FACTORY FARMING

AND

HUMAN HEALTH

A

COMPASSION IN WORLD FARMING TRUST

report by

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Foreword by
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Foreword

'Factory Farming and Human Health' is about making connections between concerns for animals and concerns for humans. The report is a devastating indictment of current meat production practices. In meticulous detail it explains how these culminate, not in the 'safest food in the world' as the politicians have tried to persuade us, but in food which imposes health risks on both humans and animals. The report's outstanding contribution is that it brings together for the first time the food safety and animal welfare agendas. They are, as the report says 'two sides of the same coin', for when animal welfare is jeopardised food safety is compromised.

It is no longer tenable to blame consumers themselves for the huge increase in food poisoning. Nor is it just a matter of 'a few cowboy producers who give the industry a bad name'. 'Factory Farming and Human Health' makes clear that it is precisely the commonplace and routine practices in animal production which are responsible. Far from being exceptional, the use of growth promoting antibiotics, and (outside of the EU) anabolic hormones in meat production are routine. Contamination of the meat supply is commonplace. Given the levels of overcrowding, unhygienic rearing, transport and slaughter conditions, it is not difficult to see why. Intensive animal production may yield high profits but only by imposing some pretty vicious costs.

Compassion in World Farming Trust has looked at other countries which have not pursued the deregulation path through food legislation with the uncritical zeal shown by our last administration and found that better welfare standards and improved hygiene can be part of a modern food economy. 1996, truly the 'annus horribilis' for UK food and farming (not only the BSE crisis, but also the world's worst outbreak of *E. coli* 0157) may have taught us some lessons. Within days of being elected, the new Labour government published plans for the setting up of the Food Safety Agency. In doing so, it read both the mood of the people and the gravity of the situation. This report must be required reading for all members of the new UK Food Safety Agency. But the report rightly looks further than these shores, towards Europe and beyond. Increasing consumer expectations in the global market will mean that better standards are not only a moral expectation but an economic necessity.

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Introduction

On March 20th 1996, speaking about victims of the new strain of Creutzveldt-Jacob Disease (CJD - the human equivalent of mad cow disease, BSE), the then UK Health Minister the Rt Hon Stephen Dorrell MP admitted: "the most likely explanation at present is that those cases are linked to exposure to BSE" (1).

It has been estimated that around half a million cows infected with BSE were eaten in the UK, before the ban on inclusion in food of the most infected tissues (brain, spinal cord, etc.) was introduced in 1989 (2).

The terrible consequences which might ensue from the apparently innocuous, but wholly unnatural, practice of feeding dead cows to live ones has brought home to everyone the impact that 'industrial' animal husbandry - animals viewed simply as production machines - might have on human health.

And one has just to scratch the surface of life down on the factory farm, to see that BSE might only be the tip of the iceberg. In a whole range of areas, from feeding practices, to animal housing, to the use of unnatural drugs in the pursuit of ever-greater productivity, human health may be being compromised by factory farming.

Imposing industrial demands on farm animals may, quite literally, be producing fatal flaws in the end product - our food.

Although Compassion in World Farming Trust's main area of concern is animal welfare, we believe that there is a strong connection between the suffering of animals kept in intensive, factory farm conditions and possible risks to human health in the meat produced from these animals.

This report investigates the links between animal welfare abuses on factory farms and the human health implications for the food produced from intensively reared animals.

It looks at the food poisoning bacteria present in the products of the factory farm; the implications of routine administration of antibiotics to intensively reared animals; the residues that have been discovered in our meat; the human and animal welfare catastrophe that might await us if we allow anabolic hormones to be administered to our food animals; and finally, at the ways in which regulations could be tightened so that farm animals and consumers may both get a better deal.

The bugs in the system

Consider the conditions which prevail on factory farms - for example, in intensive broiler chicken sheds: animals crowded together so much that the floor is scarcely visible, and where it is visible, it can be seen to be covered with excrement. An atmosphere full of dust and airborne bacteria. Scarcely any sunlight.

Would we be surprised if disease was rampant? Of course not. And yet these are the conditions in which many of our food-animals are reared.

Why, then, do we express surprise that much of the food we obtain from these animals harbours disease-causing organisms?

Salmonella

Infection is widespread

The UK Government's Advisory Committee on the Microbiological Safety of Food has stated that: "Raw poultry meat is a significant carrier of pathogens, particularly *Salmonella* and *Campylobacter*" (3). Their 1996 Report on Poultry Meat found that one in three chilled, raw UK-produced whole chickens on retail sale was contaminated with *Salmonella*; an even greater proportion (41%) of frozen birds was contaminated (4).

The type of *Salmonella* which most commonly infects chickens is *Salmonella enteritidis*, but there are several thousand other strains, in all types of farm animals.

Between 1990 and 1995, in Wales alone, *Salmonella* infection was reported in pigs, sheep, cattle and calves (5).

