] which, in a number of Gram-negative organisms, is the basis for the survival of the bacterial cell in stationary phase and during exposure to many types of environmental stress. Recent work has shown, however, that *C. jejuni* can mount adaptive responses to both acidic and aerobic conditions,[12,13] and there is increasing recognition that *Campylobacter* is more resistant to stress than had initially been thought. One theory is that the methods used to assess survival have caused confusion.[14] Because the methods have not been optimised for the recovery of damaged cells, they have provided the opportunity for an overestimation of sensitivity.

Response to high temperature

2.6 Although thermophilic in nature, campylobacters are readily inactivated by heat and do not survive pasteurisation treatments or typical cooking procedures. The organism does exhibit a heat shock response, with at least 24 proteins being preferentially synthesised by *C. jejuni* immediately after exposure to temperatures above that which is optimal for growth.[15] The response mechanism is still being investigated, but it appears to be different to those seen in other bacteria. It has been suggested that the heat shock response may play a role in both thermotolerance and colonisation, as mutants deficient in one of the heat shock proteins have severely reduced growth at 46°C and are unable to colonise chickens.[15]

Response to low temperature

2.7 *Campylobacter jejuni* does not appear to produce cold shock proteins,[10] which may explain why it cannot grow below 30ºC. Unlike other bacteria, which show a gradual reduction in growth rate near their minimal growth temperature, *C. jejuni* shows a rapid decline.[16] However, at temperatures as low as 4ºC, the organism can respire, generate ATP, and move towards favourable environments, although it is unable to replicate.[16] At lower temperatures, viability is lost rapidly but the organism can still be isolated from frozen meats and poultry products.[17]

Response to oxidative stress

2.8 Most *Campylobacter* spp. are microaerophilic, although a few species have now been found to grow under strictly aerobic or anaerobic conditions.[18] Those that are microaerophilic, which includes the main species linked to foodborne disease, have an inherent sensitivity towards oxygen and its reduction products, and require cellular defences for survival during exposure to air. Oxidative stress has been shown to contribute to the freeze-thaw induced killing of *Campylobacter*.[19]

Response to osmotic stress

2.9 Many bacteria have regulatory mechanisms which enable them to resist osmotic stress, for example through the synthesis or transport of compatible solutes. Such systems appear to be absent in *Campylobacter,*[10] and this may explain why the organism is relatively sensitive to osmotic stress. That said, there is evidence that the organism can be recovered from dry surfaces 24 hours after contamination, albeit in low numbers.[14]

Response to stationary phase and starvation

2.10 For many bacteria, entry into the stationary phase, eg. following starvation, is accompanied by profound structural changes which result in increased resistance to heat shock, oxidative, osmotic and acid stresses.[20] This change is commonly mediated by RpoS. However, as already noted, this system does not appear to be present in *Campylobacter*. In fact, it has been reported that the organism exhibits the opposite response to many other bacteria. Indeed, it has been observed that stationary phase cultures of *C. jejuni* are more sensitive to mild heat stress and oxidative stress than those containing exponential phase cells.[21]

The debate on coccoid cells and Viable Non-Culturable (VNC) *Campylobacter*

2.11 There is still debate about the role and nature of the so-called Viable Non-Culturable (VNC) form of *Campylobacter*. This concept was introduced in the mid 1980s, with reports that *C. jejuni* changed form from a culturable spiral to a non-culturable coccoidal structure. It was suggested that this was a dormant state that helps *Campylobacter* survive in hostile environments.[22] Since then, questions have been raised about whether non-culturability equates to non-viability,[23] whether it is possible to revert the VNC form to a culturable form,[24-27] and, indeed, whether a VNC form of *Campylobacter* actually exists. As it has been demonstrated that only a limited number of isolates form the VNC stage,[28] the view that the existence of a VNC form may simply be due to strain differences has also been advanced.[29]

2.12 Another view is that the coccoid form is merely a degenerative form of the spiral,[30] especially as its formation is not prevented by the inhibition of

protein synthesis or DNA replication.[31] However, the observation that different types of cocci form in response to different temperatures suggests that the situation may be more complicated.[31] It has also been suggested that, under stress conditions, young spiral cells form coccoids, whereas the older spiral cells show degenerative changes.[26]

Infectious dose

2.13 The *Campylobacter* infectious dose is thought to be very low (<500 bacterial cells). Very few data are available from outbreaks, and studies to determine the exact number of cells that will cause human infection have proved inconclusive, although examination of a bottle of bird-pecked milk, which was part of a batch implicated in an outbreak at a nursery, revealed contamination levels of less than 10 cells of *C. jejuni* per 100 ml.[32]

Pathogenicity

2.14 Whilst the mechanism of *Campylobacter* infection in humans is still not fully understood, some of the factors essential for pathogenesis have now been identified. Infection appears to include at least two stages namely, the organism adhering to the intestine of the animal and producing toxin, then invading and proliferating within the intestinal mucosa.[29] Motility is thought to contribute significantly to both colonisation and the development of disease,[33] and understanding of how this works is increasing, genes having been identified for regulatory components of a chemotaxis system and candidate receptors for signal detection.[10] It has also been shown that one of the flagellin subunit proteins is important for adhesion to host cells.[34]

2.15 The function and role of toxins in pathogenesis is yet to be fully elucidated and remains a topic of debate. A variety of toxins has been reported,[35-37] many of which are similar to those found in some other bacteria, including one apparently related to the cholera toxin[35] (the existence of which is controversial, not least because it does not appear to be encoded on the genome[10]). A cytotoxin thought to be involved in pathogenesis, cytolethal distending toxin (CDT), has been identified in *C. jejuni*,[38] although its precise function has yet to be determined.

2.16 Invasion mechanisms also remain poorly understood, although there is recent evidence to suggest that they are strain-specific.[39] Flagella-mediated motility is thought to be a major contributing factor,[34] with invasion likely to involve changes to the host cell membrane and/or the internal cytoskeletal structure.[40] *Campylobacter jejuni* secretes proteins which are felt to be essential for internalisation of the organism into mammalian cells, these being synthesised during interaction with epithelial cells.[41] Following invasion, *C. jejuni* appears to be largely confined to membrane-bound vacuoles, although some organisms have been detected free in the cytoplasm.[40]

2.17 The mechanism by which *Campylobacter* causes diarrhoea is becoming understood, although it is apparent that various hosts react differently to invasion, with fluid secretion being dependent on the extent of the host response and degree of epithelial damage.[18] The process of cell adhesion, internalisation and movement of the organism across host cells, attracts white blood cells to the site of infection, and granules released by these cells during *Campylobacter* phagocytosis cause tissue damage, inflammation and, ultimately, diarrhoea.[18] (See paragraph 3.3)

Immunity

2.18 There is a view that vaccination may be a possible control option, as infected people mount a strong immune response. In addition, immunity against *Campylobacter* is possible, in the absence of acute infection, with many abattoir workers apparently being immune to infection after initial exposure.[42] There have also been attempts to develop *Campylobacter* vaccines, whole-cell oral vaccine formulations having been tested with good results in primates,[43] and a vaccine based on whole-cell formulations and a purified flagellin giving some protective immunity in the mouse model.[44]

The acute illness

2.19 Clinical features of acute *Campylobacter* infection vary from mild diarrhoea lasting 24 hours to severe illness lasting more than a week. The incubation period is typically 2 to 5 days, although can be up to 11 days, with the onset of diarrhoea (which is often blood-stained) being preceded by malaise and, possibly, fever. Characteristic of campylobacteriosis is a persistent colicky abdominal pain which may mimic acute appendicitis. Other symptoms which may be present are headache, backache, aching of the limbs and nausea. However, vomiting is uncommon.

Long-term *sequelae* of *Campylobacter* infection

2.20 Long-term *sequelae* of *Campylobacter* infection include neurological, rheumatological and renal problems.

Guillain-Barré syndrome

2.21 The association between *Campylobacter* infection and subsequent Guillain-Barré syndrome (GBS), as well as the related Miller Fisher syndrome (MFS), is well documented. Guillain-Barré syndrome is an acute, bilateral, ascending paralysis occurring typically 1-3 weeks following onset of diarrhoea.[45] The association appears to be restricted to infection with *C. jejuni* species, which is the most commonly reported infectious trigger for GBS.

2.22 The prevalence of *C. jejuni* infection among GBS cases, based mostly on serology, ranges from 15% to 66%, compared with between 0% and 17% among controls in various settings.[46-56]

2.23 Preceding *Campylobacter* infection appears to be associated with more severe neurological symptoms, slower recovery, and poorer outcome from GBS after one year.[46] Particular *C. jejuni* serotypes, namely O:19[55] and O:41,[57] have been implicated in the development of GBS in certain settings.

2.24 The most reliable estimate of GBS incidence is from Sweden, where laboratory reports of *C. jejuni* infection were linked with cases of GBS identified through the Swedish Inpatient Register.[58] This yielded an estimate of 30.1 cases of GBS per 100,000 cases of *C. jejuni* IID for the two-month period following infection.

2.25 By applying this estimate to England figures, it has been estimated that 15 hospital admissions due to GBS occur among laboratory-confirmed cases of *C. jejuni* IID in a 12-month period.[59] However, this estimate takes into account solely those cases of *C. jejuni* IID that are reported to national surveillance (only a tenth of all cases in the community).[60] Assuming the risk of GBS is the same among both reported and unreported cases, the expected number of *C. jejuni*-associated GBS hospitalisations over a 12-month period rises to 157, representing nearly 15% of all GBS admissions in England.[59]

Post infection arthropathies

2.26 *Campylobacter* has been associated with a range of rheumatological conditions, most commonly reported as reactive arthritis (ReA), defined here as an aseptic arthritis following an enteric infection.

2.27 *Campylobacter*-induced ReA occurs an average of two weeks following onset of diarrhoea.[61,62] The condition typically affects more than one joint, most commonly the knees, ankles, wrists and lower back. The average duration of arthritic symptoms is approximately two months.[62] A predominance of the HLA-B27 genotype has been described among case reports of *Campylobacter*-associated ReA,[61,62] although this may not be the case in the population setting.[63]

2.28 Follow-up of cases from large outbreaks of *Campylobacter* IID have yielded estimates of subsequent ReA of between 0.6% and 1.1%.[64,65] However, these estimates are likely to be biased by losses to follow-up, the lack of an appropriate control group, and the fact that outbreak cases might be atypical in terms of their epidemiology and/or microbiology.

2.29 Retrospective follow-up of 210 *Campylobacter* patients presenting to general practice in Denmark yielded a probable ReA frequency of 15%.[66]

2.30 A prospective study of 870 laboratory-confirmed *Campylobacter* IID cases and 1,440 population controls in Finland yielded a ReA frequency of 7%, diagnosed by clinical examination (0% among population controls).[63]

2.31 Despite using slightly different definitions of ReA, in both studies the median interval between onset of diarrhoea and occurrence of rheumatological symptoms was two weeks. Females predominated among ReA cases, and young adults and the middle-aged were most commonly affected. ReA occurred in both *C. jejuni* and *Campylobacter coli* patients and was associated with longer duration of diarrhoeal symptoms.

2.32 Applying the Finnish estimate to England and Wales surveillance figures, an estimated 3,961 episodes of ReA would be expected among the 56,592 laboratory-confirmed cases of *Campylobacter* IID reported to the Public Health Laboratory Service (now the Health Protection Agency) Communicable Disease Surveillance Centre (CDSC) in 2001. Assuming that an incidence of 7% also applies to non-reported cases of *Campylobacter* IID, and accounting for under-reporting to national surveillance,[60] up to 40,802 episodes of ReA might have resulted from symptomatic *Campylobacter* infection in England and Wales in 2001.

Haemolytic uraemic syndrome

2.33 Evidence for a link between haemolytic uraemic syndrome (HUS) and preceding *Campylobacter* infection comes from individual case reports. Preceding infection with *C. jejuni*[67-70] and, to a lesser extent, *Campylobacter upsaliensis*[71] in the absence of verocytotoxin-producing *Escherichia coli* (VTEC) has been implicated. To date, no systematic follow-up study of *Campylobacter* IID patients for occurrence of HUS has been carried out, and no data on the frequency of this complication are available.

Conclusions

2.34 The debate about the role, indeed the very existence, of the VNC form of *Campylobacter* seems unlikely to be resolved in the short-term. It is a complex area and not one where we have been able to draw any firm conclusions. However, we are not aware of any evidence to suggest that current uncertainties give cause for concern in relation to food safety. We are not, therefore, recommending that the FSA should commit funds to further research on the VNC issue. We note that the research community continues to carry out work in this area. This should be monitored and we hope that a consensus view will eventually emerge.

2.35 *Campylobacter* isolation methods have been improved since much of the work on VNC was performed, and it is now possible to recover cells previously thought to be non-culturable. What is not yet clear is whether very highly damaged cells of *Campylobacter* now recoverable from a variety of environments, and after a variety of treatments, pose an infection threat.

2.36 It is clear that infectious intestinal disease causes a considerable burden of ill health over and above the initial event. However, little information is available on the incidence and economic cost of long term *sequelae* and it would be useful to have a more reliable measure.

Recommendations

2.37 We recommend that the Government should instigate a primary care-based sentinel surveillance system, aimed at measuring directly the incidence and economic cost of long-term *sequelae* among cases of *Campylobacter* infectious intestinal disease. (Priority B)

2.38 We recommend that serological markers for recent infection and prior immunity be developed and tested through structured, epidemiologically robust, population-based studies. This should assist with estimating the prevalence of asymptomatic infection in the population (and hence estimating more accurately the magnitude of *Campylobacter*-associated *sequelae*). (Priority C)

Chapter 3

Campylobacter Epidemiology

Introduction

3.1 Although campylobacters emerged as important pathogens more than 20 years ago, their epidemiology is still poorly understood. One of the perceived difficulties was the lack of routine microbiological characterisation of clinical strains.[72] This has militated against a systematic study of the epidemiology of the different species and sub-types of *Campylobacter*. Developing and targeting control and prevention strategies is impossible without a proper understanding of the epidemiology of *Campylobacter* infection.

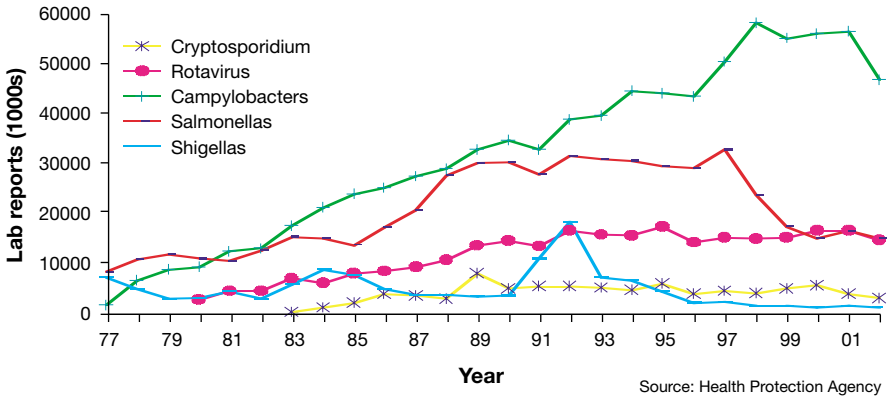
3.2 On 1 May 2000, the Public Health Laboratory Service (PHLS) – now the Health Protection Agency – launched an active, sentinel, population-based surveillance scheme for *Campylobacter* infections in England and Wales – the *Campylobacter* Sentinel Surveillance Scheme (CSSS).[73,74] The aim was to generate hypotheses for human *Campylobacter* infection using a systematic, integrated epidemiological and microbiological approach. Twenty-two District Health Authorities (DHAs), chosen for their geographical representativeness, collaborated in the three-year scheme, working with their hospital microbiology and local authority environmental health departments. The sentinel system covered a population of approximately 12.5 million people. Campylobacters isolated by National Health Service and PHLS laboratories within the DHA catchment referred all their isolates to the *Campylobacter* Reference Unit (CRU) of the PHLS Laboratory of Enteric Pathogens (LEP) for detailed strain characterisation. A standard, structured clinical and risk factor questionnaire was administered to the patient by the relevant Health or Local Authority as part of their routine investigation of foodborne infection. The Gastrointestinal Diseases Division of the PHLS Communicable Disease Surveillance Centre (CDSC) then collated epidemiological exposure data and microbiological typing information centrally. The scheme captured standardised information on approximately 15% of all laboratory confirmed *Campylobacter* infections in England and Wales between 1 May 2000 and 30 April 2003.

Disease burden

3.3 *Campylobacter* is the most commonly reported bacterial cause of gastroenteritis in the developed world. Figure 3.1 shows the trend in laboratory reporting in England and Wales since 1977. In 1998, the peak year, 58,059 laboratory confirmed cases in England and Wales were reported to the CDSC.[75] Data from the CSSS show that approximately 20% of

Campylobacter cases have travelled abroad in the fortnight before illness onset.[76]

Figure 3.1: Laboratory reporting of selected gastrointestinal pathogens in England & Wales, 1997-2002.



3.4 Under-ascertainment of infectious intestinal disease (IID) is well-recognised, and the true population burden is greater than that given by national surveillance. The ratio of infection in the community to reports to national surveillance for *Campylobacter* spp. is approximately 8 to 1.[77] This means that, in 2000, there were approaching half a million *Campylobacter* cases in the community.

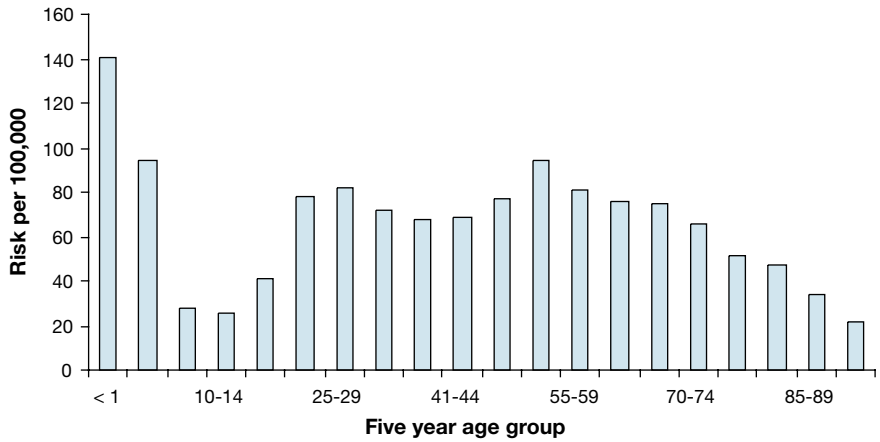
3.5 In 2000, it has been estimated that there were 1,388,772 cases of foodborne infection acquired in England and Wales (so called indigenous foodborne disease (IFD)). *Campylobacter* accounted for 359,466 of these IFD cases.[78] It is estimated that there were 171,174 presentations to general practice due to *Campylobacter* infection, 16,946 hospital admissions (accounting for 62,701 hospital bed days) and 86 deaths.[78]

3.6 The economic burden due to *Campylobacter* infection is large. In the United States, the annual estimated cost is around US$4.3 billion.[79] The average cost of a case of acute *Campylobacter* infection (excluding long-term *sequelae*) in England in 1995 was estimated to be £315.[80] Conservatively, therefore, foodborne *Campylobacter* infection cost the nation over £113 million in 2000.

Age-specific incidence estimates

3.7 Using population denominators, the highest age-specific incidence estimates are in children under the age of five years, with a secondary peak in young adults.[75,81-83] Figure 3.2 shows the age and gender distribution of *Campylobacter* cases reported through the CSSS. This reveals a third peak in the 50-54 year old age group[83] and has been a consistent finding throughout the project.

Figure 3.2: Age distribution of *Campylobacter* cases reported to the *Campylobacter* Sentinel Surveillance Scheme



Source: Health Protection Agency

3.8 Age-specific incidence estimates based on population denominators hide the fact that there is a much greater tendency to obtain samples from young children. Thus, when faecal samples are used as the denominator, the highest isolation rates are in young adults and the lowest in young children.[84,85]

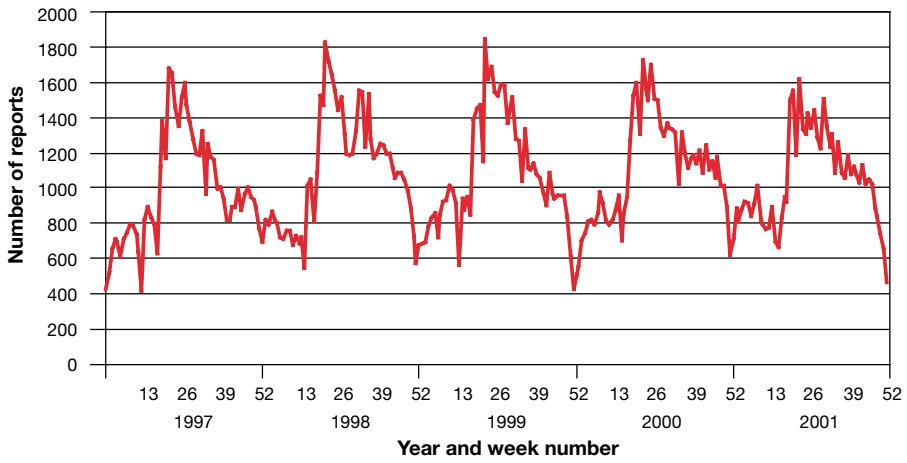
Ethnicity

3.9 Analysis of the CSSS dataset has shown that people who described their ethnic origin as Pakistani were at a higher risk of infection, experienced longer periods of illness and higher rates of hospital admission. There was a marked skewing of the age distribution towards infants and a higher proportion of males was affected. The Pakistani community reported lower levels of chicken and red meat/meat product consumption, lower levels of water consumption and lower levels of contact with animals. These findings are important because they suggest community-specific differences in routes of transmission for *Campylobacter* infection. Thus failure to take ethnicity into consideration might mask important risk factors for infection and limit understanding of disease transmission processes.[86]

Seasonality

3.10 A striking feature of the epidemiology of human *Campylobacter* infection is its remarkably pronounced and consistent seasonal pattern. Figure 3.3 shows weekly laboratory reports to the CDSC in England and Wales. There is a sharp rise in cases in the late spring and early summer, which levels off in June and July.[87,88] There is a suggestion that the precise timing of the seasonal peak varies with longitude. In a recent European study, weekly numbers peaked earliest in the western-most countries studied, peaking later further east.[88] Although well characterised, the epidemiology of the seasonal peak in humans is not well understood. Various hypotheses have been suggested, including buying puppies in the summer months,[89] consumption of bird-pecked milk,[90-93] or exposure to environmental risks.[93]

Figure 3.3: Weekly laboratory reports of *Campylobacter* spp. in England & Wales 1997-2001



3.11 In seeking to explain seasonality in human carriage rates, a variety of animal reservoirs have been examined. *Campylobacter* carriage rates in broiler chicken flocks[94-96] and dairy cattle[97] peak in the spring and late summer, in contrast to lamb and beef cattle where such marked seasonal variation in carriage rates have not been observed.[97,98]

General outbreaks

3.12 A notable characteristic of *Campylobacter* epidemiology is that general outbreaks (ie. those affecting members of more than one household) are rarely recognised.[99,100] Of the 2,374 general outbreaks of infectious intestinal disease reported to CDSC between 1995 and 1999 where an aetiological agent was identified, *Campylobacter* accounted for only 50 (2%).[100] The proportion of *Campylobacter* cases recognised as being part of outbreaks during this period was only 0.4% compared with 8% for

Salmonella and 15.5% for *E. coli* O157.[100] However, when general outbreaks of *Campylobacter* infection are recognised, they are more likely to be investigated using analytical epidemiology (as opposed to descriptive techniques) than either *Salmonella* or *E. coli* O157 outbreaks, presumably because the rarity of the event stimulates such an investigation.[100,101]

3.13 Thirty-five of the 50 outbreaks reported to CDSC between 1995 and 1999 were foodborne. Where a food vehicle was identified (24/35 outbreaks), the vehicle most frequently identified was poultry (13 chicken; one duck). Cross-contamination was the most commonly reported food-handling fault (18 outbreaks).[100]

3.14 Analysis of the CCCS dataset suggests that point source general outbreaks might be more common than is currently recognised. Of the 3,489 cases of *Campylobacter jejuni* infection reported in the first year of the study, 333 (10%) reported knowledge of an individual outside the household with a similar illness.[102] Cases who reported other illness in the community were more likely to have reported having eaten in restaurants or consumed unpasteurised milk.[102] There is a well known association between consumption of unpasteurised milk and outbreaks of *Campylobacter* infection in England and Wales,[103] so this finding, though unsurprising, adds weight to previous findings. Nevertheless raw milk for drinking remains on sale despite overwhelming scientific evidence[104-107] about the risks associated with its consumption, and despite the ACMSF's recommendation that its sale in England, Wales and Northern Ireland should be banned (it is already banned in Scotland). The impact of the restaurant setting in *Campylobacter* outbreaks is also well recognised[99,100] and accords with the findings of epidemiological studies of sporadic disease linking chicken prepared by, or eaten in, a commercial food establishment with infection.[108-110]

Risk factors for sporadic disease

Poultry

3.15 To explain the risk factors for sporadic disease (the majority of *Campylobacter* infection), case-control studies have been conducted in various settings. These have all demonstrated the complexity of the epidemiology of *Campylobacter* infection and, each time, a range of exposures has been identified.

3.16 Poultry consumption has been demonstrated to be a risk factor in several studies. Various types of poultry have been implicated as summarised below:

- any type of chicken;[111-115]

- poultry and poultry liver;[116]
- raw or rare chicken;[117-119]
- cooked chicken;[118]
- processed chicken;[120]
- chicken prepared by or eaten in a commercial food establishment.[108-110,119,120]

3.17 In a case-control study of primary, indigenous, sporadic campylobacteriosis in England and Wales, however, consumption or handling of chicken cooked and eaten in the home was found to be protective.[121] Similarly, in a study in New Zealand, recent consumption of baked or roast chicken seemed to be protective, although consumption of raw or undercooked chicken, or chicken from restaurants, was associated with illness.[108] An earlier study in New Zealand also showed that eating at home was protective.[122]

3.18 The role of poultry consumption as a risk factor for *Campylobacter* infection in epidemiological studies can be confusing since, in certain case-control studies of sporadic disease, consumption of chicken is a risk factor, whilst in others it appears to protect against developing infection. In trying to disentangle these contradictions, there is a need to distinguish between chicken as a potential source of *Campylobacter* infection and chicken as a food vehicle. There is no doubt that poultry is a major source of *Campylobacter* spp.[123] and there is scope for cross-contamination of other foods if infected poultry is introduced into the kitchen. Yet if cooked properly the contaminated chicken itself no longer poses a risk.

Other foods

3.19 Other foods implicated as risk factors for sporadic *Campylobacter* infection include:

- barbecued meat;[112,124]
- raw milk;[108,115,116,125]
- bird pecked milk;[92,114]
- bottled mineral water[126]

Water

3.20 Consumption of untreated water,[116,120] or rainwater[108] have been implicated as risk factors for *Campylobacter* infection. In an ecological study in Sweden, positive associations were found between the incidence of *Campylobacter* and average water-pipe length per person. There were similar associations with ruminant density. These observations suggest that drinking water and contamination from livestock might also be important factors in explaining at least a proportion of human sporadic Campylobacteriosis.[127]

Other risk factors

3.21 In addition to risks from food (especially poultry) and water consumption, contact with animals (either domestic pets or farm animals),[115,116,120,124,125] or reported problems with the home sewerage system, have also been implicated in infection.[108] Underlying medical conditions such as diabetes[114] or reducing gastric acidity through the use of proton pump inhibitors[128] also increase the risk of acquiring *Campylobacter* infection.

Epidemiological conundrums

3.22 Despite the multiplicity of risk factors identified for *Campylobacter* infection, in most case-control studies of *Campylobacter* infection the majority of cases remain unexplained.[108-110,114,121,124] That *Campylobacter* infection, like other foodborne zoonoses, is transmitted through more than one route is not in doubt. What is not known for certain is the relative importance of these transmission routes in the aetiology of infection.

3.23 It has been suggested that between 20% and 40% of sporadic disease might be due to the consumption of chicken.[130,131] If this is so, controlling *Campylobacter* carriage in the poultry reservoir might have a measurably beneficial effect on human disease incidence. Nevertheless, the reasons behind the majority of human disease would still not have been tackled.

3.24 The paucity of recognised outbreaks has undoubtedly hampered scientific understanding of the epidemiology of *Campylobacter* infection. By contrast, the epidemiology of verocytotoxin-producing *Escherichia coli* O157 is much better defined, and the diligent investigation of recognised outbreaks has made a major contribution to understanding the aetiology of sporadic disease.[132]

3.25 Many of the case-control studies have used a case definition incorporating *Campylobacter* spp., although recent evidence generated through the CSSS suggests that case-control studies should be conducted at least at the species level since inter-species differences in risk factors might occur.[74]

3.26 The large proportion of unexplained cases might prove to be due to as yet unidentified risk factors or exposures that are very rare among unaffected individuals in the population. If this is the case, then very high powered studies will be needed to detect their effects.[129]

Conclusions

3.27 *Campylobacter* infection is a major public health problem. The epidemiology is complex. There are extensive animal and environmental reservoirs and multiple risk factors for infection. Although epidemiological patterns, such as marked seasonality, are well described, their underlying explanations are still elusive despite much study.

