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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 Report¹ 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 FSA³ – 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 strategies

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 Agency⁷

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 Agency⁷

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 Report¹ 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 2000¹⁰ 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 regulator¹⁰ 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 30°C. 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 4°C, 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 toxin³⁵ (the existence of which is controversial, not least because it does not appear to be encoded on the genome¹⁰). 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:19⁵⁵ 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.

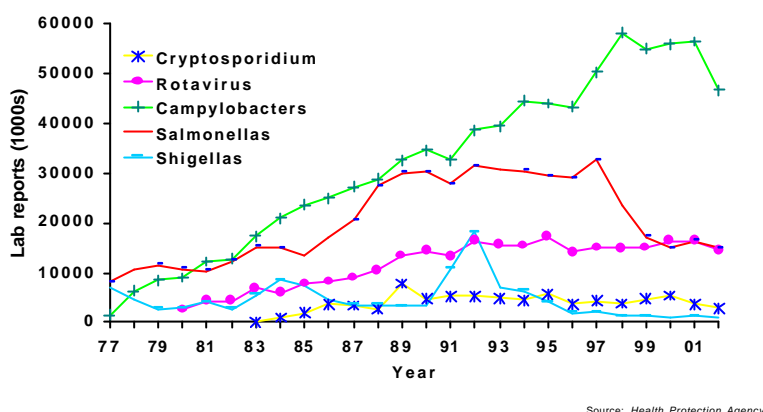
3.2 On 1 May 2000, the Public Health Laboratory Service (PHLS) – now the Health Protection Agency - launched an active, sentinel, population-based surveillance scheme for *Campylobacter* infections in England and Wales – the *Campylobacter* Sentinel Surveillance Scheme (CSSS).^{73,74} The aim was to 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. *Campylobacter*s 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

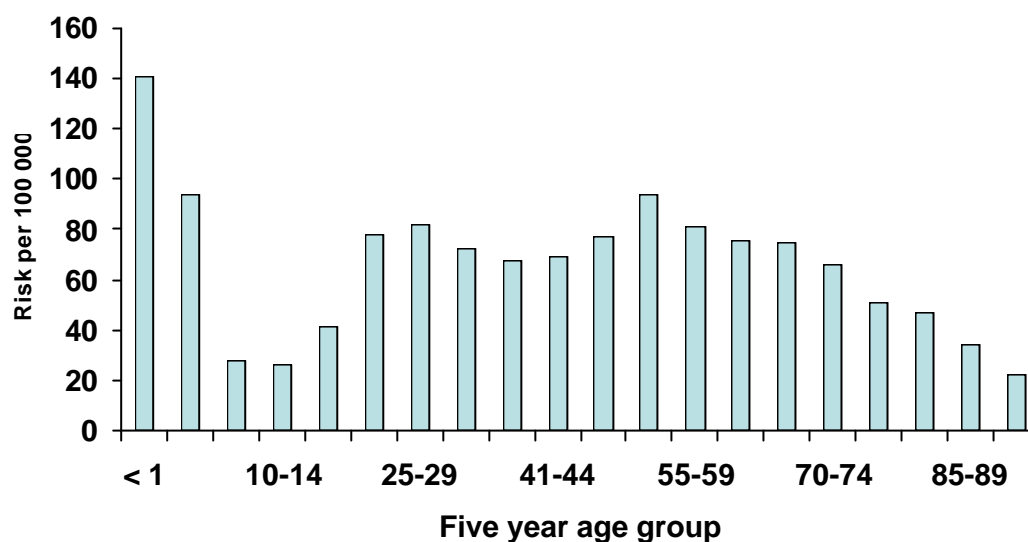
3.6 The economic burden due to *Campylobacter* infection is large. In the United States, the annual estimated cost is around US\$4.3 billion.⁷⁹ The average cost of a case of acute *Campylobacter* infection (excluding long-term sequelae) in England in 1995 was estimated to be £315.⁸⁰ Conservatively, therefore, 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 group⁸³ 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 flocks⁹⁴⁻⁹⁶ and dairy cattle⁹⁷ 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 evidence¹⁰⁴⁻¹⁰⁷ 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 water¹²⁶

Water

3.20 Consumption of untreated water,^{116,120} or rainwater¹⁰⁸ 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 diabetes¹¹⁴ or reducing gastric acidity through the use of proton pump inhibitors¹²⁸ 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 literature¹³³⁻¹³⁸ 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 *Campylobacter* 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 depopulation 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 Kapperud¹⁶⁶ 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

^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.