3.28 Poultry appears to be an important source of infection. It is noteworthy that eating food, including poultry, on commercial catering premises has been identified as a risk in several case-control studies.

3.29 In the case of poultry, some progress has been made in reducing the role of the food chain as a vehicle for *Campylobacter* infection. However, in addition to the contribution of poultry to human *Campylobacter* infection, many studies also point to numerous other sources and vehicles of *Campylobacter* infection. It is important that these are not overlooked.

Recommendations

3.30 The contribution of foodborne transmission (as opposed to other transmission modes) to the human toll of *Campylobacter* needs to be better defined and we note that the Food Standards Agency has already funded a research project designed so to do. **We support this course of action. (Priority A)**

3.31 We recommend that population studies to investigate the seasonality of *Campylobacter* infection be undertaken. An approach combining epidemiological, microbiological, environmental and veterinary expertise is likely to be needed. (Priority A)

3.32 We recommend that population studies to investigate cultural/behavioural risk factors for *Campylobacter* be undertaken. (Priority B)

3.33 We recommend that more extensive data are gathered on the levels of *Campylobacter* spp. in specific foods (eg. water, dairy products, vegetables, poultry and red meat) as well as in food-producing animals and companion animals. These are all potential sources of exposure for humans. We recommend that consideration be given to on-going surveillance as well as to "snap-shots" which tend to be the norm. It is very important that the microbiological methods employed allow meaningful comparisons to be made across the food chain (see Chapter 9). (Priority A)

Chapter 4

Measures to prevent *campylobacter* contamination of chicken meat

Introduction

4.1 Although various food vehicles are discussed in this Report as possible sources of human *Campylobacter* infection, we judge that particular attention needs to be given to chicken meat although the Working Group recognises the potential importance of other sources.

Chicken meat as a source of human *Campylobacter* infection

4.2 As noted in Chapter 1, the Food Standards Agency carried out a survey in 2001 of the microbiological status of UK and non-UK chicken meat on retail sale in the UK.[7] Fifty-six percent of fresh and 31% of frozen chicken sampled were contaminated with *Campylobacter* spp. A more detailed breakdown of these results is provided in Table 1.2.

Control of *Campylobacter* in chickens

4.3 *Campylobacter* spp, principally *Campylobacter jejuni* and, to a lesser extent, *Campylobacter coli,* are common in commercial poultry flocks. Data from current FSA-, and past MAFF-funded, research and from the scientific literature[133-138] indicate that approximately 60% of housed (broiler) poultry flocks, both in the UK and elsewhere, are *Campylobacter*-positive at slaughter age. This will vary from company to company, from farmer to farmer, and between flocks. Where numbers of colonised birds are lower than the average for housed poultry, it is likely that *Campylobacter* will only have become established towards the end of the commercial life of the flock. It is also possible that some strains of *Campylobacter* may spread more slowly in broiler flocks. There appears to be a general trend towards lower colonisation rates in the UK, reflecting the fact that farmers are becoming more successful in preventing the entry of this bacterium.

4.4 *Campylobacter* control is possible for housed birds, as interventions in Scandinavia, particularly Norway and Sweden, have illustrated (see Chapter 5) and it is this type of production that we focus on in this Report. The prevalence of *Campylobacter*-positive flocks in Denmark is currently similar to that in the UK,[139,140] although some Danish farmers routinely produce negative flocks. The interventions identified in this Chapter are primarily applicable to housed production. There seems likely to be a much more difficult problem, however, with extensive production systems. The main

reason for this is that *Campylobacter* is ubiquitous in the natural environment and thus, if chickens are outdoors, they are more likely to be exposed to these bacteria. Further research into the control of *Campylobacter* in extensively-reared (including free range and organic) chickens is necessary.

Potential sources of *Campylobacter* spp. in chickens

4.5 Over the last 25 years, since the identification of poultry meat as an important source of human infection with *Campylobacter* spp., there have been many studies in many countries into the epidemiology of this zoonotic pathogen in poultry production. As with many areas of science, there is a degree of dispute over the importance of the various routes of infection, which are identified as:

- contaminated water;
- vertical transmission from parent flocks;
- contaminated feed;
- carry-over from a previous flock;
- domestic and/or wild animals and birds;
- contaminated transport crates, vehicles and personnel at flock thinning and when birds are weighed or maintenance is carried out;
- equipment at times other than thinning;
- feed withdrawal; and
- the external environment around the broiler house;
- contaminated footwear and clothing of farm personnel and visitors;
- transfer of contaminated equipment between houses.

The potential sources shown above are not presented in rank order and it is recognised that contaminated feed, for example, is likely to be only a very rare infection route (see below).

4.6 Although the epidemiology of *Campylobacter* infection in chickens has some similarities to that of *Salmonella* spp., there is one important difference. *Salmonella* primarily enters poultry flocks when the chicks are very young. *Campylobacter* is rarely found in broiler flocks until the birds are in the third

week of life. There is currently no agreement on the reason(s) for this delay but the following have been suggested as having a role:

- maternal antibodies in young chicks, as most broiler-breeder flocks are *Campylobacter*-positive and anti-*Campylobacter* antibodies may be present in egg yolks;[141]

- the presence in birds of bacterial floras antagonistic to *Campylobacter* spp.[142]

It has not yet been possible to determine why birds apparently become susceptible to *Campylobacter* at 3-4 weeks of age. It could clearly be due to one or both of the above factors. It is also important to recognise that the birds' metabolism and gut flora will change with age, diet and environment, which may also affect susceptibility. Similarly, vaccines given in the third week of life may also play a role.

4.7 The control of *Campylobacter* spp. in broiler production is principally one of identifying ways by which the three-week *Campylobacter*-free period can be extended until slaughter age. Essentially, this means either preventing the entry of the bacteria into the flock and/or improving the resistance of the birds to colonisation. For exclusion to be achieved, the most important sources of flock infection need to be identified. A number have been implicated and are discussed below.

Contaminated water

4.8 One study, on a farm in the UK,[143,144] and a number of investigations in Scandinavia,[94,135] have demonstrated that contaminated water, particularly when untreated ground water is used, can be responsible for the introduction of *Campylobacter* spp. into poultry flocks. This was also identified as an important source in Norway when *Campylobacter* Working Group members visited that country in November 2002. Studies by Pearson[143,144] raised the intriguing prospect that viable non-culturable (VNC) *Campylobacter* was responsible for the initial colonisation event. There is much dispute about the importance of this physiological state (see Chapter 2) but improved isolation methods are now able to recover cells of *Campylobacter* thought to be VNC. Of all the potential routes, waterborne infection should be the easiest for farmers to control. It is very important that all poultry flocks receive only water of potable quality. Additional treatment, in the form of chlorine or ozone, is also likely to prove beneficial. Caution may need to be exercised with regard to the use of ozone as this compound can be toxic. The use of chlorine was part of a package of measures shown to markedly reduce flock colonisation in an on-farm trial in the East of England.[145,146] Organic acids such as peracetic acid, sometimes used in combination with hydrogen peroxide or stabilised with silver to enhance stability and antibacterial activity and reduce corrosion, may also be useful.

Work is needed to determine whether the use of such treatments prior to slaughter can help to reduce *Campylobacter* levels in the chicken gut.

4.9 Drinking water provided by bell and cup drinkers may also act as a vehicle for horizontal transmission within the broiler house once *Campylobacter* has become established, as these can allow young chickens in particular to contaminate the water. It is noted that drinkers may act as a vehicle for transmission within the broiler house once *Campylobacter* has become established and that *Campylobacter* can become part of biofilms established in broiler house water systems, a phenomenon that we believe requires further investigation [147,148].

Vertical transmission from parent flocks

4.10 There is continuing debate about the relative contribution of vertical transmission of *Campylobacter* spp. from breeding flocks. Kazwala *et al*[149] and van de Giessen *et al*[150] suggest that, because it is possible in a minority of flocks to isolate *Campylobacter* from broilers within 1-2 days after hatching, the bacteria could be acquired vertically. Recent work [151,152] showed that *C. jejuni* could colonise the oviduct, probably as a result of an ascending infection from the cloaca. Investigations in the USA also provide some evidence to support the view that certain strains of *C. jejuni* may be transmitted vertically from colonised breeder flocks.[151] However, this is a highly contentious area, and it has yet to be demonstrated beyond reasonable doubt that *Campylobacte*r spp. can be isolated from newly hatched chicks. It may be that the bacteria track up from the cloaca and become transient colonisers of reproductive tissues.[151] Published studies have largely used as the basis for their conclusion that vertical transmission has occurred, that the same *Campylobacter* strain has been isolated from parent flocks and their progeny.[153] In work of this kind, it is important to rule out infection from common sources, particularly with more common strains. *Campylobacter* has, however, been recovered from semen samples from breeder cockerels.[154] This does provide some support for vertical transmission, at least in a US setting, and more work may be needed in this area in Europe. The fact that it is very difficult to isolate *Campylobacter* from birds less than 2-4 weeks of age is an argument against vertical transmission, although it cannot yet be ruled out as an occasional route. In addition, the fact that some farms frequently produce *Campylobacter*-free flocks, often one after another, also makes vertical transmission less probable.

4.11 A body of work has also been undertaken on the survival of *Campylobacter* spp. in the contents of artificially-contaminated eggs, and on the ability of these bacteria to penetrate egg shells. Data on this are equivocal. Shanker *et al*[155] examined 187 eggs from *Campylobacter*-positive breeder flocks. Two showed evidence of penetration of the egg shell. The progeny of positive breeder flocks were also examined and it was found that

all 14 flocks examined remained *Campylobacter*-negative until slaughter. Egg contamination studies were also performed. 257 eggs were contaminated with *C. jejuni* on the surface. 162 hatched; all were *Campylobacter*-negative. *Campylobacter* was injected into the contents of 167 eggs. 12 hatched and 2 of the chicks were infected. The authors conclude that their data do not support vertical transmission of *Campylobacter* spp. When fertile eggs were infected with *Campylobacter* and incubated immediately, up to 100% of the chicks had the bacterium in their intestines. When eggs were stored for 5 days before incubation, the incidence of *Campylobacter*-positive eggs fell to 20% or less, and no chicks contained *Campylobacter* on hatching.[156] These data indicate that *Campylobacter* may not survive well in egg contents, and this has been re-examined in a recent study.[157] A temperature differential method, where cooling eggs are immersed in a culture broth, was used to contaminate eggs. It was found that *C. jejuni* had only limited ability to penetrate the egg shell. In other components of this work, *C. jejuni* was found to survive well in egg yolk, but poorly in either albumen or air sacs. *Campylobacter jejuni* was detected in 3 of 65 egg pools (5-10 eggs per pool, laid by infected breeders) when the eggs were tested soon after lay. When eggs were stored for 7 days at 18°C before testing, all 500 examined were *Campylobacter*-negative. This study also examined 500 fresh eggs from breeders shown to be *Campylobacter*-positive, and 1,000 eggs from a commercial hatchery. All were *Campylobacter*-negative. The authors conclude that this suggests that vertical transmission is a rare event. There is clearly some need to examine this aspect in European production systems. There is also a possibility that a small number of chicks are *Campylobacter*-positive at hatching, and that the bacteria take time to spread through the flock to a sufficient level to allow detection.

Contaminated feed

4.12 It is well established that contaminated feed is a potentially important route of flock infection with *Salmonella* spp.[158] This does not seem to be the case with *Campylobacter*. The ubiquity of *Campylobacter* in food animals and the environment means that raw feed ingredients will often be contaminated with these bacteria by wild bird droppings, for example. However, *Campylobacter* spp. are very sensitive to dry conditions and have been shown to die quickly when present in poultry feed.[159] We judge that the *Salmonella* control measures in place in the UK to improve feed hygiene will be adequate to control *Campylobacter* spp. However, it is important to remember that, as with water, feed can act as a vehicle for horizontal transmission in a broiler house once *Campylobacter* has become established. It is also possible that feed may be saved at de-population and used with subsequent flocks. This feed may represent a higher infection risk. Many of the studies undertaken on the survival of *Campylobacter* in feed used techniques subsequently shown to lack sensitivity. It would be useful to repeat such work using appropriate, sensitive methodologies.

Carry-over from a previous flock

4.13 Some studies have demonstrated that the same type of *Campylobacter* can be isolated from successive flocks.[160,161] One possible explanation is therefore that the bacteria were carried over from one flock to the next. It is also possible that both flocks were colonised from the same source. However, laboratory-derived data indicate that *Campylobacter* spp. are significantly more sensitive to damaging conditions than *Salmonella*.[162,163] Buildings should be of sound construction and well-maintained to prevent access by wild birds and to deter rodents. If house cleaning and disinfection are undertaken properly, then *Campylobacter* will be absent from cleaned houses, and any regime which removes *Salmonella* spp. will eliminate *Campylobacter.* It is thus unlikely that this potential source is important, although one study in Denmark found that the majority of broiler flocks (11/12) carried identical *Campylobacter* isolates in two or more flocks.[160] As discussed above, it was not possible in this study to differentiate between carry-over and a common source. Whatever the importance of carry-over, given the ability of *Campylobacter* spp. to colonise, it is essential that house cleaning and disinfection are rigorously carried out.

Domestic and/or wild animals and birds

4.14 Most warm-blooded animals carry *Campylobacter* spp. Wild animals and birds act largely as an indirect source of flock infection, as a consequence of environmental contamination. Similarly, farms with mixed animal species also run the risk of increased flock infection because farm staff may transmit the bacteria from cattle, sheep or pigs to chickens. The increased risk that this poses may seriously undermine biosecurity, and a potentially important control measure is to rear chickens on species mono-specific farms. Given that cats and dogs are also frequently *Campylobacter*-positive, it is also important that these animals are not allowed access to poultry flocks. Anti-*Salmonella* control measures which prevent the access of wild birds and rodents will contribute to protecting flocks from *Campylobacter* colonisation too.

4.15 Houseflies have also been shown to act as a source of *C. jejuni* for specific pathogen-free chicks.[164] This is likely to be the result of surface contamination, rather than faecal excretion, as Jones *et al*[165] could not isolate *Campylobacter* from flies after surface disinfection. Rosef and Kapperud[166] isolated *Campylobacter* from 43-50% of flies sampled around poultry houses. Our *Campylobacter* Working Group was able to find no published information on possible roles for flies and other insects as transmission vehicles in commercial settings. Flies and other arthropods are likely to enter the broiler house in larger numbers in the summer and may thus be involved in the 'summerpeak' of infection. This might be particularly relevant if sources of *Campylobacter* are close to broiler houses.

Contaminated transport crates, vehicles and personnel at flock thinning

4.16 Many poultry companies in the UK carry out the practice of "thinning", for welfare reasons. Broiler houses are stocked with numbers of birds which would be above the recommendation for stocking density if all the birds remained until slaughter weight. To overcome this, at approximately 5 weeks of age a cohort of birds is removed for slaughter, with the remainder being kept for 1-2 weeks further. Thinning also provides producers with the necessary flexibility to react quickly to the demands of the fresh retail market. It is not unusual for ordering by customers to exceed projected demand ten-fold from day to day. These fluctuations in demand could not be accommodated in the absence of the flexibility which thinning provides. However, thinning has a number of important public health implications, in relation to contamination introduced on-farm by staff and visitors and on crates, as well as the deleterious effects of stress. Studies in Denmark have found that this process is a significant risk factor for flock colonisation with *Campylobacter* spp.[167]

4.17 During the thinning process, crates and modules that may be contaminated can introduce *Campylobacter* into a previously negative flock.[168,169] The gloves and clothing of the catchers have also been shown to be *Campylobacter*-positive.[169] The potential ingress of *Campylobacter* is compounded by the fact that the birds often become stressed as a result of the catching process. This may render those remaining in the house more susceptible to colonisation with *Campylobacter* spp.

4.18 Birds are transported to slaughter in crates by lorry. During catching, loading and transportation to the processing plant, the crate surfaces and the lorry decks become contaminated with faeces from the birds in the crates. The cost of poultry transport crates means that they are used repeatedly. Given the high incidence of *Campylobacter* in broiler chickens, crates are frequently contaminated with these bacteria. Crates must be cleaned and disinfected after use. They are washed at the processing plant, but this process has been shown to be far from ideal.[167,169] The water is often re-cycled from the processing plant, is often used at ambient temperature, and the levels of detergents and/or disinfectants are often sub-optimal and may also be quickly neutralised by the high levels of organic matter present in the crate wash water, which will be re-cycled within the crate washer. Crates therefore often leave the washer contaminated with *Campylobacter* spp.

4.19 Schedule 1 of The Welfare of Animals (Transport) Order 1997, provides that means of transport and receptacles shall be constructed, maintained and operated so as to allow appropriate cleaning and disinfection. The Transport of Animals (Cleansing and Disinfection) (England) (No. 2) Order 2000 requires all animal transport vehicles and containers to be cleansed

and disinfected after each use and within 24 hours of the journey being completed. Assured Chicken Production (ACP)[a] has produced a leaflet entitled "Poultry standards: catching, transport and slaughter". Rule 3.7 states that "*Processing plants must provide cleaning and sanitation provisions for crates and transporters. All transporters and crates must be washed after unloading*". No information is given about perceived best practice.

4.20 The decks of vehicles used to carry the crates also become contaminated and will spread contamination if they are not adequately cleansed and disinfected between journeys. In addition, as lorry tyres are potential vectors of *Campylobacter*, there should be a disinfectant wheel bath, or each wheel should be sprayed before entry to, and exit from, a poultry unit.

4.21 Thinning has a number of important public health implications. It is therefore essential in terms of microbiological safety that, where it is practised, crate, module and lorry washing are properly carried out and that crates are not contaminated with *Campylobacter* (or, indeed, with other pathogenic microorganisms). Other biosecurity measures, in relation to clothing, footwear, etc are also essential. We believe that improved hygiene standards will yield improved benefits in flock health and may help offset the increased costs involved. Unless the poultry industry is prepared to take these necessary steps to improve the microbiological acceptability of thinning, then we strongly believe that the practice should be discontinued (the industry adjusting stocking densities as necessary to achieve required standards of welfare), thus reducing the risks of transmitting *Campylobacter* infection. In adopting this stance, we do not overlook the very strong submissions we have received from informed industry sources underlining the difficulties the industry is facing in what is a highly competitive and price sensitive sector where import penetration is a continuing threat. We nevertheless believe that the public health implications of thinning, as well as the deleterious effects of stress on stock, are too important to be ignored.

The effects of feed withdrawal

4.22 An important hygiene problem in broiler processing is the accidental contamination of the carcass at slaughter by gut contents, particularly faecal material, and, as a consequence, the spread of pathogens such as *Campylobacter*. To reduce the danger, feed is withdrawn some time before birds are loaded into their transport crates, whether at thinning or at final depopulation. Fasting periods of 4-10 hours have been recommended[170] (indeed, in our Report on Poultry Meat,[171] we concluded that, on balance, a period of between 6 and 10 hours should be allowed between feeding and kill). However, the overall period without feed will be longer than this because

[a] Assured Chicken Production (ACP), which presented evidence to the ACMSF *Campylobacter* Working Group, operates detailed poultry standards. There is more information about these in Annex D.

of the time taken to load and transport the birds to the processing plant, and any time spent waiting in lairage before slaughter. These factors must be taken into account by the farmer when deciding when to withdraw feed. The average transport time for broilers in the UK is 3.6 hours, although some birds can spend over 12 hours in crates before slaughter.[172] It is possible that broilers could spend between 7-20 hours without feed before slaughter.

4.23 There is continuing debate about whether these fasting times are, in fact, beneficial. Reducing the gut contents will reduce the pressure on the intestines and any leakage of contents on to the carcass if the gut is accidentally broken during evisceration. However, even prolonged feed withdrawal will not completely prevent defaecation occurring during *ante mortem* handling. Removing feed, or both feed and water, have similar effects on gut contents. Most reduction in weight occurs in the crop, and least in the caeca and cloaca. An important finding is that the contents of most parts of the gut, but particularly those of the crop and cloaca, get wetter with longer deprivation. In contrast, caecal contents become slightly drier. Fasting tends to progressively increase the number of Enterobacteriaceae and *Campylobacter* in all parts of the gut but especially in the caeca and cloaca.

4.24 Feed withdrawal will not eliminate cross-contamination of the plumage of live birds with faecal matter during transport. Moreover, it may also have unforeseen adverse effects by inducing stress, which may pre-dispose birds to *Campylobacter* infection. Work with *Salmonella* spp. has shown that birds may become systemically infected very rapidly (within 2 hours) after exposure to sources of infection.[173] It is likely, given the commonness of *Campylobacter* in poultry, that infection with this bacterium will be equally rapid. Feed withdrawal may also affect the microbiological flora of the gut by modifying the growth of bacteria normally present, such as lactobacilli, with subsequent changes in the pH of the gut contents. Lactobacilli are also known to have the ability to prevent/reduce intestinal colonisation with zoonotic pathogens. For example, a study, which examined the effects of stress in young monkeys[174] found that this was associated with a reduction in levels of lactic acid bacteria in the gut. Many of the stressed animals became infected with *C. jejuni*, which was endemic in the colony. It is also of interest that longer feed withdrawal times (up to 24 hours) are associated with a higher prevalence of chickens testing positive for *C. jejuni* in crop samples before slaughter.[175] Thus, do the possible increased risks of gut breakage, and greater susceptibility to infection, outweigh perceived benefits on lower carcass contamination levels with zoonotic pathogens like *Campylobacter* spp?

4.25 Whatever the pros and cons of the above, it would not be unreasonable to postulate that birds remaining after thinning might be more susceptible to infection as a result of a combination of disturbance and feed

withdrawal. This practice is only likely to have a marginal effect on the *Campylobacter* status of birds removed for slaughter.

The environment as a source of flock colonisation

4.26 Although flock colonisation is possible by any of the routes identified above, there is a general agreement in the international scientific community that the environment around the broiler house is the most important source of flock colonisation.[94,133,135,176-178] *Campylobacter* spp. can be isolated with regularity from the farm and the natural environment. It has been shown that *Campylobacter* spp. from the external environment can match those in broiler chickens.[176] The bacteria are present in the environment as a consequence of faecal contamination from wild and domestic animals and birds. A recent study in Denmark has cast some doubt, however, on the role of wild animals and birds as sources of *Campylobacter* spp. for broiler chickens, but did confirm the importance of the contaminated environment.[179] The use of manures as fertilisers also constitutes an infection risk. Investigations with one UK poultry producer, whose system is typical of UK production, demonstrated that farmers with poor farm hygiene practices were more likely to produce *Campylobacter*-positive flocks than those whose hygiene was good.[180] The inference to be drawn from this work is that "dirty" farms are likely to have a higher loading of *Campylobacter* in the environment, and that "dirty" farmers may be more likely to carry the bacteria into the broiler house. Although *Campylobacter* are generally regarded as being sensitive to the extra-intestinal environment they may be able to survive for extended periods in areas with high water levels such as puddles, drainage channels etc.

4.27 A number of different *Campylobacter* sub-types can be isolated from a broiler flock, and even from the same bird. In general, however, one or two subtypes will dominate the bacterial population. There is some dispute over whether the different subtypes indicate the entry of two different bacteria, or whether the genomic instability of *Campylobacter* leads to changes in the original strain, which produce an identifiably different bacterium.[181] The principal event in the colonisation of a broiler flock is the establishment of the bacterium in the first bird(s). Passage through a chicken has been shown to greatly increase the ability of *Campylobacter* to colonise subsequent birds.[182,183] Spread can be very rapid in a newly colonised flock, and almost all birds will be *Campylobacter*-positive within a few days of the initial colonisation event.[142] A major component of any control strategy must therefore be to prevent *Campylobacter* from the environment entering the broiler house. It would also be valuable to determine why, in some flocks, not all birds are infected and whether this represents differences in host susceptibility or bacterial pathogenicity.

4.28 The most important anti-*Campylobacter* control measures, falling within the term "biosecurity", help ensure that the bacterium is kept out of

the broiler house. It is important to note that *Campylobacter* is more difficult to exclude from chickens than *Salmonella* spp. Thus, measures which exclude *Salmonella* may not be successful with *Campylobacter*. With this bacterium, the margins for error are much smaller, and much more attention to detail may be required in order to achieve robust security. Good farming practice and high levels of stockmanship are seen as an essential basis for the successful and continuing avoidance of *Campylobacter* entry and spread.

4.29 The average broiler flock experiences many visits by different people during the growing cycle. Each one carries with it the risk of allowing *Campylobacter* into the flock. Visits should be limited to essential personnel, with each visit fully justified and recorded. There will still be at least daily visits to the flock by farm staff, and it is vital that these are undertaken as hygienically as possible. One study in SW England found that, when farm staff dipped their footwear in strong phenolic disinfectant, it was possible to either prevent or delay flock colonisation in three flocks.[180] This method may be difficult to sustain for long periods, as the disinfectant baths may not be changed with sufficient regularity and can become contaminated with soil and other organic matter. A much better approach is to supplement the foot dips by constructing a hygiene barrier at the entrance to the anteroom which adjoins the area housing the birds. Sets of dedicated outer clothing and footwear should be held on the inside of the hygiene barrier. All people who enter the broiler house should remove their own footwear and put on the protective clothing and shoes/boots. Where dedicated footwear is not in use, shoes/boots must be dipped in disinfectant baths before entry into the flock. The disinfectant should be changed frequently to ensure continued efficacy. Wider, more easily cleaned, concreted areas separating the entrance to the houses from the farm environment (as seen in Denmark during the Working Group's visit – see Chapter 5) would also increase the buffer zone, and there would also be benefit in coating the sites in coarse gravel to enhance the effectiveness of routine spraying for weeds.

4.30 The above approach has been shown to be effective in trials in the UK[145,146] and over a sustained period in the Netherlands and Scandinavia,[94,138,177,178,184] and we see no reason why this type of *Campylobacter* control requirement cannot be incorporated into farm assurance schemes in the UK. A study undertaken by the Veterinary Laboratories Agency[185] investigated Scandinavian-type intervention in the UK. Measures tested included boot dipping, changing boots and outer clothing, and hand washing. Data from this study show that, where personnel strictly followed the biosecurity programme, flocks were 3-times less likely to be *Campylobacter*-positive. Flock colonisation rates were also halved if boot dips were changed more than once per week. Such measures have the advantage of being relatively inexpensive, although we do recognise that such systems can be difficult to sustain in the long-term.[186,187]

All companies should have standard operating procedures for biosecurity and related matters. There should be a forward looking veterinary health plan which includes appropriate training of all farm staff on how to prevent the introduction of infection into flocks. Farmers also need to be convinced that no emergency, flood and fire apart, is so urgent that the broiler flocks can be entered without outer clothing but, particularly, footwear being changed. Precautions must encompass all visits to the site, both human and vehicular. A single visit can result in flock colonisation by *Campylobacter* spp.

4.31 We are confident that properly applied biosecurity will significantly reduce the incidence of *Campylobacter* colonisation in housed chickens. We recognise that this may be more difficult to apply in the UK than in Scandinavia where the winters are much harsher and the number of houses per farm may be lower. No information is currently available on the loading of *Campylobacter* in the farm environment but it is possible that the harsh winters in Scandinavia markedly reduce pathogen numbers. Work is needed to examine this. Indeed, an examination of data from Scandinavian countries illustrates that flock infection rates in summer can approach those of the UK. The ACMSF was presented with evidence which convinced us that, on many farms, effective biosecurity is an achievable objective which should be explored with some urgency by the UK industry. Some farmers are already quite successful in excluding *Campylobacter*. There are no viable alternatives at present to proper and sustained biosecurity. It may be, however, that this approach could be supported in the future by other measures such as phage treatment, pre- or probiotics.

4.32 The Working Group was presented with a wide range of opinion on the major factors for broiler flock colonisation with *Campylobacter* spp. The following summarises the key risks and potential control measures:

- Re-stocking. Measures applied between one flock and the next could be important in control and proper cleaning and disinfecting is vital.

- People entering the flock pose the greatest risk and their numbers and activities, particularly with regard to maintenance of biosecurity, should be strictly controlled.

- Water can act as a primary and secondary source and water supply hygiene is important.

- Thinning also represents a substantial risk and hygiene improvements are urgently required.

Broiler flock management and *Campylobacter* colonisation

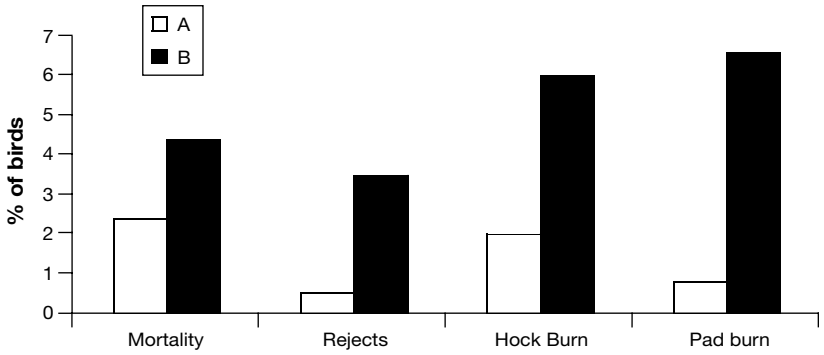
4.33 It is perhaps natural, given its commonality in poultry, to regard

Campylobacter as normal gut flora in chickens, although infection has been shown to be transient in wild birds. In poultry flocks, the high stocking densities may allow a maintenance of infection by re-cycling between birds. Given that it is now possible for many producers in the UK and elsewhere in Europe to regularly produce *Campylobacter*-negative chickens, this definition may need to be reviewed. *Campylobacter* in housed chickens does not seem to behave like either, eg., *Escherichia coli* or faecal streptococci which will be found in all chickens, irrespective of their *Campylobacter* status. A more accurate description for *Campylobacter* in housed flocks would perhaps be "frequent coloniser". There have been many studies on risk factors for broiler flock colonisation with *Campylobacter.*[94,135-138,178,179,188-192] One currently in progress in the UK includes an examination of the differences between farmers who produce broiler flocks with different *Campylobacter* infection rates. This arose from an examination of data from poultry companies which showed that farmers differ markedly in their ability to produce chickens which at slaughter age are *Campylobacter*-free. Some farmers can rear negative flocks with high frequency, whilst with others, almost all birds will be *Campylobacter*-positive. These observations give reasons for confidence that practical measures are available for reducing *Campylobacter* on a commercial basis. Work is needed to examine the behaviour of *Campylobacter* in individual birds in flocks to inform this debate.

4.34 Our *Campylobacter* Working Group was presented with preliminary data from research carried out in cooperation with one company in the South West of England. Some of the data from this work are presented in Figure 4.1 and compare two farms in the company which represent the ends of the industry spectrum of broiler flock positivity. One farm (A) had only 1.4% *Campylobacter*-positive chickens over six flock cycles. In contrast, 97% of birds from the other farm (B) were colonised over the same period. The feed was identical and both farms received the same type of birds, albeit possibly from different broiler-breeder flocks and from breeders of different ages. This latter point may be of importance, as industry data suggest that chicks from breeder flocks that are either entering or leaving the period of lay will be of potentially poorer quality than when breeders are in the peak period of productivity. There are a multitude of reasons why the two farms differ in performance with regard to the exclusion of *Campylobacter* spp. It is likely that, in general terms, the infection of broiler chickens with *Campylobacter* relies on chance and requires that a chicken in the flock is presented with sufficient cells to constitute an infective dose. In this respect, host susceptibility will be important. It would not be unreasonable to assume that chickens in poor health or kept in poor conditions may be more susceptible. With this in mind, the comparison in Figure 4.1 shows that there are marked differences between the two flocks in terms of flock mortality, the level of rejects at slaughter, and in two measures of the nature of the material upon which the birds sit, namely hock and pad burn. In each case, the birds from the farm which almost always produces *Campylobacter*-negative birds had

better production scores. One interpretation of these data is that birds in which health, performance or welfare are poor are compromised in their ability to withstand challenge from *Campylobacter.*

Figure 4.1: A comparison of low and high *Campylobacter* farms in relation to certain health/quality indicators



Farm A has consistently fewer *Campylobacter*-positive chickens.
Farm B has consistently greater numbers of *Campylobacter*-positive chickens.

NB: The vertical axis uses an arbitrary scale to compare birds from the two farms. In essence, the data on mortality and rejects at the processing plant are recorded figures whereas those for hock and pad burn are the recorded figures divided by 10. This was done to allow an easier comparison between farms.
Source: Unpublished data from the University of Bristol

4.35 One factor that might differ between the two farms is dryness of the litter, and this is known to be an important factor in the epidemiology of *Salmonella* infection. Data suggest that *Campylobacter* spp. in dry litter may be less infectious than bacteria in wet litter,[190,193] probably because the bacteria survive less well in dry litter.[194] Treatment of litter with either aluminium sulphate or sodium bisulphate also significantly reduced the incidence of *Campylobacter* colonisation.[195]

4.36 The aetiology of hock and pad burn is not yet fully understood but, essentially, they are manifestations of physical damage to the birds' feet and legs as a consequence of contact with litter of poor quality. The cause of these lesions is multi-factorial. Evidence currently available indicates that there is little relationship between the incidence of the two lesions.[196] Moreover, these problems are not confined to housed birds, and are seen with free-range birds too. Industry's view is that hock and pad burn come about from a combination of poor diet, poor ventilation, and over-supply of drinking water, leading to wet litter, but that there is no direct relationship between these conditions and the *Campylobacter* status of the birds.

4.37 There are welfare and public health needs to identify the key differences between farms which differ with respect to *Campylobacter* status. If it is true that healthier chickens are able to resist *Campylobacter*, then there are two potential benefits for the poultry industry. Productivity and profitability will be improved, and contamination levels with *Campylobacter* will be reduced. The ACMSF recognises the potential dangers of trying to over-simplify the control of *Campylobacter* in chickens, but the evidence we have seen clearly shows that some farmers are more successful than others in controlling this important zoonotic pathogen. It is vital that, if there are lessons to be learned from the more successful farmers, they are used to inform others in the industry.

Vaccination and other treatments as anti-*Campylobacter* measures

4.38 Surveillance of *Campylobacter* isolates from human cases and chickens has shown that strains present in the latter are not always found in the former. This raises the intriguing prospect that some chicken-associated *Campylobacter* strains are non-pathogenic for humans. Given that the poultry gut flora usually contain a dominant *Campylobacter* type, the non-pathogenic strains may have a role as agents to exclude potential human pathogens. Recently published work has shown that, under laboratory conditions, birds colonised with one *Campylobacter* isolate were able to resist challenge with another.[197,198] Caution may be needed with this approach. The genome of *C. jejuni* contains many hypervariable sequences[10] and these allow a high degree of genetic adaptability. Given that passage through the chicken gut increases the ability of *C. jejuni* to colonise other chickens,[182,183] it must be established beyond all reasonable doubt that the strains used as exclusion agents do not change to become human pathogens. It should also be borne in mind that human and poultry populations are surveyed in very different ways, often using different techniques. Thus, the absence of a strain in one population at a particular time cannot necessarily be taken to mean that it is always absent. Similarly, a strain may not be detected in chickens or on carcasses because it is only present in low numbers. This strain may, however, be able to infect humans, even when present at low levels.

4.39 The use of mixed bacterial cultures as an anti-*Salmonella* measure in broiler production is well established in the international poultry industry, and this approach is usually referred to as 'competitive exclusion'.[199] Some work has been undertaken to try to develop preparations with efficacy against *Campylobacter* spp. Results have been mixed so far.[200-205] Another approach may be possible. Young, *Campylobacter*-negative, broiler chickens have been shown to have a gut flora which is naturally antagonistic to *C. jejuni*.[142,202,204] Experimental data indicate that these gut bacteria, under

laboratory conditions, are able to protect against challenge with broth cultures of *C. jejuni*.[202,204] This may provide an explanation for why chickens do not usually become *Campylobacter*-positive until the third week of life. More work is needed on this approach, but it has the advantage of being a 'natural' phenomenon.

4.40 In common with all other bacteria, *Campylobacter* spp. can be attacked by viruses known as bacteriophages (or phages). These viruses generally have a limited host range, a fact which allows them to be used as typing agents for both *Campylobacter* and *Salmonella* spp. Phages are found naturally in the chicken gut and offer another potential control measure. Research on this approach continues, but it may one day be possible to treat a *Campylobacter*-positive flock a few days before slaughter to either reduce or eliminate carriage of the bacteria. A possible limitation with this approach is that it might lead to an increase in the prevalence of phage-resistant *Campylobacter* strains.

4.41 The genome of a strain of *C. jejuni* has been sequenced, which has made it possible to better understand the behaviour of this bacterium. Work is in progress to establish a library of *Campylobacter* strains with mutations in different single genes. By using these bacteria in chicken colonisation studies, it should be possible to identify the genes which enable *Campylobacter* to establish in the chicken intestine. A medium to long term aim of this work is that, by better understanding the genetics of gut colonisation, it may be possible to produce component vaccines against particular cell targets.

4.42 One reason suggested for the delay in the colonisation of broiler chickens with *Campylobacter* is the presence of maternal antibodies which protect the chicks during the first few weeks of life. There is an increasing body of evidence which suggests that chickens can mount an antibody response to *Campylobacter* spp. One study[141] determined the prevalence of anti-*C. jejuni* antibodies in breeders, the yolks of their eggs, and in broilers. High antibody levels were found in breeders and egg yolks. When broilers were examined, sera from 1 and 7 day old chicks also contained high antibody levels which then declined and became undetectable by 3-4 weeks. A recent study[206] has also provided valuable information about possible roles of maternal antibodies. Laboratory challenges were used to determine whether *Campylobacter*-specific maternal antibody (MAB) plays a protective role in young chickens. Colonisation with *C. jejuni* was compared in 3 day old broiler chicks which were MAB-positive, and 21 day old birds which were MAB-negative. Colonisation occurred much sooner in the older birds than it did in the younger ones, indicating a possible involvement of specific MAB in the delay of colonisation seen naturally. To examine this further,

Campylobacter-positive and negative specific pathogen-free chickens were raised under laboratory conditions, and their progenies with or without *Campylobacter*-specific MAB were challenged orally with *C. jejuni*. Significantly fewer colonised chickens were observed in the MAB + group during the first week post-infection. The authors state that MAB did not seem to affect the development of systemic immune response following infection with *C. jejuni*, although such responses occurred earlier and more strongly in birds infected at 21 days of age than in those infected at 3 days. Clearance of *Campylobacter* infection was also observed in chickens infected at 21 days of age.

4.43 There have been a number of other studies which have examined antibody production in response to artificial infection. 1 day old chicks challenged with a strain of *C. jejuni* showed significant increases in IgG, IgA and IgM circulating antibodies following oral challenge, with levels peaking 9, 5 and 7 weeks post-infection respectively. Specific mucosal IgG and IgA antibodies were also seen, and maternal IgG antibodies were also detected over the first 2 weeks. The major antibody response was to flagellin proteins.[207] Two other studies using artificially-infected chicks[208,209] found similar results. One[209] also demonstrated the presence of antibody in the sera of 11 of 12 naturally-colonised broiler flocks. In contrast, one study,[210] which used 11 *C. jejuni* strains, found that there was a poor antibody response to oral challenge with the bacterium. The authors found high levels of maternal antibodies and concluded that these could be responsible for delays in colonisation seen in broiler flocks. In a novel study, Noor *et al*[211] found that the vaccination of the forming embryo *in ovo* also stimulated the precocious development of immunity in chicks.

4.44 The above indicate that current data on immune responses by chickens to *Campylobacter* remain equivocal and may require further investigation. In addition to the studies mentioned earlier,[141,206] other work has been done to examine whether the administration of antibodies can protect chickens against challenge with *Campylobacter*. In one investigation,[212] cells of a *C. jejuni* strain, treated with various chicken anti-*Campylobacter* antibody preparations, were used to infect chickens. The authors concluded that pre-exposure to antibodies inhibited subsequent colonisation of chicken caeca. Other work[213] found that chickens immunised intraperitoneally with killed whole cells of *C. jejuni*, and subsequently challenged with live cells, had only 2% of the levels in caeca found in non-immunised control birds. Later work by this group[214] found that intraperitoneal vaccination with heat-killed cells reduced numbers of *C. jejuni* in the caeca of artificially-infected birds by 2-logs. The major antigen against which antibody activity was directed was flagellin protein. Another study,[215] which used oral vaccination with formalin-killed cells of *C. jejuni*, found that

reductions in colonisation in vaccinated birds ranged from 16-93% of caecal levels compared to controls. The administration of anti-*Campylobacter* antibodies prior to infection resulted in a marked reduction (>99%) in caecal *Campylobacter* levels in artificially-infected broilers. Administration of antibodies after infection had been established also reduced levels in the caecum, although effects were smaller (80-95% reduction).[216]

4.45 Evidence to date suggests that chickens can mount an antibody response to both natural and artificial challenge with *C. jejuni*. Vaccination with killed cells of *Campylobacter*, or treatment with antibodies, provide some protection. There is thus a possibility that such treatments could have commercial application. Given the economic constraints under which the poultry industry operates, the protective preparations must be able to be delivered on a mass scale and in a cost-effective manner. They should also afford protection against a broad range of *Campylobacter* strains.

4.46 Another long-term anti-*Campylobacter* measure is to develop breeds of chickens which cannot be colonised with these bacteria. It has already been established that genetic lines of chickens differ in susceptibility to *Salmonella* spp., and work is in progress to examine whether similar differences will be seen with *Campylobacter* spp.

Carcass treatments

4.47 It is our strongly held view that the main focus for the control of *Campylobacter* in chickens should be the farm and that particular attention should be given to improving biosecurity. We do not rule out the possibility that processing aids will be developed, the use of which may supplement on-farm biosecurity measures. We discuss some options below. However, we wish to stress that none of the treatments discussed below should be regarded as a substitute for good hygiene practice.

4.48 In Denmark, risk modelling has suggested that a 2-$\log_{10}$ reduction in carcass contamination levels could lead to substantial reductions in human infection rates. Research in many countries has shown that a number of approaches are possible to reduce *Campylobacter* contamination levels on chicken carcasses and most can achieve the 2-$\log_{10}$ reduction, believed to be significant in Danish calculations. It should be borne in mind that there can be marked variations in the levels of contamination from $<10^3$ to $> 10^9$ per carcass, as assessed by the enumeration of cells in a single carcass rinse.[123]

4.49 Gamma irradiation has been shown to be effective against *Campylobacter* spp. in raw ground beef.[217] Given the success of this approach with *Salmonella* spp. on chicken carcasses,[218] it is likely that it would be effective with *Campylobacter* also. Studies with artificially

contaminated chicken drumsticks demonstrated that the use of cobalt-60 at 0.5 KGy effected a 99% reduction of *C. jejuni.*[219] However, there is doubt that irradiation would be acceptable to consumers in the UK at the present time.

4.50 Chemical treatments have also been examined, although under EU legislation, only potable water can be used in poultry processing plants. Data on the effectiveness of chlorine are equivocal. One study with artificially contaminated chicken drumsticks demonstrated that the use of chlorine had only a negligible effect on *C. jejuni*.[219] In contrast, the addition of 25 ppm to wash water significantly reduced levels of naturally occurring cells of *C. jejuni* on whole chicken carcasses.[220] Improvements in poultry process hygiene, which included the use of chlorinated water sprays to limit microbial contamination on equipment and working surfaces, and an increase in the chlorine concentrations in process water, significantly reduced *Campylobacter* levels on carcasses.[221] Immersion of carcasses in water containing 10% tri-sodium phosphate solution also brought about a 1.71 $\log_{10}$/gram reduction in *Campylobacter* levels.[220] The use of 1% lactic acid as a spray significantly reduced the levels of artificially inoculated cells of *C. jejuni* on chicken carcasses.[222] Work is required to properly assess the efficacy of different treatments under commercial conditions.

4.51 One study has shown, perhaps not surprisingly, that the removal of the skin caused a significant reduction in *Campylobacter* levels on broiler carcasses.[223]

4.52 There has been quite a large body of work on the effects of either high or low temperatures on contamination levels. One study[224] examined the effects of a number of hot water treatments on *Campylobacter* levels on carcasses, namely post-scalding, immersion for 28 seconds in water at 60°C, and spraying with water at 70°C. The treatments were chosen because they did not obviously change carcass appearance but they did not reduce *Campylobacter* levels. This is probably associated with the ability of this pathogen to attach to chicken skin.[225] In contrast, another study found that spraying with water at either 55 or 60°C did reduce the numbers of *C. jejuni* by circa 0.8 $\log_{10}$ per carcass.[226] This study used artificial contamination, and attachment to carcass surfaces is likely to be different from the natural situation. Scalding at 60°C reduced the numbers of *Campylobacter* on chicken skin by > 2 $\log_{10}$.[227] In the UK, however, most carcasses are scalded at water at circa 50°C, because they are destined for the fresh market. The above study found that scalding at this temperature had no effect on skin contamination levels with *C. jejuni*. The immersion of artificially inoculated broiler skin in water at 75, 80 or 85°C for 10 seconds caused a significant reduction in *C. jejuni* levels, as did immersion for 20 seconds in water at either 80 or 85°C. This study also investigated the effects of exposure to atmospheric steam at 90°C for either 12 or 24 seconds. The former had no effect on *Campylobacter* levels while the latter did bring about a significant

fall in bacterial numbers.[228] The authors state that all treatments caused visible damage to the outer epidermal skin tissue. These data, and those above, would suggest that treatment with high temperature is unlikely to be adopted for chickens to be sold with the skin on. Very similar results were obtained from a recent study in the UK.[229] This work also highlights a need to examine the effects of attachment on the heat resistance of *Campylobacter* spp.

4.53 A potentially more effective and commercially acceptable carcass treatment is the application of low temperature such as freezing, particularly where this could be applied to carcass surfaces in a transient manner so that carcass quality is not impaired. The storage of beef trimmings inoculated with *C. jejuni* at minus 18°C for seven days caused reductions in pathogen levels of between 0.22-2.2 $\log_{10}$ cfu/g.[230] In a recent investigation, the effects of freezing on the numbers of *C. jejuni* on artificially contaminated chicken wings were examined. Storage at either minus 20°C or minus 30°C for 72 hours reduced pathogen numbers by 1.3 and 1.8-$\log_{10}$ cfu/g, respectively. The super-chilling of wings in liquid nitrogen, so that the meat did not fall below minus 3.3°C, caused *C. jejuni* reductions of 0.5 $\log_{10}$ cfu/g on wings held at minus 80°C, 0.8 $\log_{10}$ cfu/g on wings held at minus 120°C, 0.6 $\log_{10}$ cfu/g on wings held at minus 160°C and 2.4 $\log_{10}$ cfu/g on wings held at minus 196°C.[231] It is of interest that the freezing of chicken carcasses was one of the factors identified as being associated with a reduction of human *Campylobacter* cases in Iceland.[232] During 1999 in Iceland, domestic cases of campylobacteriosis reached peak levels. Approximately 62% of broiler carcass rinses were contaminated with *Campylobacter* in 1999, but during 2000, only 15% of the broiler flocks tested *Campylobacter* positive. In 2000, carcasses from flocks which tested positive on the farms at 4 weeks of age were subsequently frozen prior to distribution. It was suggested that carcass freezing, in combination with other measures such as public education and enhanced on-farm biological security measures, contributed to the subsequent large reduction in poultry-borne campylobacteriosis.

4.54 All of the processing aids discussed above suffer from some defect or other e.g. they are not permitted to be used under EU law, they are not very effective in reducing *Campylobacter* loadings, or they are unacceptable to consumers. Other possibilities, like the use of ultra violet and electron beam radiation, are being explored, and their efficacy in a commercial setting and acceptability to consumers remain to be adequately demonstrated. While we remain firmly of the view that the focus of measures to combat *Campylobacter* should be centred on the farm, we do not seek to discourage the development of new technologies, either in terms of carcass treatments, or in innovative approaches to improved hygiene at slaughter. In this latter connection, the ACMSF *Campylobacter* Working Group received information from Meyn BV about its work to improve hygiene at slaughter. The company's aim is to bring this about through a combination of better

management of feed withdrawal, and improved mechanical processing of birds at slaughter. This involves reducing faecal contamination during scalding and plucking, and improving the efficiency of evisceration. The company has developed processing line equipment which, it claims, under specified conditions yields reductions of 60-90% in Enterobacteriaceae on the skin after plucking, 70-95% in *Campylobacter* on the skin after plucking, and 50-95% in Enterobacteriaceae in scald water, compared with an identical processing line not containing its equipment.

Control of *Campylobacter* spp. in extensive chicken production

4.55 The focus of this Chapter is enhancing biosecurity as a way of reducing *Campylobacter* infection in housed birds. This reflects the importance of intensive production (some 96% of the 1.2 million tonnes of chicken meat produced annually in the UK), and the fact that robust biosecurity regimes are more easily applied in the intensive production setting. We recognise that extensive production (free range and organic) is now a significant, albeit relatively minor, feature of the UK market, and we give this some attention in Annex E. Organic and free range production systems place greater emphasis on giving birds access to the outdoors. Enhanced biosecurity measures which help reduce *Campylobacter* infection in intensive production systems may therefore be less effective when used in extensive production systems. There is general scientific agreement that the environment is the principal source of *Campylobacter* spp. in poultry, and it is not unreasonable to expect that birds with regular access to the external environment will come into more frequent contact with *Campylobacter*.

4.56 There is some evidence that chickens which have access to the external environment are more likely to be *Campylobacter*-positive than intensively-reared birds. As yet unpublished Food Standards Agency-funded research points in this direction as do studies from Denmark and elsewhere.[137,233-236] However, because this evidence is not comprehensive, we believe that it would ultimately benefit consumers if structured surveillance were carried out in the UK both of the prevalence of *Campylobacter* in extensively-reared broiler flocks and the *Campylobacter* status of extensively-produced (including free range and organic) chicken meat.

Conclusions

4.57 It is becoming clear that control of *Campylobacter* on-farm is now a practical proposition, at least with birds that are housed. We brought this view to the attention of the Food Standards Agency in September 2002 to assist the Agency in developing its *Campylobacter* strategy.[3] The first commitment must be to rigorous biosecurity, combined with high standards of stockmanship and attention to good flock health and stress control. This

will involve such measures as restricting farm visits to essential personnel; ensuring visits are undertaken as hygienically as possible; and appropriate staff training on flock infection. The control of *Campylobacter* on-farm presents a greater challenge than that associated with the control of *Salmonella*.

4.58 Our *Campylobacter* Working Group received different views, both formally and anecdotally, about the possibility of the UK poultry industry adopting 'Scandinavian style' systems of on-farm biosecurity. Where these systems have been trialed in the UK, they have been seen to be successful, although industry argues that long-term maintenance would be difficult. While Scandinavian and UK systems of production and control measures do differ in some respects, reflecting the different sizes of the industries and the very different climates, we firmly believe that the application of biosecurity, such as changing footwear, and other hygiene measures, will either delay or prevent the entry of *Campylobacter* into broiler flocks and thus reduce the incidence of colonised birds. Studies in the Netherlands support this view.[177] Changing of footwear was found to be important in a UK context,[187] and another UK study found that frequent replenishment of boot dip disinfectant was one of a few factors which reduced broiler flock infection by over 50%.[186] In the future, given current research effort, it may be possible to supplement biosecurity with pre- or probiotic approaches, competitive exclusion, and/or vaccination.

4.59 In addition, it is clear that a well-run broiler farm can reduce the incidence of *Campylobacter* through adherence to a number of key principles. It should:

- be species mono-specific (ie. farm only chickens);
- supply the birds with water of potable quality;
- properly clean and disinfect houses after flock removal, which should include disinfection of the water supply system;
- protect the house from entry by wild birds and rodents;
- supply feed which has received treatment sufficient to have eradicated *Salmonella* (and, hence *Campylobacter*), and protect it from re-contamination;
- only carry out thinning if done in association with proper crate washing (so that crates are not contaminated with *Campylobacter* spp. or other pathogenic microorganisms) and proper biosecurity measures covering eg. clothing and footwear;

- ensure that transport crates and vehicles are cleaned and disinfected properly on every occasion;
- maintain general biosecurity and hygiene barriers at a high level, to prevent infection from the farm environment;
- only allow essential visits into the poultry houses; and
- make sure all personnel including visitors follow the hygiene rules.

4.60 We strongly believe that concerted effort is needed by industry to improve the microbiological safety of thinning. If this cannot be achieved, then the case for discontinuing the practice, and taking the necessary measures to protect the welfare of stock, becomes very strong.

4.61 In risk assessment terms, a lower incidence of *Campylobacter* in broiler flocks is likely to be reflected in lower numbers of the organism in individual birds in the flock and subsequently on finished carcases. Reducing the number of *Campylobacter*-positive flocks can also be expected to have a significant impact on the numbers of contaminated carcases leaving the processing plant. Flock testing will facilitate the scheduling of slaughter of known positive flocks allowing birds from such flocks to be processed at the end of the day immediately prior to cleansing of the plant, reducing the opportunity for cross-contamination from the carcases of these birds. It would also offer the option of directing the processed carcases from positive flocks to heat treatment or freezing if these were found to be helpful in reducing *Campylobacter* loadings.

4.62 We accept the advice we have received from various parts of the poultry industry that broiler chicken production is extremely price competitive and that the industry is faced with continuing threats of import penetration.

4.63 We do recognise that many of the measures for controlling *Campylobacter* in chicken imply additional production costs. However, there is increasing evidence that there are direct links between the general health status of birds and their susceptibility to *Campylobacter* infection. In addition, the maintenance of good flock health conveys economic benefits. Measures put in place for the control of *Campylobacter* might also help reduce the risk of introducing other infections into the flock.

4.64 In order to be able to evaluate the effectiveness of measures to tackle *Campylobacter*, good quality data are needed on the *Campylobacter* status both of flocks and retail product. Flock prevalence studies are an essential feature of any evaluation process, to establish a baseline and to monitor

progress under commercial conditions. We believe that Defra should organise such studies. In addition, we assume that the Food Standards Agency will continue to use routine surveillance of retail chicken for *Campylobacter* to assess the effectiveness of *Campylobacter* reduction programmes. The potential value of industry data as an output measure should not be overlooked even if, for reasons of commercial sensitivity, such information cannot be made publicly-available outside the FSA. We discuss the valuable contribution a standardised approach to typing can make to tracing sources and routes of transmission of human *Campylobacter* infection in Chapter 9.

4.65 We recognise that free range and organic chicken production is now a small but significant feature of the UK market. Given the importance of the environment as a source of *Campylobacter*, we think it likely that chickens reared extensively will come into more frequent contact with *Campylobacter* and that robust biosecurity arrangements aimed at reducing the exposure of birds to *Campylobacter* spp. will be more difficult to apply in extensive production systems. It is important that consumers are aware of this, not least because one of the main reasons given for buying free range and organic chicken is that they see it as a healthier product. We believe that information based on structured UK surveillance of *Campylobacter* infection in extensively-reared broiler flocks and the *Campylobacter* status of extensively-produced, including free range and organic, chicken meat would be valuable in informing consumer choice. Means also need to be identified of controlling *Campylobacter* in extensive production systems.

Recommendations

4.66 Our principal recommendation is that the Food Standards Agency utilises the conclusions we have drawn to intensify its work with the poultry industry and other stakeholders to achieve wider acceptance that *Campylobacter* control of housed birds is now possible. A primary aim should be to develop an industry-wide programme to spread the "good farming" practices and biosecurity measures which lie at the heart of the matter. (Priority A)

4.67 We recommend that the FSA, in collaboration with Defra, as appropriate, should explore with industry the options for modifying thinning practices to reduce the threat to the biosecurity of broiler farms. If the necessary improvements cannot be made, the FSA and Defra should explore with industry the conditions which would allow the practice of thinning to be discontinued, notwithstanding the economic pressures to which industry has drawn attention. (Priority A).

4.68 If thinning is to continue, crate washing and other biosecurity measures (including clothing and footwear) need urgent improvement. We recommend that the FSA pursues this with stakeholders. (Priority A)

4.69 In order to facilitate evaluation of the effectiveness of *Campylobacter* reduction measures, and to improve controls at slaughter, we recommend that Defra carries out surveillance of *Campylobacter* in broiler flocks. We also recommend that the FSA continues to perform routine surveillance of *Campylobacter* in retail chicken. (Priority A)

4.70 Extensive chicken production is a minor but nevertheless important feature of the UK market. We believe that consumers would benefit from knowing more about the *Campylobacter* status of this type of product. We therefore recommend surveillance:

- **by Defra to determine the prevalence of *Campylobacter* in extensively-reared flocks and the *Campylobacter* spp. involved; (Priority B)**

- **by the FSA to determine the *Campylobacter* status of free range, organic and other extensively-produced chicken meat on retail sale in the UK. (Priority B)**

4.71 We also recommend further research into how *Campylobacter* can be more effectively controlled in extensively-reared chickens. We note that the FSA is already considering funding research in this area and welcome the fact that the Agency has invited Expressions of Interest from researchers.[237] (Priority B)

Chapter 5

Measures to prevent *Campylobacter* contamination of chicken meat in Scandinavia

Introduction

5.1 Animal and human health surveillance data, together with research reports, suggest that the incidence of *Campylobacter* in commercially-reared chickens in Scandinavia is lower than in the UK. In order to ascertain whether this was true, and to try to learn from the Scandinavian experience, a sub group of *Campylobacter* Working Group members, comprising Professor Humphrey, Professor Johnston and Mr Kyriakides, together with the Group's Scientific Secretary, Dr Back, visited Denmark and Norway from 17-21 November 2002. The sub group deemed the visit a great success as it allowed the members to gain first hand knowledge of poultry production in the two countries and to have detailed discussions with scientists, and with government and poultry industry officials. Sub group members wish to place on record their very grateful thanks to all the people, in both countries, who helped to arrange the visit and particularly to those whom the sub group met during the visit.

5.2 A report on the visit was submitted to the Food Standards Agency in January 2003 (see Annex C). Details are given in the following paragraphs. Information about the situation in Sweden, kindly provided by Dr Eva Berndtson (an ACMSF *Campylobacter* Working Group member) is also included in this Chapter.

Denmark

5.3 The first day of the visit to Denmark was taken up with meetings and presentations, while the second day was devoted to a visit to a broiler farm and a processing plant.