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 recommended¹⁷⁰ (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 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 predispose 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 monkeys¹⁷⁴ 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 sub-types 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 Agency¹⁸⁵ 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.

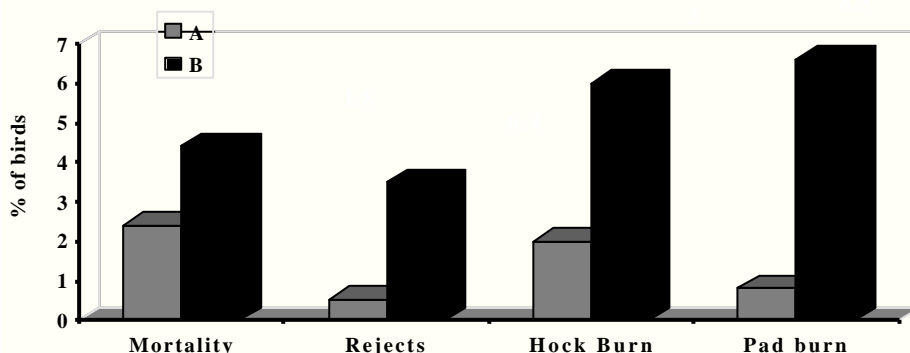
Broiler flock management and *Campylobacter* colonisation

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 sequences¹⁰ 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 study¹⁴¹ 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 study²⁰⁶ 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. One²⁰⁹ 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 work²¹³ 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 group²¹⁴ 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-log₁₀ 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-log₁₀ 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 <10³ to > 10⁹ 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 log₁₀/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 study²²⁴ 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 log₁₀ 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 log₁₀.²²⁷ 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 log₁₀ 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 or minus 30°C for 72 hours reduced pathogen numbers by 1.3 and 1.8-log₁₀ 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 log₁₀ cfu/g on wings held at minus 80°C, 0.8 log₁₀ cfu/g on wings held at minus 120°C, 0.6 log₁₀ cfu/g on wings held at minus 160°C and 2.4 log₁₀ 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 eg. 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 carcasses. Reducing the number of *Campylobacter*-positive flocks can also be expected to have a significant impact on the numbers of contaminated carcasses 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 carcasses of these birds. It would also offer the option of directing the processed carcasses 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 -18°C for 10 days. The efficacy of heat treatment (75°C 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 RCR testing protocol

Control	Samples	Analysis method	Comments
At the farm	3 x fresh faeces	PCR	If <i>Campylobacter</i> -negative, the flock can be slaughtered as <i>Campylobacter</i> -free.
At the slaughterhouse	20 samples of 25 cloacal swabs	PCR	If no <i>Campylobacter</i> is detected, the products can be sold with the <i>Campylobacter</i> -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 <i>Campylobacter</i> -free label.
Slaughterhouse samples	Chickens previously shown to be negative on the farm will be re-packed without the <i>Campylobacter</i> -free label.
Isolation of <i>Campylobacter</i> from farm samples, post-cleaning	Extra cleaning and extended control is carried out. If <i>Campylobacter</i> 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	3.1
	No	1.0
Physical hygiene barrier at entrance to chickens	Yes	1.0
	No	4.2
Routines for hand-washing	Always	1.0
	Never/sometimes	3.3
Water source	Private well	3.6
	Other private source	2.1
	Public source	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	<i>Total</i>	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/m²). Most houses are stocked to a density of at least 33 kg/m². Stocking density in houses with low welfare scores is restricted to 20kg/m².

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 <i>Campylobacter</i> -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 <i>Campylobacter</i> -positive	Year	% broiler flocks <i>Campylobacter</i> -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)**