Human *Campylobacter* infection in Denmark

5.4 There were 4,620 recorded cases of human *Campylobacter* infection in Denmark in 2001, although the true figure is thought to be much higher, and similar to the incidence in the UK. The most significant sources of infection are:

- poultry meat;

- pork and beef;
- polluted drinking water; and
- contact with cats and dogs.

5.5 The sub group was also presented with details of a contemporary epidemiological study identifying the following risk factors (Table 5.1):

Table 5.1: Contemporary epidemiological study identifying risk factors

Risk factor	Odds ratio
Under-cooked poultry meat	4.5
Travel to a foreign country	2.5
Raw milk consumption	2.3
Red meat consumed at BBQs	2.3
Grapes	1.6

5.6 There is a much more pronounced summer peak in human infection than in the UK. The consumption of poultry meat is a significant risk factor and the Danes have carried out a risk assessment which shows that, where the number of *Campylobacter* on chicken carcasses is reduced by freezing or other means, the risk of human infection is also reduced.

Campylobacter in Danish broiler flocks

5.7 All poultry flocks in Denmark are subject to surveillance to determine their *Campylobacter* status. Standard protocols are used throughout Denmark, Norway and Sweden. Control of *Campylobacter* in broiler flocks is closer to the current UK position (and less well developed than in Norway).

5.8 Some scepticism was expressed in Denmark about the possibilities for on-farm control. Very hot Danish summers present particular difficulties. It is not uncommon for some broiler houses to be left open in summer for welfare reasons, and this would undermine biosecurity. The current aim is therefore to reduce flock colonisation rather than to eliminate it. Probably reflecting perceived difficulties in on-farm control, there is a greater focus in Denmark on intervention during or after processing. *Campylobacter* is thought to be particularly sensitive to freezing and work is in hand on the effects of freezing at –18ºC for 10 days. The efficacy of heat treatment (75ºC for 15 seconds) is also being investigated. Current work suggests that these treatments bring about a 1.95 and 1.6-log reduction respectively in *Campylobacter* contamination levels.

Broiler farm visit

5.9 The sub group visited a farm supplying a major poultry processor, which was said to be typical of a good broiler farm in Denmark. It was a contract farm and had 7 houses, each containing 31,000 birds. The farmer operated an all-in all-out system, although it took a number of days to clear the site. The birds were approximately 21 days of age at the time of the visit, and mortality was higher than usual, due to a combination of Gumboro disease and a vitamin B deficiency in the parent flock. The farm was in good order and, although the houses were over 30 years old, they were in good condition. There were approximately 5-10 metres between houses, and the site was coated with coarse gravel, which was routinely sprayed with weed killer. There are broiler farms of a comparative standard in the UK.

5.10 The only intervention in place was a physical hygiene barrier about 40 cms high in each house with associated boot/protective clothing change. A wash hand basin was located away from the hygiene barrier and the house was not entered via an enclosed anteroom (in contrast to the situation observed at the Norwegian broiler farm visited by the sub group – see below). This set up was closely related to typical UK production, except for the hygiene barrier which is largely absent in the UK. The rather rudimentary hygiene precautions are sufficient to protect flocks outside of the summer months.

Poultry processing plant visit

5.11 The sub group visited a processing plant very similar to most in the UK. The company markets *Campylobacter*-free chickens, for which Danish consumers are prepared to pay a price premium. Danish legislation covering *Campylobacter*-free status requires that "*the flock shall be controlled to give a 95% guarantee that less than 1% of birds are infected with Campylobacter".* Three hundred samples per flock must be tested, although the company examined 500. The company has been involved in the development of a PCR method to provide information on *Campylobacter* status within 5 hours. The testing regime is as follows (Table 5.2):

Table 5.2: Commercial PCR testing protocol

Control	Samples	Analysis method	Comments
At the farm	3 x fresh faeces	PCR	If *Campylobacter*-negative, the flock can be slaughtered as *Campylobacter*-free.
At the slaughterhouse	20 samples of cloacal swabs	PCR	If no *Campylobacter* is detected, the products can be sold with the *Campylobacter*-free label.

5.12 Testing generates a variety of actions. Details are as follows:

Table 5.3: Actions resulting from PCR testing

Positive result	Action
Farm samples	Flock slaughtered and packed without *Campylobacter*-free label.
Slaughterhouse samples	Chickens previously shown to be negative on the farm will be re-packed without the *Campylobacter*-free label.
Isolation of *Campylobacter* from farm samples, post-cleaning	Extra cleaning and extended control is carried out. If *Campylobacter* is detected repeatedly, the control programme will be evaluated with the farm veterinarian.

Norway

5.13 Much of this leg of the visit was taken up with meetings with people largely responsible for the implementation of the Norwegian Action Plan Against *Campylobacter* in Broilers. Sub group members also gave presentations to an invited audience of around 100 people and also attended an official reception at the residence of the British Ambassador.

Human *Campylobacter* infection in Norway

5.14 There has been a marked increase in the number of human *Campylobacter* cases in Norway since 1997, the annual incidence being around 100 cases per 100,000 of the population. There is an approximate 50:50 split between the number of cases acquired in Norway and those acquired abroad. There is a very marked peak in the incidence of human infection, with approximately 75% of cases occurring in July, August and

September. It is thought that many more cases are caused by water in Norway than in the UK. The consumption of poultry purchased raw is among the principal risk factors although, unfortunately, authoritative data on the level of chicken-associated human cases prior to the introduction of broiler intervention arrangements are not available. Principal vehicles of infection are:

- the consumption of non-disinfected water;
- the consumption of poultry purchased raw;
- attending outdoor barbeques; and
- professional contact with animals.

5.15 Given the rising incidence of human campylobacteriosis, and the association with the consumption of poultry meat, Norway has introduced an Action Plan Against *Campylobacter* in Broilers. The plan was developed by the Norwegian Zoonosis Centre and has three goals, namely to:

- reduce the human exposure to *Campylobacter* from Norwegian poultry;
- improve food safety; and
- reduce the incidence of human campylobacteriosis associated with Norwegian poultry.

5.16 The action plan has three elements:

- surveillance of live animals and animals at slaughter, in accordance with WHO recommendations;
- surveillance of poultry meat products;
- follow up of *Campylobacter*-positive farms, comprising standardised consultations and the introduction of measures to reduce flock infection, namely the disinfection of drinking water and the introduction of physical hygiene barriers;
- a farm-based research programme to identify risk factors for *Campylobacter* infection in flocks.

Campylobacter in Norwegian broiler flocks

5.17 The industry is approximately 10% of the size of that in the UK and, in general, birds are killed at 32-33 days of age although, at the plant visited by the sub group, older birds (c42 days) were also being processed. Norway has a national programme for the surveillance of *Campylobacter* in poultry flocks, which is funded by the Government and the industry. Ten composite faecal samples are collected on the farms 4-8 days before slaughter. If these samples are *Campylobacter*-positive, the birds are subject to hygienic slaughter at the end of the day. The carcasses are either heat-treated or frozen for five weeks. The farmers will also receive a consultation. All flocks are also sampled at the slaughterhouse, with 10 cloacal swabs being taken. In some plants, breast feather swabs are also taken in the processing plant immediately after killing. If these samples are *Campylobacter*-positive, the farmer will receive an advisory visit.

5.18 Data from Norway's national surveillance programme indicate that, in 1991, the overall figure for *Campylobacter*-positive flocks was 18%, although there is very marked seasonal variability. By 1998, this figure had fallen to 4%. The most recent surveillance data seen by the ACMSF (covering 2001-2002, and including two summers) show an annual, on-farm incidence figure of 7.6%. Many flocks became positive in the last week of life, a phenomenon becoming increasingly common in the UK. The following data are taken from the latest surveillance:

- 3,444 flocks from 526 farms were surveyed;
- 133 farms (25%) were *Campylobacter*-positive;
- 186 flocks (5.4%) were *Campylobacter*-positive;
- 49% of the positive flocks were only detected at slaughter;
- 71% of farms delivered only 1 *Campylobacter*-positive flock; and
- 7% of farms delivered 3 or more *Campylobacter*-positive flocks.

5.19 As with human infection, there is very marked seasonality, with some 90% of the positive flocks being identified in the summer months. The following table gives data from current risk analyses.

Table 5.4: Factors associated with the risk of *Campylobacter* in broiler flocks

Variable	Category	Odds ratio
Using tractor to place litter in the broiler house	Yes No	3.1 1.0
Physical hygiene barrier at entrance to chickens	Yes No	1.0 4.2
Routines for hand-washing	Always Never/sometimes	1.0 3.3
Water source	Private well Other private source Public source	3.6 2.1 1.0

Broiler farm visit

5.20 The sub group visited a typical Norwegian broiler farm comprising 1 house containing approximately 11,000 birds. It was not possible to examine the area surrounding the broiler house as it was covered in snow. The house was entered via an anteroom, which had 3 rooms, each with doors, coming off it. One room served as an office and had a window through which the flock could be observed. Access to the flock was via a room on the other side of the anteroom, in which a physical hygiene barrier had been placed. There were dedicated overalls and footwear on the bird-side of the barrier. This room also contained a wash hand basin which the farmer used before putting on his protective clothing and footwear. All the evidence available to the sub group would suggest that this simple intervention is sufficient to protect the birds from *Campylobacter* colonisation in spring, autumn and winter and, to some extent, in summer. Some UK poultry companies have agreed to undertake collaborative research to examine whether the Norwegian system of hygiene barriers could deliver the same benefits in this country. A small trial found that one UK farmer was able to produce 5 *Campylobacter*-negative flocks in succession.

5.21 The sub group visited a poultry processing plant which was typical of most in Europe, and which employed no devices which were not already in use in the UK. The plant was smaller and much tighter for space than UK plants. Water usage seemed higher than in the UK. Also, in contrast to the UK, birds were spray-chilled with cold water. Although Norway does not market *Campylobacter*-free poultry at retail, the goal is to reduce the level of *Campylobacter* in broiler chickens at slaughter to as close to zero as possible.

Campylobacter in fresh poultry products

5.22 The prevalence of *Campylobacter* contamination in fresh poultry products in Norway ranged between 4 and 10% over the period 1995-98.

Further surveys of fresh poultry products were carried out in 2001 (at production facilities) and 2002 (in shops). Just over 1,000 samples were taken in each survey. *Campylobacter* prevalence was <10% in 2001 and around 2% in 2002.

Sweden

5.23 Sweden is a very important element in any consideration of Scandinavian broiler production. The ACMSF *Campylobacter* Working Group was very fortunate to have as a member, Dr Eva Berndtson who is *Campylobacter* Consultant to the Swedish Poultry Association. Dr Berndtson has been able to supply information about the situation in Sweden.

Human *Campylobacter* infection in Sweden

5.24 Human cases of *Campylobacter* infection in Sweden are increasing. Five-year trend data are given in Table 5.5.

5.25 Of the cases in 2001 where information is available about the country of infection, 4,884 were infected abroad. In 861 cases, it was not possible to determine the country of infection. The countries most commonly identified as the source of infection were Thailand (1,045 cases), Spain (984), Morocco (310), Turkey (212), Tunisia (176), Greece (164), France (154), India (143), Indonesia (135) and Egypt (113).

Table 5.5: Notified cases of human *Campylobacter* infection in Sweden

Year	Total	Acquired in Sweden	Total incidence (100,000 population)
1997	6,881	1,430	77.8
1998	7,397	2,506	83.6
1999	7,669	2,128	86.5
2000	8,405	2,443	94.6
2001	8,577	2,832	96.3

Source: Swedish Institute of Infectious Disease Control

5.26 With the exception of some large waterborne outbreaks, chicken meat is regarded as the most common source of *Campylobacter* infection acquired in Sweden. During 2002, there were 2,453 clinically reported cases acquired in Sweden. In most of these, no suspected source of infection was identified. For those where a suspected source was identified, the most common risk factors mentioned were:

- eating chicken meat (351 cases);
- poultry contact at work or at home (46);
- lake/stream water (31);
- domestic well water (27); and
- raw milk (9 cases).

Campylobacter in Swedish broiler flocks

5.27 Table 5.6 shows the percentage of Swedish broiler flocks positive for *Campylobacter* over the period 1993-2000. Data are also shown for part of the year 2001. The peak periods for infection, based on blocks of 4 weeks, runs from periods 7 to 10, during which the number of broiler flocks positive for *Campylobacter* ranged from around 10% to nearly 40%. Annualised flock prevalence data are given in Table 5.7.

Characteristics of Swedish broiler industry

5.28 At the time of writing, the Swedish broiler industry comprises 7 companies with 8 slaughterhouses and a total of 124 farms with approximately 500 broiler houses in total. Average flock size is around 30,000 birds (maximum 50,000). The newer farms generally have flocks of 50,000 birds and 2-4 houses or compartments.

5.29 Broiler houses are classified for welfare purposes in Sweden and stocking density is a key feature of the classification arrangements. Only the best houses are permitted to be stocked to the maximum density (36kg/m^2). Most houses are stocked to a density of at least 33 kg/m^2. Stocking density in houses with low welfare scores is restricted to 20kg/m^2.

5.30 As noted in Chapter 4, data[190,193] suggest that *Campylobacter* spp. in dry litter may be less infectious than in wet litter. A reduction in *Campylobacter* infection in flocks has been seen to correlate with litter dryness, and further improvements were seen when Swedish farmers began using scoring of foot pads as a parameter for adjusting the density of birds in a shed. The checking of the feet of birds at slaughter is a feature of the Swedish classification system. If the foot pads are in poor condition, lower density stocking is imposed for subsequent flocks. This serves as an incentive on farmers to keep litter very dry.

Table 5.6: *Campylobacter* in Swedish broiler flocks

4 week period No.	Percentage flocks *Campylobacter*-positive								
	1993	**1994**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**
1	9	6	9	0	3	5	1	3	10
2	10	9	13	1	2	4	1	2	8
3	6	3	12	3	2	5	1	1	9
4	11	8	9	5	2	2	1	2	6
5	5	5	7	4	2	7	4	8	11
6	7	12	10	6	2	5	12	12	12
7	18	14	8	13	9	14	15	18	7
8	28	25	18	14	16	12	25	18	
9	23	41	37	18	25	25	22	20	
10	17	24	24	22	27	19	21	14	
11	16	14	16	12	17	15	14	11	
12	14	4	11	10	12	2	1	12	
13	20	12	7	11	6	3	4	7	

Source: Swedish Poultry Meat Association

Table 5.7: Percentage of Swedish broiler flocks positive for *Campylobacter*

Year	% broiler flocks *Campylobacter*-positive	Year	% broiler *Campylobacter*-positive
1992	13.3	1997	9.8
1993	12.4	1998	9.1
1994	13.6	1999	9.2
1995	14.3	2000	9.9
1996	9.3		

Source: Swedish Poultry Meat Association

5.31 Biosecurity features include the requirement to change clothing and footwear at the entrance to each house, and an all in-all out production system across the entire farm. While there are significant differences in the frequency of *Campylobacter* flock infection between companies, taking year 2000 as an example, almost half of all farms had no *Campylobacter*-positive flocks.

5.32 A feature of Sweden's programme to reduce the prevalence of *Campylobacter* in broiler flocks is that the worst affected farms receive visits

from veterinary advisers. The overall aim of the programme is to reduce flock prevalence to below 2%. This would open the way for positive birds to be identified for special processing (eg. heat treatment). For this to work effectively, there will be a need for a quick and reliable method of identifying *Campylobacter*-positive flocks before slaughter. There is also a potential logistical difficulty in that there are often significant distances between broiler farms and slaughterhouses. Travelling time regulations thus make it difficult to re-route positive birds.

Campylobacter in Swedish fresh poultry products

5.33 The Committee has seen no published data on *Campylobacter* contamination levels in fresh poultry products at retail in Sweden. However, levels are thought to reflect those seen at slaughter plants (ie. 10-17%).

Conclusions from Denmark visit

5.34 Overall, the sub group concluded that the current situation in Denmark was quite close to that in the UK, although intervention has been attempted for much longer. The Danes seemed to have derived a real benefit, in terms of the quality of data produced, from closer integration of the human and animal health surveillance systems. It was also apparent that regular national testing of poultry flocks yielded important information about *Campylobacter* prevalence and seasonality, as well as about geographical differences in colonisation rates. The Danish research community has offered access to performance and flock health data on 25,000 flocks, which can be correlated with *Campylobacter* status.

Conclusions from Norway visit

5.35 The sub group felt that Norway provided some useful indications of what could be achieved by targeted on-farm intervention. Physical hygiene barriers seemed a cheap and effective counter-measure which the UK industry should be pressed to adopt as a matter of urgency. The rather different epidemiology of infection in Norwegian broilers, compared to those in the UK, could indicate a particular source of infection in the summer and the possible involvement of contaminated air in its transmission. The potential for airborne transmission on farms may need further investigation. This could necessitate some quite detailed research.

Overall conclusions from Denmark/Norway visits

5.36 The ACMSF's overall conclusions drawn from the sub group's visits to Denmark and Norway are that:

- nothing that the sub group saw in either Denmark or Norway served to undermine the Committee's views, set out in

Chapter 4, on the feasibility of the on-farm control of *Campylobacter* in housed chickens;

- indeed, the Norwegian experience especially offered further encouragement that on-farm control in housed birds is achievable on a commercial scale;
- Denmark appears to have established a premium market for *Campylobacter*-free chicken;
- Norway has succeeded in getting the contamination rate for fresh chicken products in retail outlets down below 10%;
- the UK broiler industry still has some catching up to do but is, for the most part, on the right track;
- however, the UK industry needs to be encouraged to maintain its best endeavours;
- opportunities for collaboration between researchers here and those in Denmark and Norway were identified;
- some thought needs to be given to the efficacy and wider implications of heat treating or freezing *Campylobacter*-positive carcasses;
- the potential for airborne transmission of *Campylobacter* on farms may need further investigation but could necessitate some quite detailed research.

Conclusions from Sweden's experience

5.37 Sweden has succeeded in reducing overall *Campylobacter* infection in flocks to below 10%. It is encouraging that, within this figure, around half of all broiler farms were able to keep *Campylobacter* out of flocks completely. The methods used to achieve these results (eg. robust biosecurity, dry litter) are not innovative and are readily applicable to the UK setting. This information about the situation in Sweden provides further support for the observations made and the conclusions drawn by members of the sub group following their visit to Denmark and Norway.

Chapter 6

Campylobacter in poultry other than chicken

Introduction

6.1 In relation to poultry, chickens are the main focus of this Report. Also, the market share for broiler birds (860m) in the UK is higher than that of turkeys (29m) and ducks (20m) and broilers are consumed more often. This reflects the importance of chicken meat in terms of production, trade and consumption. However, all commercial poultry species can be colonised with *Campylobacter* spp. and products derived from them can also be contaminated with this pathogen.

Campylobacter prevalence in ducks and turkeys

6.2 A survey in the USA found that 88% of **ducks** were positive for *Campylobacter jejuni* compared to 24% for chickens.[238] A study in Kenya found *Campylobacter* in 29% of healthy ducks and in 52% of healthy chickens.[239] Surveillance in Portugal demonstrated the presence of *Campylobacter* spp in 60 and 41% of chickens and ducks respectively.[240]

6.3 *Campylobacter* spp. are also found in **turkeys.**[241,242] A study on one farm in the UK found that all turkeys examined were *Campylobacter*-positive between 14-21 days after hatching.[243]

Campylobacter in foods

6.4 Surveys of foods at retail outlets also permit a comparison of *Campylobacter* contamination levels in different types of poultry. A US survey recovered *Campylobacter* from 57% of chicken samples and from 17% of game bird samples, but *Campylobacter* was found only infrequently in turkey.[244] Another US survey found *Campylobacter* in 71% and 14% of chicken and turkey samples respectively.[245] Another US survey compared anti-microbial resistance profiles of poultry-derived *Campylobacter* isolates and found higher levels of resistance in strains from turkey compared to chicken.[246]

Conclusions

6.5 Such evidence as we have seen suggests that all commercial poultry species are as susceptible as chicken to *Campylobacter* colonisation. However, we note that there appears to be little hard information available about the UK situation, and most of the data quoted in this Report come from abroad.

Recommendation

6.6 We recommend that, in addition to the work it is doing on chicken meat, the FSA carries out surveillance to establish the *Campylobacter* status of other types of poultry meat on retail sale in the UK. (Priority A)

Chapter 7

Measures to prevent *Campylobacter* contamination of meat other than chicken and other poultry meat

Introduction

7.1 As noted in Chapter 3, while poultry meat is an important source of human *Campylobacter* infection, many studies point to numerous other sources and vehicles of infection.

Campylobacter levels in animals

7.2 *Campylobacter* spp. frequently occur in the gut flora and faeces of animals used for food production such as cattle, pigs and sheep. Information on the proportion of these animals carrying the organism is not extensive and what there is must be treated with some caution as a variety of methods and sampling regimes are likely to have been used. However, it is clear that rates can be high, in some cases as high as those reported for chickens. One source of information is the annual European Commission Report on Trends and Sources of Zoonotic Agents in the European Union and Norway.[247] While information on carriage rates of *Campylobacter* is limited, a handful of European countries has provided data indicating carriage rates ranging from 0.4-72.4% in cattle, from 45.3-94.5% in pigs, and from 13-24.8% in sheep and lamb. The levels between countries, and indeed within countries when different years are compared, vary significantly.

7.3 Information on faecal carriage rates in the UK can be found in the annual Zoonoses Report published by Defra. The 2000 report[248] gives the results of a survey of cattle, sheep and pig faeces in Great Britain, carried out in 1999/2000. This found:

- 94.5% of 860 pigs positive for *Campylobacter*, the predominant species being *Campylobacter coli* (84%);
- 24.5% of 891 cattle positive for *Campylobacter*, with *C. coli*, *Campylobacter* jejuni and *Campylobacter lari* accounting for more than half the positives;
- 17% of 973 sheep positive for *Campylobacter*, *C. jejuni, C. coli* and *C. lari* accounting for over 90% of positives.

7.4 The results outlined above provide a baseline against which to measure any changes in prevalence and types of *Campylobacter* when similar surveys are carried out in the future. We are aware that Defra, in association with other Government Departments, is in the process of repeating this survey. We welcome this. It is important that up-to-date information on carriage rates in food animals is maintained.

Campylobacter levels in meat

7.5 Leaving aside chickens and other poultry, although it is clear that there can be high gut and faecal carriage rates in animals, the available information indicates that these often do not carry through to the associated meats on retail sale. However, the data are very variable and high figures have been reported for some products. For example, 23.6% of 127 samples of beef meat were positive for *Campylobacter* in a 1989 UK survey.[249] A further UK study[250] has reported contamination rates of 72.9 and 71.7% respectively in lambs' and pigs' liver. Data from other surveys carried out in the UK and the USA show *C. jejuni* and *C. coli* in a variety of retail meats ranging from 0 to 18.4%.[249] A study of campylobacters in, *inter alia*, samples of food on retail sale in the Reading area of the UK produced *Campylobacter* positives in a variety of meats sampled, ranging from 2.3 to 47%.[251] In Belgium, sampling of retail cuts of pork over the period 1997-1999 produced prevalence rates of between 2.6 and 12.5% of samples. In a Belgian survey of retail beef in 1997, 5% of samples were *Campylobacter*-positive.[247] A US study[245] reported low levels of contamination in pork (1.7%) and beef (0.5%). The ACMSF *Campylobacter* Working Group was provided with data from surveys, carried out by a leading UK multiple food retailer, of *Campylobacter* in the company's raw meat products on retail sale. No *Campylobacter* was found in 147 samples of fresh retail cuts of beef (53 samples), lamb (69) and pork (25) in February 2002. Nor was *Campylobacter* detected in 56 samples of fresh and frozen retail minced/reformed beef (41, of which 12 frozen), lamb (3, all fresh) and pork (12, all frozen) in March 2002. Finally, *Campylobacter* was also absent from 102 samples of fresh retail whole cuts of beef (39), lamb (36, of which 6 frozen) and pork (27) in September 2002.

7.6 Given the variations noted in the prevalence of *Campylobacter* in retail meat samples, it is very difficult to form any meaningful view on the risk to public health in the UK from such products. What is required is large-scale, structured surveillance of *Campylobacter* in red meat on retail sale.

Control of *Campylobacter*

7.7 The fact that the high carriage rates in red meat animals prior to slaughter does not always carry through to the final product is not perhaps surprising. Compared to poultry, there are significant differences in the way that animals such as cattle, pigs and sheep are reared, transported and slaughtered. There are control measures in place which minimise faecal

contamination of hides and fleeces, and hence *Campylobacter* contamination of carcasses during dehiding and evisceration. That said, in comparison to other enteric organisms, *Campylobacter* is rarely found on carcasses. Although this is thought to be due to the surface conditions, it is possible that isolation methods used in studies are not optimal. We note that the Food Standards Agency (FSA) is investigating this question.

7.8 Implementation of control measures in the UK is the responsibility of the Meat Hygiene Service (MHS) in Great Britain and the Department of Agriculture and Rural Development in Northern Ireland (NIDARD). MHS and NIDARD are responsible for ensuring that operators fulfil their duty to have appropriate hygiene controls in place at licensed slaughterhouses, cutting plants and cold stores handling red meat. Control measures comprise four main stages:

- *ante mortem* inspection of animals;
- checking on maintenance by the operator of hygienic process control throughout all stages of slaughter and processing;
- *post mortem* inspection of carcasses; and
- health marking.

Each of these stages plays an important role in minimising the risk to public health from pathogens such as *Campylobacter*.

Ante mortem control in cattle and sheep

7.9 In respect of *ante mortem* controls for cattle and sheep, a major advance in improving meat hygiene was the introduction in 1997 of the MHS Clean Livestock Policy (CLP).[b] The background to this was the recognition that, if the hide or fleece was contaminated with dung or dirt at the time of slaughter, there was a very real risk of the meat becoming contaminated with harmful bacteria. Even the highest standards of abattoir hygiene cannot be guaranteed to prevent contamination of the carcass and cross-contamination of nearby carcasses. Research results have shown that the dirtier the hide, the greater the potential for carcass contamination and the higher the human health risk. Wet hides and fleeces also increase the risk.

7.10 The CLP provides a cleanliness classification system which places animals presented by the operator for slaughter into one of five categories. Animals in categories 1 and 2, ie. those considered to be clean and dry, can be slaughtered for human consumption. Animals in categories 3 and 4 may only be slaughtered for human consumption after the animal has received

[b] The CLP does not apply to pigs and there are no categories of cleanliness. However, under the Fresh Meat (Hygiene and Inspection) Regulations 1995, an MHS Official Veterinary Surgeon may require the detention in a lairage, or prohibit the slaughter, of any animal which is so dirty it would be likey to prevent hygienic dressing operations if taken into a slaughterhouse.

special attention (eg. clipping or being allowed to dry in overnight lairage). Alternatively, other measures such as slowing the line speed to enable hygienic slaughter may be appropriate. Animals in category 5 are unsuitable for slaughter for human consumption. Such animals are killed separately and disposed of as an animal by-product. The CLP is operated by MHS and NIDARD staff who are able to reject for slaughter any animal that does not meet the required standards of cleanliness.

7.11 At the time the CLP scheme was launched, research was put in place to study the factors involved in producing cattle that were both visibly and microbiologically clean. The output from this work was the launch by the FSA in 2003 of an initiative on Clean Cattle and Meat Safety.[252] This initiative, which was produced in consultation with stakeholders, highlights 10 key messages for producing clean cattle. These are disseminated via promotional literature and a series of events aiming to provide advice to farmers, livestock hauliers, veterinarians, abattoir managers, butchers and retailers.

Hygiene control during slaughter

7.12 Compliance with hygiene legislation is the responsibility of plant operators. However, MHS and NIDARD staff work with plant operators to ensure that hygiene controls to minimise the risk of cross-contamination are maintained throughout the slaughter process. Specifically, the MHS and NIDARD enforce legislative requirements aimed at making sure that premises operate to recognised hygiene standards. Failure to meet these requirements may result in enforcement action against premises, and this could ultimately lead to prosecution and suspension and/or revocation of their licence to operate.

Hazard Analysis Critical Control Point (HACCP)

7.13 The ACMSF is a strong supporter of HACCP and we championed its cause in our Report on Poultry Meat.[171] One of the major changes in relation to hygiene in red meat plants was the introduction of The Meat (Hazard Analysis and Critical Control Point) Regulations 2002,[253] which require operators to put in place hygiene procedures based on HACCP principles and to undertake microbiological checks. To aid plants in introducing HACCP, the FSA has produced a range of materials, including:

- HACCP guidelines[252] – a booklet explaining what the seven principles of HACCP are and how the legal requirements can be complied with in general terms;

- Meat Plant HACCP Manual[252] – produced with the benefit of feedback from pilot plants, this manual has been sent to all operators and their Official Veterinary Surgeons (OVSs);

- a CD-ROM version of the manual containing extra material including video clips, sample documentation and some model HACCP plans.[252]

- newsletters – a number have been issued, providing advice and information on implementation of the Regulations.[252]

Microbiological testing

7.14 The Meat (HACCP) Regulations 2002[253] also introduce a requirement for microbiological testing in red meat plants, with the need to undertake both carcass tests (for Aerobic Colony Counts and Enterobacteriaceae*)* and surface tests (for Aerobic Colony Counts). The purpose of this testing is very much to look for trends, with plants using the results to identify the need to make improvements in slaughter hygiene or cleaning and disinfection processes. To assist operators and laboratories, the FSA has produced draft guidelines on microbiological testing – a booklet explaining how to undertake the sampling, testing calculation and expression of results.

Post mortem inspection and health marking

7.15 Individual carcasses are assessed through *post-mortem* inspection. Any visible faecal contamination must be trimmed off before a carcass can be presented as safe, wholesome and fit for sale for human consumption.

7.16 Fresh meat for sale for human consumption produced in licensed slaughterhouses must carry an official health mark. This indicates that the carcass has passed *ante* and *post-mortem* inspection and that hygiene regulations have been complied with.

7.17 We note that changes are proposed to the EU's rules governing meat and poultry inspections in slaughterhouses. The current rules are based on the principle of individual inspection and, where necessary, palpation and incision of lymph nodes, offal and carcass meat, supplemented where applicable by bacteriological, parasitological or chemical examination.

7.18 Important features of the proposed new arrangements include all red meat animals and poultry having to be accompanied to slaughter by "chain information" supplied by the farmer. This will be information relevant to food safety, such as previous *post-mortem* inspection findings in respect of animals from the same herd, flock or holding, and the status of the herd or flock in relation to a zoonosis which is subject to monitoring (eg. the *Salmonella* status of a pig herd). If this information is not available, the animals will be slaughtered but their meat will be excluded from the food chain.

7.19 Unnecessary *post-mortem* inspections for some conditions may not have to be carried out, where area or herd-based guarantees of freedom from disease can be provided. *Post-mortem* handling of carcasses and offal will be progressively minimised, following advice from the European Food Safety Authority on appropriate procedures for individual types of animal. *Ante* and *post-mortem* inspection findings of significance for public health (or animal health and welfare) will be required to be collected and communicated to public and animal health officials as appropriate, as well as to the farmer of origin of the stock and his/her veterinary surgeon.

7.20 Among the perceived advantages of the new proposals over the current meat inspection system are that, in extending official controls to the entire food chain, they allow controls to be made at the most effective points along the chain; in allowing only those animals with a known history to be slaughtered for human consumption, they will provide traceability and allow procedures to be put in place to manage identified risks (eg. slaughtering a group of animals last in the day, followed by a total clean down of the slaughterline so as to avoid cross-contamination); and in providing a risk basis for *post-mortem* procedures, they will reduce incision and palpation of otherwise normal but infected carcasses, and prevent resultant contamination and cross-contamination of meat.

Conclusions

7.21 *Campylobacter* spp., including those which cause human disease, are likely to be widespread in the environment, and it is not surprising that food producing animals such as cattle, sheep and pigs are exposed to this organism. In terms of risk management, it seems sensible to assume that all flocks and herds will contain animals which are likely to be colonised with *Campylobacter* and to take steps during the slaughter process to minimise the likelihood that these are transferred to the final products leaving the plant. The control measures required to achieve this aim will be essentially the same for *Campylobacter* as for organisms such as *Salmonella* and VTEC. We do not therefore consider that there is a need for *Campylobacter*-specific measures.

7.22 We recognise that both Government and industry have developed and put in place a number of measures to minimise the possibility of faecal material being transferred from the gut (or indeed the hide and fleece) during the slaughter process. We believe that, if properly applied, these will provide an effective barrier against *Campylobacter* contamination.

7.23 Cross-contamination is a potential risk and opportunities for cross-contamination should be avoided in relation to *Campylobacter*. We believe that the proposed new EU meat inspection requirements, by reducing the use of palpation and incision, will help reduce the risk of cross-contamination. We agree that improving the flow of information across the food chain is likely to assist traceability and facilitate application of disease control and food hygiene measures at the most effective points. We believe the number of red meat carcasses at risk from cross-contamination will be lower than for poultry.

7.24 We believe that the quality of the information trail would be further enhanced if *Campylobacter* flock prevalence data were available at slaughter. We address this is Chapter 4.

Recommendations

7.25 In view of the variations noted above in the prevalence of *Campylobacter* in retail meat samples, and in order to obtain a clearer picture of the risk if any to public health from such products, **we recommend that the Food Standards Agency should undertake UK wide, large-scale, structured surveillance of the prevalence of *Campylobacter* in red meat on retail sale**. **(Priority A)** We note that the Agency has recently requested pilot work in this area.

Chapter 8

Measures to prevent *Campylobacter* cross-contamination in domestic and catering environments

Risk factors for *Campylobacter* infection

8.1 In assessing the relative importance of domestic and catering practices in controlling *Campylobacter*, it is recognised that most incidents of infection with these bacteria do not form part of outbreaks (0.4% cases between 1995 and 1999 were outbreak-associated) and the causes of incidents are not clearly understood.[100] In the Study of Infectious Intestinal Disease (IID) conducted in England in 1995, one of only two factors identified as significant in elevating the risk of campylobacteriosis was the consumption of chicken at restaurants.[110,254] The other factor was travel abroad.

Modes of transmission and outbreak settings

8.2 Investigation of the 50 outbreaks of campylobacteriosis in England and Wales between 1995 and 1999 identified, as modes of transmission, 35 (70%) foodborne; 4 (8%) waterborne (non municipal supply); 1 (2%) animal contact (chicks); 1 (2%) person-to-person; and 9 (18%) unknown.[100] Outbreaks mainly occurred in commercial catering premises (32/50, 64%) including 16 in restaurants, 10 in hotels, 4 in public houses or bars and 1 in each of a hall and canteen. The majority of the remainder occurred in schools (12%) and the armed services (8%). Of the 35 foodborne outbreaks, poultry products (13 chicken and 1 duck) were the most commonly identified likely vehicles. The reasons identified as contributing to the outbreaks included cross-contamination (18 outbreaks), inadequate heat treatment (10 outbreaks), and inappropriate storage (7 outbreaks).

Raw poultry meat as source of *Campylobacter* infection

8.3 Although a variety of animals, environments and foods are recognised as potential sources of *Campylobacter* spp., the most significant known source is raw poultry (whole or portioned, fresh and frozen), and chicken, in particular. Other raw foods, such as red meat, are also known to be contaminated with *Campylobacter*, but neither the levels nor the incidence in retail products appear to compare with those found in raw poultry. Nevertheless, raw foods such as meat are, like poultry and will continue for the foreseeable future to be, sources of *Campylobacter* into domestic and catering premises. Such foods must therefore be recognised at all times as

presenting a risk if not adequately cooked, or if they come into contact with ready-to-eat foods.

8.4 Raw chicken is known to be contaminated with *Campylobacter* spp. at a high frequency, often in excess of 50%.[7,255] In addition, the levels of the organism reported on fresh chicken carcasses can exceed 100,000 colony forming units (cfu).[7]

Tackling *Campylobacter* in domestic and catering environments

Excluding Campylobacter *from domestic and catering environments*

8.5 Given the often low infectious dose of the organism, with as little as 500 cells having been reported to be capable of causing an infection, foods such as raw chicken entering a domestic or catering facility represent a significant cross-contamination and in turn, infection, risk.[1] With levels of over 100,000 cfu on some chicken carcasses, as little as 0.5% of the original contaminants need to be transferred to a ready-to-eat food to cause a potential infection, and it is almost inevitable that even minor lapses in food hygiene practices will result in cross-contamination. Any attempt to reduce *Campylobacter* infections must address the high levels entering the food supply chain and kitchen, as well as the practices that should be in place in domestic and kitchen settings to destroy or prevent contamination with the organism. Indeed, a quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* spp. in chicken estimated that in order to achieve a 30-fold reduction in human disease, kitchen hygiene would have to improve by approximately 30-fold, whereas a reduction in the number of the *Campylobacter* on chicken carcasses by 2-log cfu would achieve the same effect.[256]

Temperature abuse

8.6 *Campylobacter* spp. do not grow at temperatures below approximately 30^0C and are not believed to be especially heat resistant. We have previously reported the factors affecting the growth and survival of *Campylobacter* in foods.[1] As *Campylobacter* cannot grow at ambient and sub-ambient temperatures, the main risk in the domestic and catering kitchen will be associated with cross-contamination of raw foods to ready-to-eat foods, either directly or indirectly from hands and work surfaces/kitchen utensils, and undercooking of contaminated raw foods. **Notwithstanding the fact that *Campylobacter* cannot grow at temperatures below 30^0C, we stress the importance of preventing temperature abuse by keeping hot foods at elevated temperatures e.g. >63^0C, or cooling them rapidly to temperatures precluding the growth of many pathogenic microorganisms e.g. 5^0C or less**.

Effective cooking

8.7 One of the most important intervention measures employed to control the organism is effective cooking. *Campylobacter* is not heat resistant and is readily destroyed by pasteurisation temperatures applied to milk (71.7^{0}C, 15 seconds) and meat (70^{0}C, 2 minutes). Previous advice given to producers and caterers on the heat processing requirements to ensure safety of cooked meat (70^{0}C for 2 minutes or an equivalent temperature) still remain valid.[257] In the catering environment, it is essential that effective controls are put in place to guarantee that raw foods likely to be contaminated with *Campylobacter* are cooked to the correct temperature and time to destroy the organism. We believe that the proper and hygienic use of suitable meat thermometers in the catering and domestic settings would yield real benefits in terms of ensuring effective cooking. The benefits of meat thermometers should be more widely communicated. The Food Standards Agency may also, as part of its review of HACCP implementation in the catering sector, wish to consider whether documentary evidence of effective temperature control checks should be required to be kept.

8.8 As noted in Chapter 3, poultry liver consumption has been identified as a risk factor for human campylobacteriosis,[116] as has eating raw or rare chicken.[117-119] Consumers need to recognise that a current culinary trend of serving poultry liver 'pink' means that any *Campylobacter* present will not have been destroyed. We received anecdotal evidence of an even more worrying development, namely the addition of undercooked material and blood to poultry liver dishes to enhance the pinkness. This is clearly highly undesirable and dangerous from a food safety perspective.

Manufacturers' instructions

8.9 On-pack instructions are usually present on pre-packaged food to give guidance on the conditions necessary to ensure effective cooking. In our Report on Poultry Meat,[171] we recommended that 'the food industry should introduce more informative labelling, in relation to raw, flash-fried poultry products, in order to make clear to consumers that such products require thorough cooking'. Cooking instructions, although offering guidance only, must be generated using appropriate, calibrated equipment and under controlled conditions, in order to be as precise as possible. Best practice for generation of such advice is available through some research associations, but it is not published or widely available. This needs to be remedied. Industry guidance produced through trade associations would help ensure a consistent approach to the generation of on-pack cooking instructions. Such instructions need to be displayed prominently on the packaging. In addition, it is not common practice for foods purchased over the counter (butchers, meat counters, etc) to be labelled with cooking instructions. Thought needs to be given to how appropriate cooking guidance can be provided for such products, and industry should examine the feasibility of providing cooking guidance on all raw meat and poultry products, including those sold from service counters, butchers and similar outlets.

Cross-contamination

8.10 Once a food is cooked, every effort must be made to prevent it becoming re-contaminated with *Campylobacter*. A variety of sources offer opportunities for post-process contamination in the kitchen or catering premises, including people, pets, raw foods, and the environment.

8.11 *Campylobacter* has been found to be readily spread in the kitchen during preparation of raw foods such as chicken, and studies examining consumer behaviour in the kitchen have shown that practices likely to lead to cross-contamination of *Campylobacter* from raw foods, especially chicken, to ready-to-eat foods are common.[258-260] One study involving the observation of 108 consumers from all socio-economic backgrounds making prescribed meals found 58% occurrence of the handler not washing their hands after handling raw meat/poultry.[258] In the same study, one-third of consumers washed raw chicken, and 15% failed to cook foods to a temperature of at least 74^0C. A questionnaire/interview-based study of 1,030 consumers assessing practices in relation to the handling of raw meat identified that the majority routinely washed raw meat, with whole chicken being the highest (80%).[259]

8.12 Research has shown that *Campylobacter* can be spread significant distances in the kitchen and improvements in isolation techniques have demonstrated that the bacteria can also survive for long periods of time.[14] The exact risk that this presents clearly depends on the levels of the organism and the likelihood of it contaminating a ready-to-eat food but, nevertheless, any practice which spreads these organisms within the kitchen should be minimised. Washing raw meat and poultry is likely to spread *Campylobacter* in the kitchen through splashes, droplets and aerosols and should be actively discouraged. *Campylobacter* may also be found on the outside of chicken packaging, especially if leakage of blood has occurred, and the industry is encouraged to continue efforts to reduce leakage from pre-packaged raw poultry products through more effective packaging and seal integrity.

8.13 In a study of the cross-contamination potential of *Campylobacter* during the preparation of Sunday lunch made from raw chicken, 25 participants were allowed to prepare a meal in their own kitchens. Of the 11 where *Campylobacter* was isolated from the raw chicken, the organism was recovered from hands (3), oven handles (2), counter tops (3) and the draining board (4) following preparation of the chicken.[261]

8.14 We note with interest the UK national survey of in-use kitchen cloths which failed to detect any *Campylobacter* in 1,009 cloths taken from homes in 2001.[262] The survey did, however, find other organisms, including *Salmonella* spp. (1 sample), *Escherichia coli* (367 samples) and *Listeria monocytogenes* (14 samples), indicating the potential for cloths to be

colonised with microorganisms and become a vehicle for cross-contamination.

8.15 Levels of contamination with *Campylobacter* can be effectively reduced in the domestic kitchen by adherence to a prescribed cleaning regime using detergent, hot water and disinfectant. Some research has shown that using the former two alone is less effective on surface contamination.[263] It is also clear that effective hand washing makes an important contribution to improving hygiene. A recent review determined that washing hands with soap could be expected to decrease the risk of diahhroeal disease in the community by almost half.[264]

Hygiene advice

8.16 We note that, despite the existence of an industry code of practice,[265] on-pack hygiene advice (with the exception of cooking instructions) is infrequently provided on food products such as raw meat and poultry, which may be contaminated with enteric pathogens. Some retailers provide food safety advice on such products, but this does not appear to be common practice. More needs to be done, and all producers and retailers of food where pathogens such as *Campylobacter* may be present should provide advice on key safety steps. This should be applied as a minimum to raw poultry products. Advice should include measures for effective cooking, and avoidance of cross-contamination. Washing raw poultry should be discouraged.

8.17 Barbecued foods have frequently been highlighted as a potential risk factor for campylobacteriosis, although neither the Study of IID in England, nor the *Campylobacter* Sentinel Surveillance Scheme in England and Wales, found evidence to support this practice being significantly associated with infection.[110,266] Nevertheless, we believe that barbecuing raw meats, particularly poultry, represents an important potential risk, from cross-contamination when handling contaminated raw foods, and from undercooking due to the use of poorly controlled or unfamiliar heating sources/methods. The FSA has, in the past, provided targeted barbecue hygiene and cooking advice to consumers, and this should be repeated each summer. In addition, industry should provide food safety and hygiene advice on the packaging for barbecues and raw meats intended for barbecuing.

Companion animals

8.18 Risks in the domestic kitchen are also presented by companion animals which can carry *Campylobacter* spp. In one survey, the organism was isolated from 32% of faecal samples from dogs suffering diarrhoea.[267] Moreover, surveys of consumer practices have shown that it is not uncommon for pets to remain in the kitchen during the preparation of a meal.[259,260] The extent to which these animals also present a risk of

Campylobacter infection to their owners due to factors other than foodborne transmission e.g. stroking dogs, dogs licking people's faces, etc., are not clear. An association was found between *Campylobacter* infection in dogs, and diarrhoea in human households, although this was not considered statistically significant.[267] Indications from the *Campylobacter* Sentinel Surveillance Scheme do however reveal an elevated risk of infection (27 cases per 100,000) associated with dog ownership, in comparison to other pet ownership (0.7 cases per 100,000).[266] The FSA should consider how best to communicate the potential risks associated with the carriage of *Campylobacter* in companion animals and the hygienic precautions applicable to them.

Food handlers

8.19 Any attempt to control *Campylobacter* in domestic and catering premises cannot overlook the importance of education and training of food handlers in the basic principles of food hygiene and safety. We are aware of the difficulties associated with ensuring adequacy of such knowledge in a large and disparate population both in the home, and in catering where a large number of the businesses employ few people (<10) and have a high staff turnover. Catering businesses have a legislative obligation to provide safe food using a hazard analysis approach. Under revised European Union hygiene legislation, which is likely to apply from 1 January 2006, they will be required to put in place food safety management systems based on HACCP principles. Infected food handlers and ancillary staff working in food handling areas are an important route of transmission of foodborne infections. We addressed this subject in some depth in Chapter 5 of our Report of Foodborne Viral Infections[268] and reiterate the importance of the advice given therein about pre-employment health assessment, good hygiene practice, and excluding from food handling areas staff who are ill.

8.20 We are encouraged by the continued efforts of the FSA to build food hygiene into its own promotional campaigns and, in collaboration with other Government Departments, into education syllabuses. However, we believe that further measures are required in order to embed food hygiene and safety principles into the education of primary and secondary school pupils.

8.21 We note the increased activity the Food Standards Agency has undertaken in recent months to highlight the need for adoption of effective hygienic precautions in catering businesses, as part of its Food Hygiene Campaign. We are aware of the intention to extend this to consumers and we endorse this approach. However, we feel that more needs to be done to draw attention to the enhanced risks associated with raw poultry. As basic precautions may be insufficient to prevent *Campylobacter* cross-contamination from highly contaminated foods like raw chicken, the FSA should consider how best to highlight to caterers and consumers the heightened risks associated with foods such as raw poultry.

Conclusions

8.22 Raw poultry meat, particularly chicken, is, and will continue for the foreseeable future to be, a significant vehicle by which *Campylobacter* is introduced into the domestic and catering environments. Levels of *Campylobacter* contamination of $>10^5$ cfu are seen on some chicken carcasses. The human infectious dose is reported to be as low as 500 cells. Given the likely difficulties involved in controlling high levels of contamination in the kitchen, especially from sources such as raw poultry, we reiterate the critical significance of reducing the levels and incidence of *Campylobacter* on such products to lessen the burden on domestic and catering premises in dealing with such hazards.

8.23 If *Campylobacter* can be effectively tackled at the primary production stage, then the anticipated reduction in the number of *Campylobacter* cells reaching the kitchen would enhance the effectiveness of normal hygiene measures taken there. We make a number of recommendations below designed to enhance the effectiveness of action in the domestic and catering environments.

Recommendations

8.24 We strongly recommend the proper use in catering of meat thermometers, as a means of ensuring the effective cooking of raw poultry products in particular. The use of such devices in the home may also yield benefits and we recommend that the Food Standards Agency (FSA) considers communicating the benefits of the use of cooking thermometers for domestic and catering settings. We also recommend that, as part of its review of HACCP implementation in the catering sector, the FSA considers whether documentary evidence of effective temperature checks should be required to be kept. (Priority A)

8.25 We recommend that industry guidance is produced through trade associations, to ensure a consistent approach to the generation of on-pack cooking instructions. In addition, where guidance is provided, this should feature prominently on the packaging. (Priority A)

8.26 In the case of meat which is not pre-packed, we recommend that the industry examines the feasibility of providing cooking guidance on all raw meat and poultry products, including those sold from service counters, butchers and other similar outlets. (Priority A)

8.27 We believe that the practice of washing raw meat and poultry is likely to lead to increased risk of spread of *Campylobacter* in the kitchen through splashes, droplets and aerosols, given the high levels which may be present on raw chicken. We recommend that this practice be actively discouraged by the FSA and industry. If necessary, consumers should be advised only to wipe down a chicken with a disposable paper towel. (Priority A)

8.28 Whilst it is understood that the consumer does not always read such advice, we recommend, in the public interest, that all producers and retailers of foods, where enteric pathogens such as *Campylobacter* may be present, should provide advice on the key food safety steps which should be taken to prevent infection. This should, as a minimum, be applied to all raw poultry products, as the levels of the organism are known to be high. Advice should include measures for effective cooking and for the avoidance of cross-contamination from the raw food to ready-to-eat food (through separation of foods and utensils, and through hand washing). (Priority A)

8.29 We are aware of previous activity by the FSA to provide targeted advice to consumers regarding improved cooking/hygiene practices when barbecuing and we recommend that this approach be repeated prior to each summer period. (Priority A)

8.30 We recommend that the industry provides food safety/hygiene advice on the packaging of foods for barbecues, particularly raw meats, and especially poultry. (Priority A)

8.31 We recommend that attention is drawn to the potential risks associated with carriage of *Campylobacter* in domestic pets, and to the hygiene precautions applicable to them. (Priority A)

8.32 We recommend that further measures are taken to embed food hygiene and safety principles into the education of primary and secondary school children. (Priority C)

8.33 In light of the fact that basic precautions may not be sufficient to prevent *Campylobacter* cross-contamination from highly contaminated foods such as raw chicken, we recommend that the FSA considers what measures can be taken to highlight to caterers and consumers the heightened risks associated with certain foods such as raw poultry. (Priority A)

Chapter 9

Campylobacter detection and typing

Introduction

9.1 Since the ACMSF last considered *Campylobacter* detection and typing, a number of developments has occurred.

9.2 In 1997, a national *Campylobacter* Reference Unit was established by the Public Health Laboratory Service (PHLS), and the reference service was piloted in Wales and the North West of England.[93] A routine reference service was subsequently rolled out across England and Wales on a sentinel basis in 2000. Through the *Campylobacter* Sentinel Surveillance Scheme (CSSS), standardised epidemiological and microbiological reference typing data have been captured for about 15% of all laboratory-confirmed *Campylobacter* infections in England and Wales.[74]

9.3 There are many typing methods available, and these have served to emphasise the complexity of the epidemiology of *Campylobacter* infection in humans and food animals. A European Study to attempt to standardise and harmonise molecular sub-typing techniques for *Campylobacter jejuni* (CAMPYNET) has been undertaken (www.svs.dk/campynet). Despite numerous developments in typing methods, evidence to support their value in informing the epidemiology of *Campylobacter* infection is hard to find. Thus there has been no clear success in developing interventions and the proportion of foodborne infections has not been established.

9.4 The publication of the Chief Medical Officer's strategy for health protection in England signalled the dissolution of the PHLS and the creation of a Health Protection Agency (HPA).[269] This, along with other developments in the National Health Service (NHS), has had implications for the future delivery of clinical and food, water and environmental laboratory services, including reference services.

Campylobacter detection

9.5 Various methods for the isolation of thermophilic *Campylobacter* from clinical specimens are published, and the bacteria can be isolated from human diarrhoeal faecal samples using any of the four microaerobic-atmosphere-generating systems available.[270] What is not known is the extent to which protocols for screening clinical specimens are standardised across clinical laboratories in the United Kingdom, except for the former PHLS

laboratories, where standard operating procedures for the handling of food, water and clinical specimens were employed. The development of the HPA affords the opportunity to standardise testing protocols across the NHS. There is also a need to determine the extent of technique bias with regard to the strains and species of *Campylobacter* isolated.

9.6 Most clinical laboratories do not perform speciation. For example, in 2001, only 9.4% of *Campylobacter* reported to the Communicable Disease Surveillance Centre (CDSC) had been identified to the species level. This has fallen from 17% at the time of our Interim Report.[1] Results from the CSSS show that there might be important differences in the epidemiology of *C. coli* and *C. jejuni*, so speciation is valuable.[74]

9.7 Isolating *Campylobacter* from food specimens usually requires an enrichment step, although the choice of enrichment broth can significantly affect recovery of organisms.[271] As with clinical specimens, it is also likely that the choice of enrichment media in particular will influence the population structure of strains isolated from food and environmental specimens. This will occur because *Campylobacter* strains differ in sensitivity to the antibiotics in selective media[272] and are likely to grow at different rates. Given the continued debate about the importance of various food animal species as sources of human infection, it is important that there is as much standardisation as possible in the isolation methodologies used with foods, food animals and human cases. *Campylobacter* spp. do not always produce what are regarded as 'typical' colonies on selective agars, and there are potential problems in recognising these pathogens. This may reduce isolation rates. In addition, the method of sampling the food can influence the numbers of *Campylobacter* recovered.[123] This latter point has implications for microbiological risk assessment where enumeration of organisms, as well as detecting their presence or absence, is important.

9.8 Success with molecular method development affords the opportunity to detect *C. jejuni* in food samples much more quickly than is possible using traditional methods,[273,274] bearing in mind that molecular methods may identify non-viable, as well as viable, organisms.

9.9 Finally, it appears that no single method will lead to isolation of all strains from clinical or non-clinical samples. It is, therefore, important that a decision is made at the outset of any investigation about the most appropriate method to be used. The isolation of *Campylobacter*, particularly, from non-clinical samples, has suffered from the same developmental issues that have bedevilled typing. There are a lot of methods available but most have not been rigorously tested in multi-laboratory trials and there are marked variations in efficacy.[275] As with any pathogen in a mixed population, the isolation of *Campylobacter*, requires a proper balance between suppressing competing flora while encouraging the growth of the target

pathogen. This can be particularly difficult with *Campylobacter* as it is easily damaged by exposure to the extra-intestinal environment. This will lead to sensitivity to selective agents, which may affect viability in selective media.[276,277] It would seem that the strategies adopted for *Salmonella*, where foods are inoculated in non-selective media, will not always be successful for *Campylobacter* because of over-growth by competing bacteria.[278] There is a need for a properly structured study of isolation media sensitivity and selectivity.

Campylobacter typing

9.10 Scientific debate about the utility of typing methods for *Campylobacter* spp. continues unabated. There would seem to be general agreement that *Campylobacter* typing in the outbreak situation is an important tool for helping to unravel epidemiology. However, information derived from the use of phenotypic and/or genotypic typing methods in outbreaks gives two types of picture:

- outbreak cases linked epidemiologically, patients all infected with the same strain; and
- outbreak cases linked epidemiologically, but patients infected with different strains.[100]

9.11 A typing method is any technique which can distinguish between epidemiologically unrelated strains. There is no assumption that different laboratories using the same method on the same strains would necessarily get the same results. Many of the genotypic methods developed for *Campylobacter* are fingerprinting methods ie. they produce patterns. These can be compared with patterns from other strains for similarities. A typing method should provide a type designation or label to these patterns. This has been a major challenge for PulseNet in the United States where a key development has been the creation of a standardised nomenclature system for pulsed field gel electrophoresis (PFGE) patterns.[279] Typing methods are useful in investigating certain problems such as a localised outbreak investigations, but are not necessarily useful for larger epidemiological studies.[280]

9.12 A typing scheme is one used for discrimination between epidemiologically unrelated isolates belonging to the same microbial species. It should be capable of identifying strains accurately (type designations) and reproducibly at different times and in different laboratories.

9.13 Three main characteristics that need to be considered when evaluating a typing method/scheme are typeability, reproducibility and discriminatory power. Cost, ease of use, and turnaround time are also important considerations.[280]

9.14 Typeability is the proportion of isolates that can be typed using the method in question.

9.15 Reproducibility includes three concepts:

- *in vitro* reproducibility (the proportion of strains which are typed with the same result on repeat examination);
- *in vivo* reproducibility (requiring repeat testing of multiple strains over time to assess the stability of the organism under study, and hence its type); and
- reproducibility between centres (the extent to which identical methods used in different centres produce identical results).[280]

9.16 Discriminatory power is defined as the probability that two strains, chosen at random from the population or unrelated strains, will be distinguished by the typing method used.[280]

9.17 Tables 9.1 and 9.2, at the end of this Chapter, describe the features of the main phenotypic and genotypic methods employed for typing using the six parameters described in paragraph 9.13.

9.18 Probably the only widely accepted phenotypic typing scheme is serotyping using the Penner scheme.[281] Indeed, this was considered the only practical method for surveillance on a broad scale.[282,283] Despite the effort that has gone into typing on a broad scale, typing studies to below species level have yet to add significantly to our understanding of the epidemiology of *Campylobacter* infections. Relatively high levels of non-typeability, especially when applied to poultry or environmental isolates,[284] coupled with reproducibility problems[285] has led researchers on a quest to find improved methods for *Campylobacter* typing.

9.19 Where typing methods have been used in targeted, hypothesis-driven studies, there has been greater achievement. Random amplification of polymorphic DNA (RAPD), PFGE and flagellin gene restriction fragment length polymorphism (*fla* typing) have been used with success in tracing organisms across the food chain.[130,179,286-290] However, multilocus sequence typing (MLST), which has also been used in this way, offers the advantage that direct comparison between laboratories can be made much more easily than with some of the other methods.[290-292] The scientific consensus that seems to be emerging is that MLST is probably the most promising of the genotyping methods to date.

9.20 Many typing methods have been developed using, and/or applied to, small and/or eccentric collections of strains. The national *Campylobacter* Reference Unit at Colindale has amassed a large, representative set of clinical isolates with accompanying standardised epidemiological surveillance data collected through the CSSS. Analyses of the dataset are not yet complete but, early on, the benefits of speciation and antimicrobial resistance testing were demonstrated.[74,293] In judging the importance of *C. coli* as a foodborne pathogen, it is worth reflecting that, in 2000, *C. coli* was estimated to account for over 25,000 cases of illness, and cost patients and the NHS nearly £4 million.[294] Tackling even the smaller portion of *Campylobacter* infection in England and Wales is likely to have important public health benefits.

9.21 If the objective of typing is to unravel the epidemiology of *Campylobacter* infection, and hence inform control measures, using the CSSS strain collection affords the opportunity to determine the utility of methods like MLST in a public health setting where good epidemiological data are also available.

Lessons from typing studies

9.22 A summary of the features of the main methods for typing *Campylobacter* is given in Table 9.3, also at the end of this Chapter. There is not yet a universally accepted solution to the question of *Campylobacter* typing. First, no matter what method is employed, some well-defined clonal lines can be identified readily, although these are in the minority, and there is a wide range of variation within the remainder, which comprise the majority. Second, although there has been much work on method development, each new method spawns a series of slight adaptations, so-called "creeping featurism". This makes direct comparison between slightly different methods very difficult, and comparisons between laboratories even harder. Third, although there has been much activity in the research setting, there is less evidence of the application of these methods in a public health service setting so that, despite the large investment in typing methods over recent years, there have been few tangible epidemiological or public health benefits. However, where typing methods have been used in focused studies, e.g. for local outbreak investigation or for targeted studies across the food chain, there has been greater success.

DNA microarrays

9.23 Although a wide variety of typing approaches has been developed for *Campylobacter*, the availability of whole genome sequence data offers the prospect of another potentially valuable approach. It is established that DNA microarrays based on the complete set of sequenced genes offer a unique opportunity to investigate and compare genome composition for individual isolates of a species. This approach has been applied to

Campylobacter[295,296] and its further development and validation may provide a more complete genome-based data set and a novel typing approach of practical value for the future.

Conclusions

Detection

9.24 It is likely that, for the foreseeable future, traditional culture techniques will be employed for the examination of clinical and non-clinical samples for *Campylobacter* spp. More rapid methods are now available but there is a need to ensure that they have sufficient sensitivity. Any method will suffer from inherent bias and this will continue to be a problem in studies on epidemiology and in comparison of different surveillance schemes. The isolation of *Campylobacter* would seem to be more difficult than for *Salmonella*, for example, and there is a need for more rigour in method choice and for a properly structured, multi-laboratory study of the most commonly used methods. This is an area our *Campylobacter* Working Group will need to consider in reviewing research needs relating to *Campylobacter* (see Chapter 1).

Typing

9.25 Speciation of *Campylobacter* has proved useful in differentiating epidemiologically between *C. coli* and *C. jejuni*.

9.26 *Campylobacter* typing should be driven by objectives and/or specific hypotheses. These might be:

- tracing sources and routes of transmission of human infection;
- identifying and monitoring, both temporally and geographically, strains with important phenotypic or genotypic characteristics;
- developing strategies to control organisms within the food chain;
- monitoring trends in antimicrobial resistance; and
- outbreak identification.

The method(s) chosen should then be dictated by specific objectives and/or hypotheses. In addition to this, appropriate sampling frames should be used.

9.27 Typing has confirmed the complexity of the epidemiology of *Campylobacter* infection but, on a broad scale, has not yielded the expected public health benefits in terms of identifying a big target amenable to control.

Routine typing is probably not useful for source tracing and global epidemiology because of the carriage of multiple strains in animals, and the extreme diversity of those strains.[290] Completing the analyses of the CSSS is, however, needed since the requirement for national strain-specific epidemiological studies might yet be demonstrated.[93]

9.28 Where the objective is to make an assessment of *Campylobacter* across the food chain in relation to human infection, veterinary, food and clinical laboratories should use the same methods. One of the problems with comparative epidemiology is that different techniques have been applied to different specimen types. It is important that, when carrying out research and surveillance in animals, isolation and typing methods should take the lead from, and be consistent with, methodologies used for clinical isolates unless there are specific reasons not to do so. This recognises the fact that *Campylobacter* is primarily of public health significance.

9.29 The technological revolution in clinical medicine means that, in future, direct detection and typing using clinical samples will be possible.[297] An obvious benefit of this approach is the speed of diagnosis and the potential for real time epidemiology. DNA sequence-based methods like MLST therefore have the greatest potential to be "future-proof".

9.30 The variability and genetic instability of *Campylobacter* cautions us against believing that there exists some magical solution to the typing of all campylobacters of human health significance. However, any improvement in the tools available for differentiating the origins or food sources of *Campylobacter* spp. would greatly assist the Food Standards Agency in tackling human campylobacteriosis. Examination of the history of the many typing methods summarised in Tables 9.1 and 9.2 prompted much discussion within the *Campylobacter* Working Group as to whether the ACMSF could make a firm recommendation in this area. We have now concluded that the DNA sequence-based MLST method offers an opportunity in the short to medium-term to improve our knowledge of what is a very complex epidemiological story.

9.31 The availability of whole genome sequence data offers the prospect of another potentially valuable approach to typing. DNA microarrays based on the complete set of sequenced genes offer a unique opportunity to investigate and compare genome composition for individual isolates of a species. This approach has been applied to *Campylobacter* and its further development and validation may provide a novel typing approach of practical value in the future.

Recommendation

9.32 As with all typing methods for food poisoning pathogens, different laboratories will take different approaches, and agreement will be difficult to reach. **We therefore believe that the FSA needs urgently to take a firm initiative in bringing together laboratories capable of applying MLST so that investigative programmes can be designed to improve our epidemiological understanding in the next few years. We so recommend**. **(Priority A)**

9.33 We want to be very clear that this is in no way a recommendation that, at this stage, the FSA should be funding large research programmes on typing methods. Rather, it is a recommendation that advantage is taken of the opportunity to get the most out of MLST in the shorter-term.

Table 9.1: Summary of the features of the main phenotypic methods for typing *Campylobacter*

Method	Typeability	Reproducibility	Discriminatory Power	Cost	Ease of use	Turnaround time	Comments
Serotyping				Low, although set-up costs are high	Easily applied by both clinical and reference laboratories, provided that antisera are available.[282]	Less than 24 hours[306]	Main disadvantage of serotyping methods is the lack of commercially available, high quality antisera.[282] It has been said that subculturing, storage and freezing may affect stability of phenotyping[306] although this experience is not universal.[305] Serotyping generally produces around 80% typeability for poultry isolates[308,309] although at least one author suggests that up to 40% of poultry isolates are untypeable using the scheme described by Frost *et al*.[284]
• Penner[298]	70-90%[282,304]	Good[306]	0.898[307]				
• Lior[299]	70-90%[282,304]	Good[306]					
• Frost[300]	80%[305]	Not available	10 serotypes accounted for 53% of *C. jejuni* isolates tested; 3 serotypes accounted for 69% of C. coli isolates tested.[305]				
Biotyping				Low	Easy and available to most laboratories.	24-48 hours[281,303]	Produces only a few markers among strains when used alone. Needs to be used in conjunction with another method.[281,282,307]
• Lior[301]	100%[282]						
• Preston biotyping[302]	100%		0.945[307]				
• Resisto-typing[281, 303]	100%[303]	>98%;[303] occasionally problematic[281]	Good – no single resistotype accounted for more than 25% of isolates[303]				
Phage typing						24 hours	Typeability improves when serotyping and phage-typing are used in combination (around 97%)[313]
• Grajewski[310]	88-94%;[308] 82%[282]	94%[308]	46% of isolates represented by the four most common phage patterns[308]				Repeatability and reproducibility depend on individual interpretation of lysis reactions so that a standard procedure for recording lysis reactions is needed, and a standard taxonomy of types is needed.[313,314]
• Salama[311]	94%[313]	Good	0.908[307]				
• Khakhria[312]	81% overall	Good	Nine phage types represented 57% of strains				

Table 9.2: Summary of the features of the main genotypic methods for typing *Campylobacter*

Method	Typeability	Reproducibility	Discriminatory Power	Cost	Ease of use	Turnaround time	Comments
Flagellin gene restriction fragment length polymorphism *(fla typing)*	100%[306,315-317]	Good[317]	Fair;[306] Better than ribotyping but not as good as PFGE[322]	Low[306]	Relatively quick and simple[322] Equipment becoming widely available	<24 hours	Method samples a small proportion of the genome. Procedures (especially primers and restriction enzymes used) need to be standardised otherwise inter-laboratory comparisons are impossible. Vulnerable to genetic instability.[284]
Pulsed field gel electro-phoresis (PFGE)	100%;[306] 95%[318]	Good[321]	Good;[306] Better than ribotyping and phage-typing[321]	Needs specialised and expensive equipment[324]	Preparation process for the DNA samples is lengthy, labour-intensive, not amenable for use with large numbers of samples	3 to 5 days generally although shorter protocols have been published[325, 326]	This is the method of choice for PulseNet in the US but application of a standard method is strictly adhered to and enforced.[326] Conditions used in different studies vary widely (especially restriction enzymes and electrophoretic conditions), interpretation of results is difficult since genetic instability, even during *in vitro* culture, can lead to minor or major changes in profile.[284,327]
Ribotyping	100%;[282] >89%[319]	Good[306]	Poor[323]	Expensive	Low throughput, complicated technique		Choice of restriction endonuclease is of critical importance[323] and variations in the restriction enzymes and probes used make inter-laboratory comparisons difficult.[306]
Automated ribotyping	100%[320]	Good[306]	0.97[284]	High cost (both equipment and consumables)		Within the working day	Automation enhances reproducibility and enables inter-laboratory comparisons.[284]

Table 9.2 (continued): Summary of the features of the main genotypic methods for typing *Campylobacter*

Method	Typeability	Reproducibility	Discriminatory Power	Cost	Ease of use	Turnaround time	Comments
Random amplification of polymorphic DNA (RAPD)	87%;[328] 100%[286, 287]	Poor[329]	0.999 using computer-based analysis[287]	Low[306]	Quicker and cheaper than PFGE	<24 hours	Unlike other PCR–based identification and typing methods, does not require prior knowledge of the target DNA sequence. Less sensitive to, but is affected by, genetic instability.[284]
Amplified fragment length polymorphism (AFLP)	100%[306]	94.2%;[330] 98%[331]	Better than PFGE[332,333]	Average[306]	Interpretation of AFLP is complex.[332] Interpretation of single-enzyme AFLP is less so.[333]	2-3 days[306]	A random portion of the whole genome is sampled and AFLP is not dependent on prior sequence knowledge.[284] Not susceptible to genetic instability.[306]
DNA sequencing e.g. Multilocus sequence typing (MLST)	100%	High[291]	High[291]	$37 per isolate[334]	Automatable, high throughput possible.[334]		Not vulnerable to genetic instability.[334] Direct comparison between laboratories possible.[291,292]

Table 9.3: Summary of the features of the main methods of typing *Campylobacter*

	Typeability	Reproducibility	Discriminatory Power	Cost	Ease of use	Turnaround time
Serotyping						
Biotyping						
Phage typing				?	?	
***fla* typing**						
PFGE						
Ribotyping						?
Automated ribotyping					?	
RAPD						
AFLP						
MLST						?

Key	Good		Intermediate		Poor		Not reported	?

Chapter 10

Summary of conclusions and recommendations

Introduction

10.1 For ease of reference, this Chapter summarises the conclusions we have reached throughout this Report and the recommendations we have made.

10.2 As noted in the Summary to this Report, we have endeavoured to prioritise our recommendations. The summary of recommendations which follows is thus listed as Priority A (where action is required in the short-term to assist the Food Standards Agency (FSA) in developing and implementing its *Campylobacter* strategy); or Priority B (where the Committee feels that work should start in the next year or so); or Priority C (where we consider that work can be put in hand as and when possible, and in the light of competing priorities).

Chapter 1: Background

10.3 Having published an Interim Report on *Campylobacter* in 1993, the ACMSF decided in 2000 to revisit the subject with a view to identifying means of reducing the incidence of *Campylobacter* infection in humans. This decision reflected the fact that *Campylobacter* is the major cause of infectious intestinal disease in the UK and was taken against the background of the FSA's targets for reducing the incidence of foodborne disease and *Campylobacter* contamination of retail chicken.

10.4 As a first step, we held a workshop in February 2002 to take stock of research findings and to identify major knowledge gaps justifying on-going research. The workshop also aimed to help us decide whether there were food chain interventions which would reduce consumer exposure to *Campylobacter* and which would assist the FSA in its efforts to reduce the burden of foodborne disease. We were seized of the need to make our advice available to the FSA as soon as possible, given the fact that its foodborne disease target was time-bound. We therefore fed our advice into the Agency in tranches as soon as it was ready. We also resisted taking an in-depth look at research opportunities and needs where there were significant knowledge gaps.

10.5 Despite the scientific advances made, *Campylobacter* remains a poorly characterised microorganism and this impacts on its epidemiology and control. There is therefore a need for continued fundamental research, especially in the area of functional genomics. While the focus of this Report has been on the practical measures which will help the FSA develop its strategy for tackling *Campylobacter* in the shorter-term, the ACMSF

Campylobacter Working Group will meet again with the aim of identifying where research outputs, had they been available, would have contributed to progressing the objectives identified as desirable in this Report more quickly. It should be recognised, however, that any research requirements identified through this planned review can only yield results in the medium to longer-term, given the time lag involved between identifying research and being able to apply practical outputs.

Chapter 2: The organism, human immune response, and pathogenesis

10.6 The debate about the role, indeed the very existence, of the VNC form of *Campylobacter* seems unlikely to be resolved in the short-term. It is a complex area and not one where we have been able to draw any firm conclusions. However, we are not aware of any evidence to suggest that current uncertainties give cause for concern in relation to food safety. We are not, therefore, recommending that the FSA should commit funds to further research on the VNC issue. We note that the research community continues to carry out work in this area. This should be monitored and we hope that a consensus view will eventually emerge.

10.7 *Campylobacter* isolation methods have been improved since much of the work on VNC was performed, and it is now possible to recover cells previously thought to be non-culturable. What is not yet clear is whether very highly damaged cells of *Campylobacter* now recoverable from a variety of environments, and after a variety of treatments, pose an infection threat.

10.8 It is clear that infectious intestinal disease causes a considerable burden of ill health over and above the initial event. However, little information is available on the incidence and economic cost of long term *sequelae* and it would be useful to have a more reliable measure.

10.9 We recommend that the Government should instigate a primary care-based sentinel surveillance system, aimed at measuring directly the incidence and economic cost of long-term *sequelae* among cases of *Campylobacter* infectious intestinal disease. (Priority B)

10.10 We recommend that serological markers for recent infection and prior immunity be developed and tested through structured, epidemiologically robust, population-based studies. This should assist with estimating the prevalence of asymptomatic infection in the population (and hence estimating more accurately the magnitude of *Campylobacter*-associated *sequelae*). (Priority C)

Chapter 3: *Campylobacter* epidemiology

10.11 *Campylobacter* infection is a major public health problem. The epidemiology is complex. There are extensive animal and environmental

reservoirs and multiple risk factors for infection. Although epidemiological patterns, such as marked seasonality, are well described, their underlying explanations are still elusive despite much study.

10.12 Poultry appears to be an important source of infection. It is noteworthy that eating food, including poultry, on commercial catering premises has been identified as a risk in several case-control studies.

10.13 In the case of poultry, some progress has been made in reducing the role of the food chain as a vehicle for *Campylobacter* infection. However, in addition to the contribution of poultry to human *Campylobacter* infection, many studies also point to numerous other sources and vehicles of *Campylobacter* infection. It is important that these are not overlooked.

10.14 The contribution of foodborne transmission (as opposed to other transmission modes) to the human toll of *Campylobacter* needs to be better defined and we note that the FSA has already funded a research project designed so to do. **We support this course of action. (Priority A)**

10.15 We recommend that population studies to investigate the seasonality of *Campylobacter* infection be undertaken. An approach combining epidemiological, microbiological, environmental and veterinary expertise is likely to be needed. (Priority A)

10.16 We recommend that population studies to investigate cultural/behavioural risk factors for *Campylobacter* be undertaken. (Priority B)

10.17 We recommend that more extensive data are gathered on the levels of *Campylobacter* spp. in specific foods (eg. water, dairy products, vegetables, poultry and red meat) as well as in food-producing animals and companion animals. These are all potential sources of exposure for humans. We recommend that consideration be given to on-going surveillance as well as to "snap-shots" which tend to be the norm. It is very important that the microbiological methods employed allow meaningful comparisons to be made across the food chain (see Chapter 9). (Priority A)

Chapter 4: Measures to prevent *Campylobacter* contamination of chicken meat

10.18 It is becoming clear that control of *Campylobacter* on-farm is now a practical proposition, at least with birds that are housed. We brought this view to the attention of the FSA in September 2002 to assist the Agency in developing its *Campylobacter* strategy. The first commitment must be to rigorous biosecurity, combined with high standards of stockmanship and attention to good flock health and stress control. This will involve such

measures as restricting farm visits to essential personnel; ensuring visits are undertaken as hygienically as possible; and appropriate staff training on flock infection. The control of *Campylobacter* on-farm presents a greater challenge than that associated with the control of *Salmonella*.

10.19 Our *Campylobacter* Working Group received different views, both formally and anecdotally, about the possibility of the UK poultry industry adopting 'Scandinavian style' systems of on-farm biosecurity. Where these systems have been trialed in the UK, they have been seen to be successful, although industry argues that long-term maintenance would be difficult. While Scandinavian and UK systems of production and control measures do differ in some respects, reflecting the different sizes of the industries and the very different climates, we firmly believe that the application of biosecurity, such as changing footwear, and other hygiene measures, will either delay or prevent the entry of *Campylobacter* into broiler flocks and thus reduce the incidence of colonised birds. Studies in the Netherlands support this view. Changing of footwear was found to be important in a UK context, and another UK study found that frequent replenishment of boot dip disinfectant was one of a few factors which reduced broiler flock infection by over 50%. In the future, given current research effort, it may be possible to supplement biosecurity with pre- or probiotic approaches, competitive exclusion, and/or vaccination.

10.20 In addition, it is clear that a well-run broiler farm can reduce the incidence of *Campylobacter* through adherence to a number of key principles. It should:

- be species mono-specific (ie. farm only chickens);
- supply the birds with water of potable quality;
- properly clean and disinfect houses after flock removal, which should include disinfection of the water supply system;
- protect the house from entry by wild birds and rodents;
- supply feed which has received treatment sufficient to have eradicated *Salmonella* (and, hence *Campylobacter*), and protect it from re-contamination;
- only carry out thinning if done in association with proper crate washing (so that crates are not contaminated with *Campylobacter* spp. or other pathogenic microorganisms) and proper biosecurity measures covering eg. clothing and footwear;

- ensure that transport crates and vehicles are cleaned and disinfected properly on every occasion; and

- maintain general biosecurity and hygiene barriers at a high level, to prevent infection from the farm environment.

10.21 We strongly believe that concerted effort is needed by industry to improve the microbiological safety of thinning. If this cannot be achieved, then the case for discontinuing the practice, and taking the necessary measures to protect the welfare of stock, becomes very strong.

10.22 In risk assessment terms, a lower incidence of *Campylobacter* in broiler flocks is also likely to be reflected in lower numbers of the organism in individual birds in the flock, and on finished carcasses. Reducing the number of *Campylobacter*-positive flocks can be expected to have a significant impact on the numbers of contaminated birds leaving the slaughterhouse and may also facilitate flock testing to enable positive birds to be put through the slaughterhouse at the end of the day, immediately before plant and machinery are shut down and cleansed. It might also offer the option of directing positive flocks to heat treatment or freezing if these were found to be helpful in reducing *Campylobacter* loadings. An important factor in consumer exposure to *Campylobacter* in poultry meat is the frequency and level of contamination of the chicken brought into the home or into catering kitchens.

10.23 We accept the advice we have received from various parts of the poultry industry that broiler chicken production is extremely price competitive and that the industry is faced with continuing threats of import penetration.

10.24 We do recognise that many of the measures for controlling *Campylobacter* in chicken imply additional production costs. However, there is increasing evidence that there are direct links between the general health status of birds and their susceptibility to *Campylobacter* infection. In addition, the maintenance of good flock health conveys economic benefits. Measures put in place for the control of *Campylobacter* might also help reduce the risk of introducing other infections into the flock.

10.25 In order to be able to evaluate the effectiveness of measures to tackle *Campylobacter*, good quality data are needed on the *Campylobacter* status both of flocks and retail product. Flock prevalence studies are an essential feature of any evaluation process, to establish a baseline and to monitor progress under commercial conditions. We believe that Defra should organise such studies. In addition, we assume that the FSA will continue to use routine surveillance of retail chicken for *Campylobacter* to assess the effectiveness of *Campylobacter* reduction programmes. The potential value

of industry data as an output measure should not be overlooked even if, for reasons of commercial sensitivity, such information cannot be made publicly-available outside the Agency. We discuss the valuable contribution a standardised approach to typing can make to tracing sources and routes of transmission of human *Campylobacter* infection in Chapter 9.

10.26 We recognise that free range and organic chicken production is now a small but significant feature of the UK market. Given the importance of the environment as a source of *Campylobacter*, we think it likely that chickens reared extensively will come into more frequent contact with *Campylobacter* and that robust biosecurity arrangements aimed at reducing the exposure of birds to *Campylobacter* spp. will be more difficult to apply in extensive production systems. It is important that consumers are aware of this, not least because one of the main reasons given for buying free range and organic chicken is that they see it as a healthier product (see Annex E). We believe that information based on structured UK surveillance of *Campylobacter* infection in extensively-reared broiler flocks and the *Campylobacter* status of extensively-produced, including free range and organic, chicken meat would be valuable in informing consumer choice. Means also need to be identified of controlling *Campylobacter* in extensive production systems.

10.27 Our principal recommendation is that the Food Standards Agency utilises the conclusions we have drawn to intensify its work with the poultry industry and other stakeholders to achieve wider acceptance that *Campylobacter* control of housed birds is now possible. A primary aim should be to develop an industry-wide programme to spread the "good farming" practices and biosecurity measures which lie at the heart of the matter. (Priority A)

10.28 We recommend that the FSA, in collaboration with Defra, as appropriate, should explore with industry the options for modifying thinning practices to reduce the threat to the biosecurity of broiler farms. If the necessary improvements cannot be made, the FSA and Defra should explore with industry the conditions which would allow the practice of thinning to be discontinued, notwithstanding the economic pressures to which industry has drawn attention. (Priority A)

10.29 If thinning is to continue, crate washing and other biosecurity measures (including clothing and footwear) need urgent improvement. We recommend that the FSA pursues this with stakeholders. (Priority A)

10.30 In order to facilitate evaluation of the effectiveness of *Campylobacter* reduction measures, and to improve controls at slaughter, we recommend that Defra carries out surveillance of *Campylobacter* in broiler flocks. We also recommend that the FSA

continues to perform routine surveillance of *Campylobacter* in retail chicken. (Priority A)

10.31 Extensive chicken production is a minor but nevertheless important feature of the UK market. We believe that consumers would benefit from knowing more about the *Campylobacter* status of this type of product. We therefore recommend surveillance:

- **by Defra to determine the prevalence of *Campylobacter* in extensively-reared flocks and the *Campylobacter* spp. involved; (Priority B)**

- **by the FSA to determine the *Campylobacter* status of free range, organic and other extensively-produced chicken meat on retail sale in the UK. (Priority B)**

10.32 We also recommend further research into how *Campylobacter* can be more effectively controlled in extensively-reared chickens. We note that the FSA is already considering funding research in this area and welcome the fact that the Agency has invited Expressions of Interest from researchers. (Priority B)

Chapter 5: Measures to prevent *Campylobacter* contamination of chicken meat in Scandinavia

10.33 The ACMSF's overall conclusions drawn from visits to Denmark and Norway by some of the members of its *Campylobacter* Working Group are that:

- nothing that the sub group saw in either Denmark or Norway served to undermine the Committee's views on the feasibility of the on-farm control of *Campylobacter* in housed chickens;

- indeed, the Norwegian experience especially offered further encouragement that on-farm control in housed birds is achievable on a commercial scale;

- Denmark appears to have established a premium market for *Campylobacter*-free chicken;

- Norway has succeeded in getting the contamination rate for fresh chicken products in retail outlets down below 10%;

- the UK broiler industry still has some catching up to do but is, for the most part, on the right track;

- however, the UK industry needs to be encouraged to maintain its best endeavours;
- opportunities for collaboration between researchers here and those in Denmark and Norway were identified;
- some thought needs to be given to the efficacy and wider implications of heat treating or freezing *Campylobacter*-positive carcasses;
- the potential for airborne transmission of *Campylobacter* on farms may need further investigation but could necessitate some quite detailed research.

10.34 Sweden has succeeded in reducing overall *Campylobacter* infection in flocks to below 10%. It is encouraging that, within this figure, around half of all broiler farms were able to keep *Campylobacter* out of flocks completely. The methods used to achieve these results (eg. robust biosecurity, dry litter) are not innovative and are readily applicable to the UK setting. This information about the situation in Sweden provides further support for the observations made and the conclusions drawn by members of our *Campylobacter* Working Group following their visit to Denmark and Norway.

Chapter 6: *Campylobacter* in poultry other than chicken

10.35 Such evidence as we have seen suggests that all commercial poultry species are as susceptible as chicken to *Campylobacter* colonisation. However, we note that there appears to be little hard information available about the UK situation, and most of the data quoted in this Report come from abroad.

10.36 We recommend that, in addition to the work it is doing on chicken meat, the FSA carries out surveillance to establish the *Campylobacter* status of other types of poultry meat on retail sale in the UK. (Priority A)

Chapter 7: Measures to prevent *Campylobacter* contamination of meat other than chicken and other poultry meat

10.37 *Campylobacter* spp., including those which cause human disease, are likely to be widespread in the environment, and it is not surprising that food producing animals such as cattle, sheep and pigs are exposed to this organism. In terms of risk management, it seems sensible to assume that all flocks and herds will contain animals which are likely to be colonised with *Campylobacter* and to take steps during the slaughter process to minimise the likelihood that these are transferred to the final products leaving the plant. The control measures required to achieve this aim will be essentially the same for *Campylobacter* as for organisms such as *Salmonella* and VTEC.

We do not therefore consider that there is a need for *Campylobacter*-specific measures.

10.38 We recognise that both Government and industry have developed and put in place a number of measures to minimise the possibility of faecal material being transferred from the gut (or indeed the hide and fleece) during the slaughter process. We believe that, if properly applied, these will provide an effective barrier against *Campylobacter* contamination.

10.39 Cross-contamination is a particular concern in relation to *Campylobacter*. We believe that the proposed new EU meat inspection requirements, by reducing the use of palpation and incision, will help reduce the risk of cross-contamination. We agree that improving the flow of information across the food chain is likely to assist traceability and facilitate application of disease control and food hygiene measures at the most effective points.

10.40 We believe that the quality of the information trail would be further enhanced if *Campylobacter* flock prevalence data were available at slaughter. We address this is Chapter 4.

10.41 In view of the variations in the data for the prevalence of *Campylobacter* in retail meat samples, and in order to obtain a clearer picture of the risk if any to public health from such products, **we recommend that the Food Standards Agency should undertake UK wide, large-scale, structured surveillance of the prevalence of *Campylobacter* in red meat on retail sale**. **(Priority A)** We note that the Agency has recently requested pilot work in this area.

Chapter 8: Measures to prevent *Campylobacter* cross-contamination in domestic and catering environments

10.42 Raw poultry meat, particularly chicken, is, and will continue for the foreseeable future to be, a significant vehicle by which *Campylobacter* is introduced into the domestic and catering environments. Levels of *Campylobacter* contamination of $>10^5$ cfu are seen on some chicken carcasses. The human infectious dose is reported to be as low as 500 cells. Given the likely difficulties involved in controlling high levels of contamination in the kitchen, especially from sources such as raw poultry, we reiterate the critical significance of reducing the levels and incidence of *Campylobacter* on such products to lessen the burden on domestic and catering premises in dealing with such hazards.

10.43 If *Campylobacter* can be effectively tackled at the primary production stage, then the anticipated reduction in the number of *Campylobacter* cells reaching the kitchen would enhance the effectiveness of normal hygiene measures taken there. We make a number of recommendations below

designed to enhance the effectiveness of action in the domestic and catering environments.

10.44 We strongly recommend the proper use in catering of meat thermometers, as a means of ensuring the effective cooking of raw poultry products in particular. The use of such devices in the home may also yield benefits and we recommend that the Food Standards Agency (FSA) considers communicating the benefits of the use of cooking thermometers for domestic and catering settings. We also recommend that, as part of its review of HACCP implementation in the catering sector, the FSA considers whether documentary evidence of effective temperature checks should be required to be kept. (Priority A)

10.45 We recommend that industry guidance is produced through trade associations, to ensure a consistent approach to the generation of on-pack cooking instructions. In addition, where guidance is provided, this should feature prominently on the packaging. (Priority A)

10.46 In the case of meat which is not pre-packed, we recommend that the industry examines the feasibility of providing cooking guidance on all raw meat and poultry products, including those sold from service counters, butchers and other similar outlets. (Priority A)

10.47 We believe that the practice of washing raw meat and poultry is likely to lead to increased risk of spread of *Campylobacter* in the kitchen through splashes, droplets and aerosols, given the high levels which may be present on raw chicken. We recommend that this practice be actively discouraged by the FSA and industry. (Priority A)

10.48 Whilst it is understood that the consumer does not always read such advice, we recommend, in the public interest, that all producers and retailers of foods, where enteric pathogens such as *Campylobacter* may be present, should provide advice on the key food safety steps which should be taken to prevent infection. This should, as a minimum, be applied to all raw poultry products, as the levels of the organism are known to be high. Advice should include measures for effective cooking and for the avoidance of cross-contamination from the raw food to ready-to-eat food (through separation of foods and utensils, and through hand washing). (Priority A)

10.49 We are aware of previous activity by the FSA to provide targeted advice to consumers regarding improved cooking/hygiene practices when barbecuing and we recommend that this approach be repeated prior to each summer period. (Priority A)

10.50 We recommend that the industry provides food safety/hygiene advice on the packaging of foods for barbecues, particularly raw meats, and especially poultry. (Priority A)

10.51 We recommend that attention is drawn to the potential risks associated with carriage of *Campylobacter* in domestic pets, and to the appropriate hygiene measures that should be adopted. (Priority A)

10.52 We recommend that further measures are taken to embed food hygiene and safety principles into the education of primary and secondary school children. (Priority C)

10.53 In light of the fact that basic precautions may not be sufficient to prevent *Campylobacter* cross-contamination from highly contaminated foods such as raw chicken, we recommend that the FSA considers what measures can be taken to highlight to caterers and consumers the heightened risks associated with certain foods such as raw poultry. (Priority A)

Chapter 9: *Campylobacter* detection and typing

Detection

10.54 It is likely that, for the foreseeable future, traditional culture techniques will be employed for the examination of clinical and non-clinical samples for *Campylobacter* spp. More rapid methods are now available but there is a need to ensure that they have sufficient sensitivity. Any method will suffer from inherent bias and this will continue to be a problem in studies on epidemiology and in comparison of different surveillance schemes. The isolation of *Campylobacter* would seem to be more difficult than for *Salmonella*, for example, and there is a need for more rigour in method choice and for a properly structured, multi-laboratory study of the most commonly used methods. This is an area our *Campylobacter* Working Group will need to consider in reviewing research needs relating to *Campylobacter* (see Chapter 1).

Typing

10.55 Speciation of *Campylobacter* has proved useful in differentiating epidemiologically between *C. coli* and *C. jejuni*.

10.56 *Campylobacter* typing should be driven by objectives and/or specific hypotheses. These might be:

- tracing sources and routes of transmission of human infection;
- identifying and monitoring, both temporally and geographically, strains with important phenotypic or genotypic characteristics;
- developing strategies to control organisms within the food chain;
- monitoring trends in antimicrobial resistance; and
- outbreak identification

The method(s) chosen should then be dictated by specific objectives and/or hypotheses. In addition to this, appropriate sampling frames should be used.

10.57 Typing has confirmed the complexity of the epidemiology of *Campylobacter* infection but, on a broad scale, has not yielded the expected public health benefits in terms of identifying a big target amenable to control. Routine typing is probably not useful for source tracing and global epidemiology because of the carriage of multiple strains in animals, and the extreme diversity of those strains. Completing the analyses of the CSSS is, however, needed since the requirement for national strain-specific epidemiological studies might yet be demonstrated.

10.58 Where the objective is to make an assessment of *Campylobacter* across the food chain in relation to human infection, veterinary, food and clinical laboratories should use the same methods. One of the problems with comparative epidemiology is that different techniques have been applied to different specimen types. It is important that, when carrying out research and surveillance in animals, isolation and typing methods should take the lead from, and be consistent with, methodologies used for clinical isolates unless there are specific reasons not to do so. This recognises the fact that *Campylobacter* is primarily of public health significance.

10.59 The technological revolution in clinical medicine means that, in future, direct detection and typing using clinical samples will be possible. An obvious benefit of this approach is the speed of diagnosis and the potential for real time epidemiology. DNA sequence-based methods like MLST therefore have the greatest potential to be “future-proof”.

10.60 The variability and genetic instability of *Campylobacter* cautions us against believing that there exists some magical solution to the typing of all campylobacters of human health significance. However, any improvement in the tools available for differentiating the origins or food sources of *Campylobacter* spp. would greatly assist the Food Standards Agency in tackling human campylobacteriosis. Examination of the history of the many typing methods summarised in Tables 9.1 and 9.2 prompted much discussion within the *Campylobacter* Working Group as to whether the ACMSF could make a firm recommendation in this area. We have now concluded that the DNA sequence-based MLST method offers an opportunity in the short to medium-term to improve our knowledge of what is a very complex epidemiological story.

10.61 The availability of whole genome sequence data offers the prospect of another potentially valuable approach to typing. DNA microarrays based on the complete set of sequenced genes offer a unique opportunity to investigate and compare genome composition for individual isolates of a species. This approach has been applied to *Campylobacter* and its further development and validation may provide a novel typing approach of practical value in the future.

10.62 As with all typing methods for food poisoning pathogens, different laboratories will take different approaches, and agreement will be difficult to reach. **We therefore believe that the Food Standards Agency needs to take a firm initiative in bringing together laboratories capable of applying MLST so that investigative programmes can be designed to improve our epidemiological understanding in the next few years. We so recommend. (Priority A)**

10.63 We want to be very clear that this is in no way a recommendation that, at this stage, the FSA should be funding large research programmes on typing methods. Rather, it is a recommendation that advantage is taken of the opportunity to get the most out of MLST in the shorter-term.

ANNEX A: Membership of the Advisory Committee on the Microbiological Safety of Food and the *Campylobacter* Working Group. Participants in the ACMSF *Campylobacter* workshop

Advisory Committee on the Microbiological Safety of Food

Terms of reference

To assess the risk to humans from microorganisms which are used or occur in or on food and to advise the Food Standards Agency on any matters relating to the microbiological safety of food

Membership

Chairman

Professor B Reilly	Head, Gastrointestional and Zoonoses Section, Health Protection Scotland

Members

Mr J Bassett	Microbiological Risk Assessor, Unilever Safety & Environmental Assurance Centre
Dr D W G Brown	Director, Enteric, Respiratory and Neurological Virus Laboratory, Health Protection Agency
Ms S Davies	Principal Policy Adviser, Consumers' Association
Professor M J Gasson	Head of Food Safety Science Division, Institute of Food Research
Dr K M Hadley	Senior Lecturer, Department of Immunology and Bacteriology, University of Glasgow. Honorary Consultant in Clinical Microbiology, North Glasgow University Hospitals NHS Trust, Western Infirmary, Glasgow
Professor T J Humphrey	Professor of Food Safety, University of Bristol
Professor P R Hunter	Professor of Health Protection, University of East Anglia
Mr A Kyriakides	Head of Product Safety, Sainsbury's Supermarkets Ltd
Ms E Lewis	Computer consultant. Consumer representative
Mr P McMullin	Senior Veterinarian & Managing Director, Poultry Health Sciences

Mr P Mepham	Environmental Health Manager (Policy and Support), Leeds City Council
Professor S J O'Brien	Professor of Health Sciences & Epidemiology, University of Manchester
Mr B J Peirce	Caterer
Mr D J T Piccaver	Farmer
Professor L J V Piddock	Professor of Microbiology, Univeristy of Birmingham
Dr Q D Sandifer	Director of Health Improvement, Kent and Medway Strategic Health Authority
Professor P Williams	Professor of Microbiology, University of Leicester

Assessors

Mr P J R Gayford	Department for Environment, Food and Rural Affairs
Dr J Hilton	Food Standards Agency
Dr G McIlroy	Northern Ireland Department of Agriculture and Rural Development
Dr S Pryde	Food Standards Agency (Scotland)
Mrs J Whinney	Food Standards Agency (Wales)

Secretariat

Administrative Secretary

Dr L Foster	Food Standards Agency

Scientific Secretary

Dr P E Cook	Food Standards Agency

Administrative Secretariat

Mrs E A Stretton	Food Standards Agency
Miss C L Wilkes	Food Standards Agency

Campylobacter Working Group

Terms of reference

To identify any important gaps and omissions in action taken to reduce *Campylobacter* in food and food sources and in the knowledge base; and to develop advice which will assist the Food Standards Agency in evolving its strategy for reducing the incidence of foodborne *Campylobacter* infection in humans

Membership

Chairman

Professor D L Georgala[1]

Professor B Reilly[2]

Members

Ms S Davies

Professor M J Gasson

Professor T J Humphrey

Professor P R Hunter

Professor A M Johnston

Mr A Kyriakides

Ms E Lewis

Professor S J O'Brien

Mr B J Peirce

Mr M Attenborough	Technical Director, Meat and Livestock Commission
Dr E Berndtson[3]	Svenska Klackeribolaget AB, Sweden. *Campylobacter* consultant to the Swedish Poultry Association

Assessors

Mr P J R Gayford

Dr J Hilton

Professor C H McMurray[4]	Northern Ireland Department of Agriculture and Rural Development
Dr S Neill[5]	Northern Ireland Department of Agriculture and Rural Development

Secretariat

Administrative Secretary

Mr C R Mylchreest[6]

Dr L Foster[7]

Scientific Secretary

Dr J P Back	Food Standards Agency

[1] Until 31 March 2004
[2] From 1 April 2004
[3] Until December 2002
[4] Until 16 August 2002
[5] From October 2002
[6] Until 5 April 2004
[7] From 6 April 2004

Administrative Secretariat
Mrs E A Stretton

Miss C L Wilkes

Participants in ACMSF *Campylobacter* Workshop: Britannia International Hotel, London Docklands: 13-14 February 2002

Participants

ACMSF members

Professor D L Georgala

Dr G R Andrews

Dr D W G Brown

Ms S Davies

Dr K M Hadley

Professor T J Humphrey

Mr A Kyriakides

Ms E Lewis

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Mr P J R Gayford

Professor C H McMurray

Dr S Pryde

Dr R Skinner Food Standards Agency

ACMSF Secretariat

Dr J Hilton

Mr C R Mylchreest

Mrs E A Stretton

Miss C L Wilkes

External participants

Mr M Attenborough

Dr J P Back

Dr E Berndtson

Dr K Callaghan Food Standards Agency

Miss M Castle	Food Standards Agency
Dr P E Cook	Food Standards Agency
Dr J M Cowden	Scottish Centre for Infection and Environmental Health
Miss O Doyle	Food Standards Agency
Mrs J Frost	Public Health Laboratory Service Central Public Health Laboratory
Dr E Hartnett	Department for Environment, Food and Rural Affairs Veterinary Laboratories Agency
Dr K Jones	Lancaster University
Dr J Knight	Food Standards Agency (Scotland)
Mrs J Lock	Food Standards Agency
Professor D Newell	Department for Environment, Food and Rural Affairs Veterinary Laboratories Agency
Dr R L Salmon	Public Health Laboratory Service Communicable Disease Surveillance Centre, Wales
Dr W van Pelt	National Institute of Public Health and the Environment, the Netherlands
Dr M Wooldridge	Department for Environment, Food and Rural Affairs Veterinary Laboratories Agency
Dr B Wren	London School of Hygiene and Tropical Medicine

Presentations

Mrs J Frost	*Campylobacter* detection and typing research: an overview
Dr K Callaghan	*Campylobacter*: the disease and the immune system: summary of FSA-commissioned work
Miss O Doyle	Other *Campylobacter* research
Professor D Newell	Animal models of *Campylobacter jejuni* disease
Dr B Wren	What has the *Campylobacter* genome sequence/genomics done for us?
Dr J Cowden	*Campylobacter* in the Infectious Intestinal Disease (IID) Study
Professor S O'Brien	What are the main sources/vehicles for human *Campylobacter* infection?
Dr W van Pelt	Some questions and possibilities for studies on *Campylobacter*: a Dutch point of view

Mr P Gayford	Prevalence of *Campylobacter* in animals
Dr P Cook	Prevalence of *Campylobacter* in meat and poultry
Miss O Doyle	Epidemiological studies of *Campylobacter* in Iceland
Professor D Newell	*Campylobacter* seasonality in human beings and food-producing animals
Dr K Jones	*Campylobacter* seasonality in food animals
Dr E Hartnett	Quantitative risk assessment for *Campylobacter* in chicken meat
Professor T Humphrey	The on-farm control of *Campylobacter* spp.: is this an achievable objective?
Dr K Jones	Environmental presence and persistence of *Campylobacter*

ANNEX B: Organisations supplying information to the ACMSF

B.1 Organisations representing a wide range of interests and expertise were invited to supply the ACMSF *Campylobacter* Working Group with information. Not all responded. Those who did, to whom we are especially grateful, are detailed below.

Oral evidence

B.2 The following organisations gave oral evidence to the Working Group:

- Assured Chicken Production
- British Poultry Council
- Farm Fed Chickens
- Institute for Animal Health
- Lloyd Maunder Ltd
- Marks and Spencer plc
- Moy Park Ltd
- Northern Ireland Department of Agriculture and Rural Development
- O'Kane Poultry Ltd
- University of Nottingham

Written evidence

B.3 The following organisations provided written evidence to the Working Group:

- Advisory Committee on Animal Feedingstuffs
- Danish Veterinary Institute
- Food and Drink Federation
- Local Authorities Coordinators of Regulatory Services (LACORS)

- Meyn Food Processing Technology BV
- Norwegian Zoonosis Centre

Visits

B.4 Some members of the Working Group undertook a familiarisation visit to Swanham's Farm, a broiler farm supplying Lloyd Maunder Ltd, and a Lloyd Maunder processing plant.

B.5 Three Working Group members visited Denmark and Norway. A report on this visit is included in Chapter 5.

ANNEX C

Advisory Committee on the Microbiological Safety of Food

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E. mail: colin.mylchreest@foodstandards.gsi.gov.uk

Dr J R Bell
Acting Chief Executive
Food Standards Agency
Aviation House
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WC2B 6NH

24 January 2003

ACMSF *CAMPYLOBACTER* WORKING GROUP

1. In connection with the efforts being made to tackle *Campylobacter*, especially in chickens, I thought it appropriate at this time to let you have the ACMSF's views on the situation in Scandinavia.

2. Three members of the *Campylobacter* Working Group (Tom Humphrey, Mac Johnston and Alec Kyriakides) made a short visit to Denmark and Norway in the week beginning 17 November 2002. We wanted the group to investigate whether the incidence of *Campylobacter* in commercially-reared chickens really was lower in these countries. We also asked the group to look at how the Danes and Norwegians were tackling *Campylobacter* in chickens, and to see whether there were any lessons which could be applied in a UK context. Jonathan Back (who is the *Campylobacter* Working Group's Scientific Secretary) also participated in the visit so is well placed to use the information gathered in developing the Agency's *Campylobacter* strategy.

3. Because, in setting up the *Campylobacter* Working Group, we had co-opted Dr Eva Berndtson, a *Campylobacter* consultant to the Swedish Poultry Association, we had not planned a visit to Sweden, the other major player in Scandinavia. Unfortunately, Dr Berndtson has recently had to resign from the Working Group because of pressure of other work. We nevertheless hope

that she will be able to provide us with useful material on the situation in Sweden and we are currently pursuing this with her.

Denmark

4. There were 4,620 recorded cases of human *Campylobacter* infection in Denmark in 2001, although the true figure is believed to be much higher, and similar to the UK incidence. There is a much more pronounced summer peak of infection than in the UK. The consumption of poultry meat is a significant risk factor and the Danes have carried out a risk assessment which shows that, where the number of campylobacters on chicken carcasses is reduced by freezing or other means, the risk of human infection is also reduced.

5. All poultry flocks in Denmark are subject to surveillance to determine their *Campylobacter* status. Standard protocols are used throughout Denmark, Norway and Sweden. Control of *Campylobacter* in broiler flocks is closer to the current UK position (and less developed than in Norway).

6. The Danes are sceptical about the possibilities for on-farm control. Very hot Danish summers present particular difficulties. Some broiler houses are left open for welfare reasons, and this undermines biosecurity. Danish action against *Campylobacter* is thus more focussed on intervention during or after processing. *Campylobacter* is thought to be particularly sensitive to freezing and work is in hand on the effects of freezing at –18°C for 10 days. The possible use of heat treatment at 75°C for 15 seconds is also being investigated.

7. The group visited a typical, broiler farm. There are broiler farms in the UK of a comparable standard. There were 7 houses each containing 31,000 birds. The farmer operated an all in/all out system. The farm was in good order and the buildings, though over 30 years old, were in good condition. There were 5-10 metres between houses and the site was coated with coarse gravel which was routinely sprayed for weeds. Each house had a 40 cm high, physical hygiene barrier. A wash hand basin was located away from the barrier and the house was not entered via an enclosed ante-room.

8. The group also visited a processing plant, similar to most in the UK. The company does, however, market *Campylobacter*-free chickens, sold at a premium. The requirement of Danish legislation is that “the flock shall be controlled to give a 95% guarantee that less than 1% of birds are infected with *Campylobacter*.” 300 samples per flock must be tested. The company has been involved in the development of a PCR method to provide information on *Campylobacter* status within 5 hours.

9. Overall, the group concluded that the current situation in the UK was close to that in Denmark. However, the Danes seemed to derive a real benefit, in terms of the quality of data produced, from a closer integration of

the human and animal health surveillance systems. It was also apparent that the regular testing of poultry flocks yielded important information about *Campylobacter* prevalence and seasonality, as well as about geographical differences in colonisation rates.

Norway

10. There has been a marked increase in the number of human cases of *Campylobacter* in Norway since 1997, the annual incidence being around 100 cases per 100,000 of the population. There is an approximate 50:50 split between numbers of cases acquired in Norway and those acquired abroad. There is a marked peak in human infections, approximately 75% of cases occurring in July-September. It is thought that many more cases are caused by water in Norway than in the UK. The consumption of poultry purchased raw is among the principal risk factors although, unfortunately, authoritative data on the level of chicken-associated human cases prior to the introduction of broiler intervention arrangements (see paragraph 11) are not available.

11. Given the rising incidence of human campylobacteriosis, and the association with poultry meat, Norway has introduced an Action Plan Against *Campylobacter* in Broilers. This provides for the surveillance of live animals, animals at slaughter, and poultry meat products. Ten composite faecal samples are collected on farms 4-8 days prior to slaughter. If these samples are *Campylobacter*-positive, the birds are slaughtered at the end of the day. Carcasses are either heat-treated, or frozen for 5 weeks. There is also follow up action on *Campylobacter*-positive farms. This comprises standardised consultations and the introduction of measures to reduce flock infection, namely the disinfection of drinking water and the introduction of hygiene barriers. There is also a farm-based research programme to identify risk factors for *Campylobacter* infection in flocks.

12. The Norwegian poultry industry is only about a tenth the size of the UK industry. Most birds are killed earlier than in the UK (at 32-33 days). In 1991, 18% of broiler flocks (sampled on-farm) were *Campylobacter*-positive. This had fallen to 4% in 1998. The most recent surveillance (2001-2002) produced an on-farm incidence figure of 7.6%. As with human infection, there is a marked seasonality, with around 90% of positive flocks being identified in the summer months.

13. The group visited a typical Norwegian broiler farm, comprising 1 house of 11,000 birds. Access to the house was via an ante-room which had three rooms, each with a door, coming off it. One room served as an office and had a window through which the flock could be observed. Access to the flock was through a door on the other side of the ante-room in which a physical hygiene barrier had been placed. There were dedicated overalls and

footwear on the bird side of the barrier. The room also contained a wash hand basin which the farmer used before putting on protective clothing and footwear. **These simple interventions were sufficient to protect birds from *Campylobacter* in spring, autumn and winter and, to some extent, in summer.**

14. The group also visited a poultry processing plant which was typical of most in Europe and employed no devices which were not already in use in the UK. The plant was smaller, and tighter for space than in the UK. Water usage was high. Unlike in the UK, birds were spray-chilled with cold water. Although Norway does not sell "*Campylobacter*-free" poultry at retail, the goal is to reduce the level of *Campylobacter* in broiler chickens at slaughter to as close to zero as possible.

15. The prevalence of *Campylobacter* contamination in fresh poultry products ranged between 4 and 10% over the period 1995-1998. Further fresh product surveys were carried out in 2001 (at production facilities) and 2002 (in shops). Just over 1,000 samples were taken in each survey. *Campylobacter* prevalence was <10% in 2001 and around 2% in 2002.

16. The group felt that Norway provided some useful indications of what could be achieved by targeted on-farm intervention. Hygiene barriers seemed a cheap and effective counter-measure which the UK industry should be pressed to adopt as a matter of urgency. The rather different epidemiology of infection among broilers in Norway, compared with the UK, perhaps indicates a particular source of infection in the summer and the possible involvement of contaminated air in its transmission. The potential for airborne transmission on farms may need further investigation. This could require some quite detailed research.

Overall conclusions from Denmark/Norway visits

17. Our overall conclusions drawn from the group's visits are that:

- nothing the group saw in either Denmark or Norway served to undermine the advice I sent you on 26 September 2002 about the feasibility of the on-farm control of *Campylobacter* in chickens;

- indeed, the Norwegian experience especially offered further encouragement that on-farm control is achievable on a commercial scale;

- Denmark appears to have established a premium market for *Campylobacter*-free chicken;

- Norway has succeeded in getting the contamination rate for fresh chicken products in retail outlets down below 10%;
- the UK broiler industry still has some catching up to do but is, for the most part, on the right track;
- however, the UK industry needs to be encouraged to maintain its best endeavours;
- opportunities for collaboration between researchers here and those in Denmark and Norway were identified;
- we need to give some further thought to the efficacy and wider implications of heat treating or freezing *Campylobacter*-positive carcasses.

Sweden

18. As noted earlier, we are actively seeking information about the situation in Sweden which we can incorporate into our final Report, along with a more detailed summary of the Denmark/Norway visits, as part of a Scandinavian overview. In the meantime, if what we obtain about Sweden provides any new insights into how best to tackle *Campylobacter*, I will let you know.

19. I am copying this letter to Andrew Wadge and Judith Hilton.

Yours sincerely

DOUGLAS L GEORGALA

ANNEX D: Assured Chicken Production

Introduction

D.1 Assured Chicken Production (ACP) is one of the organisations which gave evidence to the ACMSF *Campylobacter* Working Group. ACP sets nutrition and welfare standards for poultry and verifies compliance with these standards by producers who are members of the Scheme. ACP is also committed to developing standards to achieve high levels of food safety and environmental care.

ACP structure

D.2 In its evidence to the *Campylobacter* Working Group, ACP explained that it is an independent company owning and developing the Assured Chicken Production Scheme standards for poultry. It is a company limited by guarantee. Membership of the company comprises the British Retail Consortium, the British Poultry Council, and the National Farmers' Union of England and Wales.

D.3 ACP is controlled by a Board of Directors responsible for the direction, overall management, and administration of the company. A Technical Advisory Committee monitors and sets the standards for the Scheme. A company operates a certification system on behalf of ACP. This is linked to a Certificate of Approval and the approved scheme mark. The certification system requires the examination of product, the production process, the production environment, and assessment of the quality management system.

D.4 The ACP Scheme covers some 90% of the poultry industry. Any site involved in chicken production is eligible to apply to join the ACP Scheme. Following assessment and acceptance, members' performance is subject to on-going surveillance.

ACP poultry standards[335]

D.5 ACP operates very detailed poultry standards applicable in respect of breeder replacement farms, breeder layer farms, and free range chickens for human consumption. The standards cover the farm site and emergency plan; health and hygiene; management and stockmanship; feed and water; the environment; provisions for chicks and breeder layer flocks; records; and depopulation.

D.6 Similar standards apply in relation to hatcheries; and there are detailed provisions covering catching, transport and slaughter.

D.7 Features of the standards include

- health and welfare programmes tailored to the needs of individual units;
- measures covering bird health;
- training to improve stockmanship;
- detailed rules on feed and water, the construction and maintenance of buildings, and lighting, temperature, ventilation, air quality and litter.

D.8 Standards stipulate the biosecurity measures which must be employed. ACP Scheme members are required to monitor for *Salmonella* but there are no measures specifically aimed at tackling the problem of *Campylobacter*. In its evidence to the *Campylobacter* Working Group, ACP identified areas where it thought *Campylobacter* could be most effectively addressed on-farm. These were the clothing, equipment and behaviour of stockmen, the sanitation of crates and other equipment, improved biosecurity in poultry houses, and staff education in general. Other areas being closely monitored are vaccine development, competitive exclusion, the use of bacteriophage, and the breeding of genetically resistant birds.

ANNEX E: The UK market for extensively-reared poultry

Production and marketing

E.1 Although a growing sector, extensively-reared (ie. organic or free range) chicken comprises a relatively small element of the overall chicken meat market. Approximately 1.2 million tonnes (carcass weight equivalent) of chicken meat is currently produced annually in the UK. Of this, approximately 4% (c. 50,000 tonnes) is produced extensively.[336]

E.2 However, there has been a steady increase in sales of organic meat (including poultry meat) in the UK in recent years (see Table E.1).

Table E.1: Sales of organic meat (including poultry meat) by value, 1996-2001

	Value (£m)	Index	Value (£m) at 1996 prices	Index
1996	26	100	26	100
1997	32	123	32	123
1998	42	162	41	159
1999	53	204	52	199
2000	67	258	66	253
2001 (est)	83	319	81	310

Source: Mintel[337]

E.3 Organic poultry meat is estimated to have accounted for 0.54% of the British poultry meat market in 2001, a figure predicted to rise to over 1% by 2008 (see Table E.2).

Table E.2: The British market for organic poultry meat

Year	Volume (tonnes)	Value ($ million)	Increase over 1998 value
1998	853	7.3	–
1999	1,200	9.8	34%
2000	1,956	14.7	101%
2001	3,500	25.0	242%
2008 (Forecast)	7,259	50.3	589%

Source: Organic Monitor[338]

E.4 One source[338] has organic chicken accounting for more than 95% of organic poultry meat sold in the UK (the only other significant organic poultry meat sold in the UK being organic turkey), and more than one-third of organic poultry meat sold in the UK coming from imports in 2001. France is identified as the major source of these imports, although it is noted that a significant expansion in UK production resulted in a fall in imports in 2002 (when they accounted for only 14% of supplies).

E.5 The shares of the principal UK producers of organic poultry meat to the domestic market are shown in Table E.3.

Table E.3: The British market for organic poultry: market shares of major suppliers: 2001

Company	Production (tonnes)	Market share
Moy Park	1,000	28.6%
Premier Fresh Foods	1,000	28.6%
Lloyd Maunder	400	11.4%
Others	1,100	31.4%
Total	**3,500**	**100.0%**

Source: Organic Monitor[338]

Consumer perceptions and demand

E.6 Research suggests that consumers are drawn to organic foods for a variety of reasons. A Consumers' Association survey in 2001[339] found that 30% of respondents always, usually or sometimes bought organic. Thirty two per cent of these said the reason was because 'it's healthier/better for you generally'. Twelve per cent bought organic for 'better standards of animal welfare.' The most popular reason given was, however, 'taste' (34%). The results are shown in Table E.4.

E.7 Data from the annual TGI survey looking at food safety in 2001, and drawn from a nationally-representative sample of 25,000 adults, found that 32.8% agreed with the statement 'I buy free-range products wherever I can'. This was down slightly on the 2000 figure of 34.9%. This survey also found that 20% of those surveyed felt that it was worth paying more for organic foods. This rose to 25% in the 25-44 age group, and 29% for socio-economic group ABs. Therefore, while this is still a limited sector, many consumers are now choosing organic and free range poultry products. Both of these terms are defined within EU regulations which apply across the Community.

Table E.4: Reasons for buying organic

Prefer the taste	34%
Less use of pesticides	32%
It's healthier/better for you generally	32%
It's a more natural process	24%
Less use of drugs in animals	17%
It's better for the environment	14%
Concern about GM	14%
It has more vitamins and minerals	12%
Better standards of animal welfare	12%
Prefer the texture/appearance	7%
My family/friends prefer it	5%
Lower risk of BSE	4%
Lower risk of food poisoning	3%
Don't know	8%

(Base: 482 – all buy organic food at some time)

Source: Consumers' Association

Organic production standards

E.8 Organic poultry production is based on the rigorous application of a range of production and welfare considerations. The term 'organic', when applied to agricultural products and foodstuffs, is controlled by the Organic Products Regulations 2001 which implement the requirements of EC Council Regulation 2092/91. Standards for organic livestock production came into effect from August 2000, following an amendment to the legislation.

E.9 Disease prevention in organic livestock is based on the following principles:

- the selection of appropriate breeds;
- the application of animal husbandry practices appropriate to the requirements of each species, encouraging strong resistance to disease and the prevention of infections;
- the use of high quality feed, together with regular exercise and access to pasturage, having the effect of encouraging the natural immunological defence of the animal;
- ensuring an appropriate density of livestock, thus avoiding over-stocking and any resultant animal health problems;

- prohibition on the use of substances to promote growth or production (including antibiotics, coccidiostats and other artificial aids for growth promotion purposes);

- prohibition on the use of chemically-synthesised allopathic veterinary medicinal products or antibiotics for preventive treatments;[a]

- an 81 day minimum age for slaughter of poultry (except in the case of slow growing breeds);

- specific provisions for transport aimed at minimising stress. For example, during transit each bird should have sufficient space to rest and stand up without restriction, and birds should be protected from undue fluctuations in temperature, humidity or air pressure, and sheltered from extremes of weather;

- full inspection of the production unit at least once a year, and the possibility of unannounced inspection visits by the inspection body.

Free range standards

E.10 While organic poultry must be free-range,[b] poultry labelled as 'free range' will not necessarily be organic. The criteria for stocking densities also differ between the two categories – with stricter criteria for organic production. The term 'free-range' is defined within EC Council Regulations 1906/90 and 1538/91 which lay down certain marketing standards for poultry meat.

E.11 'Free range' may only be used where the stocking rate in the house is 13 birds (not more than 27.5 kg liveweight) per square metre and where the birds are slaughtered at 56 days or later. In addition, the birds must have continuous daytime access to open-air runs comprising an area mainly covered by vegetation of not less than $1m^2$ per chicken. The feed formula used in the fattening stage must contain at least 70% of cereals. The poultry house must be provided with pop holes of a combined length of at least equal to 4m per $100m^2$ surface of the house.

[a] the prohibition on preventative treatment does not mean that veterinary medicines cannot be used (albeit with increased withdrawal periods) if the health of the stock warrants it. This could include action where there were indications of *Campylobacter* infection.

[b] In practice, standards for organic poultry are slightly less onerous than for birds officially designated as 'free range'. Free range birds must have continuous day time access to the open air. Organic birds on the other hand must have access to an open air run whenever weather conditions permit and, where possible, must have access for at least one-third of their lives.

Campylobacter control measures

E.12 As noted in Chapter 4, we think that extensively-reared chickens are likely to come into more frequent contact with *Campylobacter* which is ubiquitous in the environment. We also believe that it will be very difficult to maintain high levels of biosecurity in the extensive production setting. Given the fact that an important reason why consumers buy organic is because they consider organic produce to be healthier and better for you,[339] it is important that consumers are aware of this risk. This question is addressed in Chapter 4. Given consumer interest in this sector – for a variety of reasons, including animal welfare – it is also important that more research is undertaken into both the prevalence of *Campylobacter* in extensively-reared birds and how this could be more effectively controlled.

ANNEX F: Implementation of Recommendations

Recommendation	Response
(2.37) We recommend that the Government should instigate a primary care-based sentinel surveillance system, aimed at measuring directly the incidence and economic cost of long-term *sequelae* among cases of *Campylobacter* infectious intestinal disease. (Priority B)	• Possible FSA research requirement in 2005-06. • The Agency is currently considering funding for a second Infectious Intestinal Disease (IID) study to provide updated information on the burden of illness including campylobacteriosis but this will not include the long-term *sequelae*. Due consideration will be given to this recommendation and mechanisms of funding once the proposed second IID study has been progressed.
(2.38) We recommend that serological markers for recent infection and prior immunity be developed and tested through structured, epidemiologically robust, population-based studies. This should assist with estimating the prevalence of asymptomatic infection in the population (and hence estimating more accurately the magnitude of *Campylobacter*-associated *sequelae*). (Priority C)	• Possible FSA research requirement in 2005-06. • The FSA is already funding work using serological markers to look for evidence of VTEC O157 infection using non-invasive samples (e.g. saliva). Further work is needed to develop robust markers for *Campylobacter* before such tools can be applied in population-based studies to identify patterns and trends.
(3.30) The contribution of foodborne transmission (as opposed to other transmission modes) to the human toll of *Campylobacter* needs to be better defined and we note that the FSA has already funded a research project designed so to do. We support this course of action. (Priority A)	• The Agency is funding several research projects including a case control study of risk factors for *Campylobacter* infectious intestinal disease in England and Wales, and work on the burden of environmental and waterborne sources of *Campylobacter*, and will take stock when this programme of research completed.

Recommendation	Response
(3.31) We recommend that population studies to investigate the seasonality of *Campylobacter* infection be undertaken. An approach combining epidemiological, microbiological, environmental and veterinary expertise is likely to be needed. (Priority A)	• The Agency is planning a meeting of key groups to be held in 2005 to look at the feasibility of linking studies on *Campylobacter* in human illness, animals, and the environment to the rolling surveillance of food. • Current surveillance of retail chicken in Wales is already providing information on seasonality of contamination and more extensive sentinel surveillance is to be initiated by LACORS/HPA/FSA in November 2004. This study will also include a Local Authority administered follow-up questionnaire for laboratory confirmed sporadic cases of campylobacteriosis and salmonellosis to enable the HPA to identify outbreaks and common factors linked to infections. • FSA are also funding work in NW England looking at the role of environmental factors such as water.
(3.32) We recommend that population studies to investigate cultural/behavioural risk factors for *Campylobacter* be undertaken. (Priority B)	• To await outcome of study on cross-contamination in 2004-05. • The Agency will be funding a review of studies on cross-contamination in the home and this may assist in identifying specific gaps where population studies could be undertaken. • Some information is also likely to arise from the *Campylobacter* epidemiology studies being put in place. In addition to the LACORS/HPA/FSA sentinel surveillance of poultry meat, LAs will be gathering information on sporadic cases of campylobacteriosis and salmonellosis to enable the HPA to identify outbreaks and common factors linked to infections.

Recommendation	Response
(3.33) We recommend that more extensive data are gathered on the levels of *Campylobacter* spp. in specific foods (e.g. water, dairy products, vegetables, poultry and red meat) as well as in food-producing animals and companion animals. These are all potential sources of exposure for humans. We recommend that consideration be given to on-going surveillance as well as to "snap-shots" which tend to be the norm. It is very important that the microbiological methods employed allow meaningful comparisons to be made across the food chain. (Priority A)	• Surveillance of chicken to continue for at least the next 3 years probably on rolling basis. The Agency will review this as a basis for considering roll-out surveillance to other types of food. A sentinel LA based sampling program has been established by LACORS/HPA/FSA to provide data on *Salmonella* and *Campylobacter* contamination of raw chicken on an ongoing basis. Sampling under this new initiative is expected to start in November 2004. A Defra-funded abattoir survey has been carried out by the VLA. • The Agency also tries to make sure *Campylobacter* is included in current and future HPA/LACORS surveys, where appropriate. Collation of this survey data by the Epidemiology of Foodborne Infections Group (which reports to the ACMSF) will help identification of priorities for further work.
(4.65) Our principal recommendation is that the Food Standards Agency utilises the conclusions we have drawn to intensify its work with the poultry industry and other stakeholders to achieve wider acceptance that *Campylobacter* control of housed birds is now possible. A primary aim should be to develop an industry-wide programme to spread the "good farming" practices and biosecurity measures which lie at the heart of the matter. (Priority A)	• The Agency launched the second stage of the biosecurity campaign in October 2004. • Initial stage of biosecurity campaign to educate and train poultry farmers in best practice was launched on 19 January 2004. • Stage 2 of the campaign, launched on 4 October, involves face-to-face communication to farmers of the basic biosecurity messages, why they are important, and the evidence that they are effective in reducing *Campylobacter*. The campaign messages will continue to be communicated at Growers meetings over the autumn, with follow-up seminars in spring 2005.
(4.66) We recommend that the FSA, in collaboration with Defra, as appropriate, should explore with industry the options for modifying thinning practices to reduce the threat to the biosecurity of broiler farms. If the necessary improvements cannot be made, the FSA and Defra should explore with industry the conditions which would allow the practice of thinning to be discontinued, notwithstanding the economic pressures to which industry has drawn attention. (Priority A)	• The Agency has commissioned research to provide detailed and practical information on best practice, which will enable publication of a code of best practice on thinning for the UK poultry industry.

Recommendation	Response
(4.67) If thinning is to continue, crate washing and other biosecurity measures (including clothing and footwear) need urgent improvement. We recommend that the FSA pursues this with stakeholders. (Priority A)	• As per 4.66. • The Agency will work with stakeholders to identify improvements in other biosecurity measures and promote these in the next stage of the biosecurity campaign. • Output from FSA project MO1023 will identify the best operating regime for existing crate washing systems, identify simple improvements which can be made to the equipment now, and propose measures which can be incorporated in future designs. Best practice information developed within this project will be communicated to the poultry processing industry during Autumn 2004 as part of the Agency's *Campylobacter* biosecurity campaign.
(4.68) In order to facilitate evaluation of the effectiveness of *Campylobacter* reduction measures, and to improve controls at slaughter, we recommend that Defra carries out surveillance of *Campylobacter* in broiler flocks. We also recommend that the FSA continues to perform routine surveillance of *Campylobacter* in retail chicken. (Priority A)	• Surveillance of chicken to continue for at least the next 3 years, probably on rolling basis. • The Agency has been working with LACORS/HPA to develop a rolling survey approach to monitor the prevalence of *Campylobacter* in raw chicken The survey is expected to start in November 2004. • FSA are currently discussing options for funding *Campylobacter* flock surveillance as an add-on to flock surveillance for *Salmonella* carried out by Defra under the Zoonoses Directive.
(4.69) Extensive chicken production is a minor but nevertheless important feature of the UK market. We believe that consumers would benefit from knowing more about the *Campylobacter* status of this type of product. We therefore recommend surveillance: • by Defra to determine the prevalence of *Campylobacter* in extensively-reared flocks and the *Campylobacter* spp. involved; (Priority B) • by the FSA to determine the *Campylobacter* status of free range, organic and other extensively-produced chicken meat on retail sale in the UK. (Priority B)	• The Agency has been working with LACORS/HPA to develop a rolling survey approach for *Campylobacter* in raw chicken. The survey is expected to start towards the end of 2004 and will include chickens of different production types. The number of extensively produced chicken samples will be small reflecting market share although any differences in *Campylobacter* prevalence should be reflected in the longer term. However, differences between production types (if any) are likely to be masked by contamination arising during slaughter and processing. • As per 4.68.

Recommendation	Response
(4.69) *(contininued)*	• Defra is currently funding an epidemiological study with the aim of developing an effective farm to fork Quantitative Risk Assessment model that identifies practical control measures for *Campylobacter* in broiler flocks, and part of the study will also consider prevalence in extensively reared flocks as a potential source of infection.
(4.70) We also recommend further research into how *Campylobacter* can be more effectively controlled in extensively-reared chickens. We note that the FSA is already considering funding research in this area and welcome the fact that the Agency has invited Expressions of Interest from researchers. (Priority B)	• Research on the control of *Campylobacter* in extensively reared flocks will begin in November 2004. • Research projects will consider the use of plant extracts, probiotic bacteria, and dietary manipulation to control *Campylobacter.* • Under the Government Partnership Awards scheme, the Agency will part-fund a BBSRC project investigating bacteriophage therapy as an option for controlling *Campylobacter* in poultry.
(6.60) We recommend that, in addition to the work it is doing on chicken meat, the FSA carries out surveillance to establish the *Campylobacter* status of other types of poultry meat on retail sale in the UK. (Priority A)	• Await outcome of HPA/LACORS survey for data on turkey and other poultry meats in 2004-5. The Agency will consider this once the UK findings from the 2004 EC Co-ordinated sampling programme on poultry meat (including turkey) are known.
(7.25) In view of the variations in the data for the prevalence of *Campylobacter* in retail meat samples, and in order to obtain a clearer picture of the risk if any to public health from such products, we recommend that the Food Standards Agency should undertake UK wide, large-scale, structured surveillance of the prevalence of *Campylobacter* in red meat on retail sale. (Priority A) We note that the Agency has recently requested pilot work in this area.	• FSA will also take into account the findings from the ongoing surveillance of meats by the HPA/LACORS before undertaking any national meat surveillance. The Agency expects to commission pilot work in 2005-6 to develop methodology for a meat survey.

Recommendation	Response
(8.24) We strongly recommend the proper use in catering of meat thermometers, as a means of ensuring the effective cooking of raw poultry products in particular. The use of such devices in the home may also yield benefits and we recommend that the Food Standards Agency (FSA) considers communicating the benefits of the use of cooking thermometers for domestic and catering settings. We also recommend that, as part of its review of HACCP implementation in the catering sector, the FSA considers whether documentary evidence of effective temperature checks should be required to be kept. (Priority A)	• The Agency has, since 2002, published information on how to use probe thermometers and minimum cooking times for meat, as part of its Food Hygiene Campaign initiatives targeted at caterers and food business. • The Agency is considering what documentation and record keeping is appropriate for catering businesses of different types and sizes.
(8.25) We recommend that industry guidance is produced through trade associations, to ensure a consistent approach to the generation of on-pack cooking instructions. In addition, where guidance is provided, this should feature prominently on the packaging. (Priority A)	• Labelling rules already require instructions for use to be given if it would be difficult to make appropriate use of the food without them, and that where such instructions are given, they should be sufficiently detailed to enable appropriate preparation to be made of the food. The ACMSF has previously advised on the provision of appropriate cooking instructions for raw beef and poultry products, which are currently appended to our food labelling guidance. The Agency will consider incorporating this and the new recommendations into best practice advice on labelling of meat and meat products, which would be developed in consultation with industry. • The ACMSF is currently revisiting its advice on the time/temperature requirements for safe cooking of burgers and other minced meat products.
(8.26) In the case of meat which is not pre-packed, we recommend that the industry examines the feasibility of providing cooking guidance on all raw meat and poultry products, including those sold from service counters, butchers and other similar outlets. (Priority A)	• As above

Recommendation	Response
(8.27) We believe that the practice of washing raw meat and poultry is likely to lead to increased risk of spread of *Campylobacter* in the kitchen through splashes, droplets and aerosols, given the high levels which may be present on raw chicken. We recommend that this practice be actively discouraged by the FSA and industry. If necessary, consumers should be advised only to wipe down a chicken with a disposable paper towel. (Priority A)	• The Agency highlighted the risks of cross contamination from chicken in its hygiene campaign for consumers which included information on meat handling/washing. • Some retailers are considering including a note on poultry labelling about not washing chickens.
(8.28) Whilst it is understood that the consumer does not always read such advice, we recommend, in the public interest, that all producers and retailers of foods, where enteric pathogens such as *Campylobacter* may be present, should provide advice on the key food safety steps which should be taken to prevent infection. This should, as a minimum, be applied to all raw poultry products, as the levels of the organism are known to be high. Advice should include measures for effective cooking and for the avoidance of cross-contamination from the raw food to ready-to-eat food (through separation of foods and utensils, and through hand washing). (Priority A)	• FSA will take forward in parallel with the Food Hygiene Campaign. Campaign activities to date include use of TV advertising and web based activity to promote good hygiene practices and messages on safe cooking and how to avoid cross contamination. • FSA is aware that some retailers already provide food safety advice on their products although there is a need for more consistency in the advice that is given. • A number of retailers added advice on hygiene in connection with barbecuing to charcoal packaging in the summer of 2003, which has been retained in 2004. • The British Retail Consortium will be asked to bring this recommendation to the attention of their members.
(8.29) We are aware of previous activity by the FSA to provide targeted advice to consumers regarding improved cooking/hygiene practices when barbecuing and we recommend that this approach be repeated prior to each summer period. (Priority A)	• Prior to each summer period, the Agency will continue to issue advice on summer eating and barbecue cooking (supported by long-lead media activity). • The Summer Eating and barbecue campaign (including television and radio advertising, leaflet and website publicity) was launched in summer 2002. During summer 2003 and 2004, targeted advice relating to summer eating and barbecuing was repeated in leaflets and on the web site. Advice and leaflets are also promoted at publicity events.

Recommendation	Response
(8.30) We recommend that the industry provides food safety/hygiene advice on the packaging of foods for barbecues, particularly raw meats, and especially poultry. (Priority A)	• See recommendations 8.25 and 8.26.
(8.31) We recommend that attention is drawn to the potential risks associated with carriage of *Campylobacter* in domestic pets, and to the hygiene precautions applicable to them. (Priority A)	• This will be considered where food safety advice is being developed or revised.
(8.32) We recommend that further measures are taken to embed food hygiene and safety principles into the education of primary and secondary school children. (Priority C)	The Agency is working with the DfES and Devolved Administrations to raise the profile of food hygiene in the national curriculum. A number of measures have been launched including: • A "Cooking Bus" in November 2003 with the aim of delivering healthy eating and food safety messages to school children around the country. • The Agency's Bad Food Live video with full supporting teacher's pack has been made available to all primary and secondary schools across England and Wales. The video is aimed at 10-14 year olds and aims to highlight basic food hygiene messages and raise hygiene awareness. • The Agency will be supporting the roll out of 'Mission Possible!', which won the FoodLink National Food Safety Communications award in 2003. The scheme is aimed at primary school children aged 8-10 and is expected to reach 10,000 in this age category in 2005. • The Agency's website includes resources aimed at raising awareness of food hygiene issues amongst children and the material is due to be updated in 2004-05. • Several of the 2004-05 Local Authority food hygiene grants include initiatives relevant to raising hygiene awareness amongst children. • The Agency is funding research at Surrey University looking at the ways of getting hygiene messages into schools. • The Agency is also working with the Scout association to look at opportunities to raise food hygiene awareness amongst children and developing hygiene competencies in the DfES Getting to Grips with Grub scheme.

Recommendation	Response
(8.33) In light of the fact that basic precautions may not be sufficient to prevent *Campylobacter* cross-contamination from highly contaminated foods such as raw chicken, we recommend that the FSA considers what measures can be taken to highlight to caterers and consumers the heightened risks associated with certain foods such as raw poultry. (Priority A)	• FSA launched a high profile TV campaign in 2004 aimed at consumers to promote good hygiene practices and messages on how to avoid cross contamination from foods such as chicken. The FSA supported the FDF's FoodLink Food Safety week which also featured cross contamination.
(9.32) As with all typing methods for food poisoning pathogens, different laboratories will take different approaches, and agreement will be difficult to reach. We therefore believe that the Food Standards Agency needs to take a firm initiative in bringing together laboratories capable of applying MLST so that investigative programmes can be designed to improve our epidemiological understanding in the next few years. We so recommend. (Priority A)	• The Agency will initiate a programme of work in 2005 to bring together the key human, food and veterinary laboratories to examine the scope and resources required to undertake this work on a routine basis. • There is a new head of the HPA's *Campylobacter* Reference Unit and it will be important to establish links to identify the likely direction of future typing work. Preliminary discussions suggest that HPA will be considering implementing MLST and possibly other molecular-based typing tools. • The Agency is already funding work where MLST and other molecular based approaches are being applied to gain a better understanding of *Campylobacter* epidemiology. • We would also aim to explore the potential for using this approach in characterising *Campylobacter* isolates from the ongoing surveillance of retail chicken. • The Agency will explore the options for a longer-term archiving resource so that isolates collected as part of surveys and research are available for future comparisons using the most appropriate typing tools.
(9.33) We want to be very clear that this is in no way a recommendation that, at this stage, the FSA should be funding large research programmes on typing methods. Rather, it is a recommendation that advantage is taken of the opportunity to get the most out of MLST in the shorter-term and set in place the technology to adopt universal sequence-based techniques as they become available.	• If the key laboratories decide to put MLST in place as a front line typing tool then there may be scope for a ring trial/quality assurance exercise to assess performance. This is unlikely to be needed until 2005-2006.

Glossary (including acronyms)

Aetiology	The cause or origin of a disease.
Aetiological agent	The organism causing an infectious disease.
Antigen	A substance which elicits an immune response when introduced into an individual.
ATP	Adenosine 5'-triphosphate.
Case-control study	A study comparing a group of people with a particular disease (the cases) with a group of people free from the disease (the controls) to determine whether the cases have been exposed more or less often than the controls to a specific factor.
Chemotaxis	A taxis (see below) in which the stimulus is a concentration gradient of a particular chemical.
Cloaca	Common, faecal, urinary and oviduct outlet.
CLP	Clean Livestock Policy.
Coccoid cells	Spherical (or near-spherical) bacterial cells.
CSSS	*Campylobacter* Sentinel Surveillance Scheme.
Cytoplasm	The protoplasm (ie. the living contents) of a cell contained within the cell membrane, but excluding the nucleus.
DNA	Deoxyribonucleic acid, the genetic material of humans, bacteria, some viruses, etc. It is a polymer of nucleotides connected by sugars.
Enrichment	The process of increasing the proportion of a particular microorganism in a mixed population.
Epidemiology	The study of the occurrence, transmission and control of epidemic disease.
Epithelial cells	Cells which form the layer (the epithelium) lining the inner surface of the intestines.
fla typing	Flagellin gene restriction fragment length polymorphism.
Flagella-mediated motility	Bacterial locomotion through the action of flagella on cell surfaces.

Flagellin	The protein sub-unit of the filament of a bacterial flagellum.
Flagellum	A thread-like appendage on the surface of a cell whose movement is used for cellular locomotion.
Fomites	Objects or materials which have been associated with infected persons or animals and which potentially harbour pathogenic microorganisms.
FSA	Food Standards Agency.
GBS	Guillain-Barré syndrome: a disorder characterised by acute, bilateral ascending paralysis.
Gene clusters	A cluster of functionally-related genes.
Genome	The genetic material of an organism (ie. the DNA – see above – or RNA – see below – of a virus).
Global regulators	These subject genes and operons with diverse functions and independent control to a coordinated and overriding system of regulation.
Genotyping	Distinguishing and grouping organisms by their content of genetic information.
HPA	Health Protection Agency.
IFD	Indigenous foodborne disease.
IID	Infectious intestinal disease.
Insertion sequence	A small bacterial transposon (see below) which carries only the genes needed for its own transposition (see below).
Microarray	DNA microarrays are specially-treated microscope slides which carry an ordered mosaic of sequences representing most or all of the genes of an organism. DNA microarrays offer the ability to genotype or to monitor the expression of all genes in an organism at once (ie. they provide a snapshot of all the genes that are active in a cell at a particular time).

Microaerophilic	Describes a gaseous environment in which oxygen is present but at a concentration significantly lower than in air (partial pressure). A microaerophilic organism prefers, or can only survive in, such an environment.
MFS	Miller Fisher syndrome.
MLST	Multilocus sequence typing.
Operon	A group of contiguous structural genes which are transcribed as a single transcription unit from a common promoter and can thereby be subject to coordinated regulation.
Passage	The transfer of a pathogen from one to another of a succession of animals, tissues, etc, growth of the pathogen occurring before each transfer.
PFGE	Pulse field gel electrophoresis.
Phagocytosis	The process in which particulate matter is ingested (and may be subsequently digested) by certain types of cell or microorganism.
Phenotyping	Distinguishing and grouping organisms by their appearance and/or physiological (functional) properties.
RAPD	Random amplification of polymorphic DNA.
ReA	Reactive arthritis: a non-infective arthritis which may be secondary to an episode of infection elsewhere in the body.
Risk factor	A factor known, on the basis of epidemiological evidence, to be associated with a particular disease.
RNA	Ribonucleic acid, a nucleic acid consisting of ribonucleotides each of which contains one of the bases adenine, guanine, cytosine or uracil or, in some RNAs, a modified form of one of these bases.
Sequelae	Conditions which follow the occurrence of a disease e.g. late complications or long-term or permanent ill effects.
Serotyping	A method of distinguishing types of bacteria (serotypes) within a single species by defining their antigenic properties (see antigen) on the basis of their reaction to known antisera.

Taxis	A locomotive response to an external stimulus exhibited by certain motile cells or organisms.
Thermophilic	Thermophilic campylobacters are those which grow well at 42°C and 37°C, but not at 25°C.
Transposition	The translocation of a discrete DNA segment from one site to another (target) site.
Transposon	A genetic element which, in addition to encoding functions necessary for its transposition, also carries genes with functions unrelated to transposition (e.g. genes for resistance to antibiotics).
Vacuole	Any of the membrane-delimited compartments within a cell.
VNC	Viable Non-Culturable.

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Advisory Committee on the Microbiological Safety of Food

Second Report on *Campylobacter* Memorandum on Research

Advises the Food Standards Agency on the Microbiological Safety of Food

Introduction

1. The Advisory Committee on the Microbiological Safety of Food (ACMSF) recently submitted a draft of its Second Report on *Campylobacter* ('our Report') to the Chairman of the Food Standards Agency, prior to consulting publicly on the draft.

2. In the draft, we explained that, in order for our Report to be as useful as possible to the FSA in developing its *Campylobacter* reduction strategy, we had focused on short to medium-term practical options for tackling *Campylobacter*. We had not addressed those research opportunities and gaps falling into a longer time frame. However, we signalled the ACMSF *Campylobacter* Working Group's intention to meet again with the aim of identifying where research outputs, had they been available, would have contributed to progressing more quickly the objectives identified as desirable in our Report.

3. The *Campylobacter* Working Group met on 23 January 2004 to take matters forward. A summary of research[1] opportunities identified by the Working Group is given in the following paragraphs.

Research opportunities

Human immunity

4. There are still large gaps in our knowledge of human immunity to *Campylobacter* infection, and this lack of information hampers risk assessment and epidemiological studies. As we note in our Report, infected people mount a strong immune response to *Campylobacter*. Vaccination may therefore offer a possible control option. We also note that immunity against *Campylobacter* is possible in the absence of acute infection, many abattoir workers apparently being immune to infection after initial exposure. Given the continuing uncertainties surrounding human vulnerability and immunity to *Campylobacter* infection, improving our understanding of the mechanisms of protective immunity continues to be an important research objective. We do not discount the possibility that acquired immunity may be having a significant biasing effect on case-control studies.

[1] For the purposes of this exercise, the ACMSF has not defined 'research' rigidly. The term 'research' therefore also covers 'surveillance', particularly where surveillance results can play an important role in epidemiological research.

5. We propose that the Food Standards Agency (FSA), in collaboration with the Health Departments, should consider the possibility of undertaking further research to increase our knowledge and understanding of the human immune response to *Campylobacter* infection. We also take this opportunity to draw fresh attention to the recommendation in paragraph 2.38 of our Report that serological markers for recent infection and prior immunity should be developed and tested through structured epidemiologically-robust, population-based studies. We hope that this will assist in estimating the prevalence of asymptomatic infection in the population and, hence, estimating more accurately the magnitude of *Campylobacter*-associated *sequelae*.

Immunity in chickens

6. We note in our Report that a number of suggestions have been made to explain why chickens do not usually become *Campylobacter*-positive until the third week of life. However, current data on immune responses by chickens to *Campylobacter* remain equivocal, and further investigation could prove useful. Research to improve understanding of lag phase immunity could help inform the development of vaccination or other protection strategies.

Responses to stress

7. We touch in Chapter 2 of our Report upon *Campylobacter*'s response to environmental stresses and the debate as to the extent to which the organism is sensitive to these. We believe that further research in this area would enhance our understanding of the persistence and survival of *Campylobacter* in the environment and in food. Consideration should be given to funding work which seeks to explain bacterial behaviours as well as observing them. The complex micro-flora of the gastro-intestinal tract and its interaction with the host are important contexts that warrant more detailed study.

Seasonality

8. *Campylobacter* infection in humans and in food animals displays a noticeable pattern of seasonality. However, while seasonality patterns are well described, their underlying cause is poorly understood. We believe that further work is needed to improve understanding of both temporal and spatial variations in infection. One of the recommendations from our Report is that population studies should be undertaken to investigate the seasonality of *Campylobacter* infection, and that an approach combining epidemiological, microbiological, environmental and veterinary expertise is likely to be needed.

Food vehicles

9. We strongly believe that there is an important association between poultry meat and human *Campylobacter* infection. At the same time, we recognise that, in addition to the contribution of poultry to human *Campylobacter* infection, many studies also point to numerous other sources and vehicles of infection. It is important that these are not overlooked and we recommend, in our Report, that more extensive data are gathered on the levels of *Campylobacter* in water and specific foods (e.g. dairy products, vegetables, poultry and red meat), as well as in food producing animals and companion animals. We also recommend that consideration be given to on-going surveillance, as well as to the 'snap shot' surveillance projects which tend to be the norm.

Processing aids

10. We cover at some length in Chapter 4 of our Report the possible use of carcass treatments and other processing aids aimed at reducing *Campylobacter* on chicken carcasses. We believe that this is an area worth reviewing at regular intervals, to assess the effectiveness of such aids in reducing *Campylobacter* loadings. Of course, it is necessary to keep in mind any EU proscriptions on the use of processing aids, as well as consumer resistance to their use. We also wish to emphasise that the main focus for the control of *Campylobacter* in chickens should be the farm, and robust biosecurity regimes. Carcass treatments should not be regarded as a substitute for good hygiene practice.

Poultry other than chickens

11. It seems that all other commercial poultry species are as susceptible as chicken to *Campylobacter* colonisation. However, as we note in our Report, there are few data about the *Campylobacter* status of poultry meat (other than chicken) on retail sale. We have therefore recommended FSA surveillance to help clarify the picture.

12. We recognise that, compared with the market for chicken, consumption of other poultry is much lower (although turkey consumption is significant, especially over Christmas). We nevertheless believe that flock prevalence surveillance would yield useful data about the *Campylobacter* status of the live birds and suggest that this is something Defra might contemplate undertaking, perhaps on a 5-year cycle.

Functional genomics

13. The impact of whole genomic sequencing on *Campylobacter* research was highlighted in our Second Report on *Campylobacter*. Continued post-genomic and functional genomic research will be important in advancing understanding of disease-causing potential and pathogen survival throughout the food chain. In particular, a better definition of the genetic basis of pathogenicity and virulence, as well as its variation between strains, would improve *Campylobacter* epidemiology. There is considerable potential to exploit DNA microarrays to define isolated strains on the basis of their complement of functional genes, and this should be further developed.

Tackling the immediate problem

14. The research opportunities identified in our review exercise may only yield results in the medium to longer-term, given the time lag involved between identifying research and surveillance opportunities, and being able to apply practical outputs.

15. **We therefore wish to stress that implementation of the practical measures covered in our Report should not be delayed until the results of this further research or surveillance are available.**

Advisory Committee on the Microbiological Safety of Food
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