And, as one might expect, the environment in which farm animals are confined also appears to be significantly contaminated. A survey of litter and dust samples from commercial turkey flocks in Canada, for example, found *Salmonella* at 86% of flocks (6).

In England and Wales, between 1981 and 1987, there was a 13-fold increase in the rate of isolation of *Salmonella enteritidis* from layer chickens (7), and in 1991, a report in Which? magazine claimed that up to 7,000 of the 30 million eggs eaten in Britain each day carry the *Salmonella* bacterium (8).

Although *Salmonella*-infected animals can sometimes show few, if any, symptoms, this is by no means always the case, and many animals suffer as a result of *Salmonella* infection, just as humans do. A report in The Veterinary Record in 1995 described a batch of layer replacement chicks, bought in at a day old. "They were soon seen to be huddled together with drooping wings", and "by six days old 70 of 100 had died". The chicks were found to be infected with *Salmonella pullorum* (9).

The factory farm - a haven for disease

So, how is it that *Salmonella* infection of farm animals is so common? How is the infectivity spread? The overcrowded and unhygienic conditions of the factory farm seem to be the key.

Airborne infection

According to scientists at the Central Veterinary Laboratory: "Bacterial infections can be spread by the airborne route in farm animals, particularly when reared intensively. For example, poor ventilation in poultry houses can cause high concentrations of ammonia to develop and irritate the respiratory tract, predisposing to infection" (10).

Airborne infection of chicks, calves and laying hens with *Salmonella* bacteria has been reported (11,12,13). In the case of the laying hens, within two days of exposure the hens had a generalised infection; lungs, liver, spleen, kidneys, ovary and oviduct were all infected (13).

Faecal contamination

In the case of the airborne-infected laying hens just described, the infected hens excreted *Salmonella* bacteria for up to 28 days. Clearly, with laying hens so closely confined in battery cages, the possibility of cross-contamination is very high.

The fundamentally unhygienic conditions of intensive broiler chicken production have been described by researchers into the widespread contamination of poultry by *Salmonella*: "Broilers are reared in confined housing and eat, sleep and defecate on the floor or litter inside the house ... During transportation to the processing plant, some broilers may defecate inside the transport containers ... When they are processed .. there is evidence that all the broilers that were inside the transport container will have some fecal material on their feet and breast feathers. The fecal material on the breast also comes from the broilers' practice of resting on their breasts on the floor of the broiler house" [selective breeding of broiler chickens has produced many birds whose legs are so weakened that they are unable to support the weight of their bodies, and therefore can spend much of their time squatting on the floor] (14).

In fact, *Salmonella* contamination of poultry houses can be so severe that pressure-washing or steam cleaning of the house between the rearing of separate batches of chickens can lead to an **increase** in levels of bacteria (15).

And *Salmonella* infectivity from the factory farm can also be spread to animals which are being reared in less intensive conditions. A report in The Veterinary Record in 1995 described an incident in which slurry from a piggery, where animals were excreting large numbers of *Salmonella typhimurium* DT104, appears to have spread infectivity to cattle on nearby farms. The cattle suffered dysentery, and some animals died from the infection (16).

There is even evidence that *Salmonella* infectivity has become so widespread in poultry and humans that the circle is complete, and poultry flocks are being infected by faecal contamination from humans. Scientists in the United States have found strong evidence linking an outbreak of *Salmonella enteritidis* in a poultry flock in southern California with infection from a municipal sewage treatment plant located less than a mile away (17).

Contaminated feed

Another way in which *Salmonella* infection can be introduced and spread is via contaminated feed.

The Advisory Committee on the Microbiological Safety of Food has stated: "Animal feed is an important potential vector for the transmission of microbiological infection to poultry flocks"

(18), and: "We regard animal feed as an important potential vehicle for the transmission of *Salmonella* infection" (19). They also note that "we firmly believe that it is technologically possible and economically feasible for the feed industry to produce *Salmonella*-free poultry feed" (19).

Research in Holland found that 10% of the poultry feed examined was contaminated with Salmonella. Pelleted feed showed lower levels of contamination than mash feeds, and yet the feed given to those breeder-birds from which egg laying hens are produced (layer-breeders) is not usually pelleted, in order to maintain a low level of feed intake by the birds. Even with pelleted feeds, most of those feed mills surveyed used a pelleting temperature of 70-75C, whereas it has been established that the temperature must exceed 80C in order to reduce *Salmonella* levels below the limit of detection (20).

And contaminated feed may produce elevated levels of *Salmonella* in farm animals even though *Salmonella* is not the contaminating agent. In 1994, an outbreak of *Salmonella choleraesuis* was reported in pigs fed material contaminated with aflatoxin and vomitoxin (poisons present in feed contaminated with certain fungi), which seemed to compromise the immune and metabolic systems of the pigs, predisposing them to *Salmonella* (21).

So a picture begins to emerge of practices adopted to pare production costs to the bone: restricted feeding regimes, feed manufacturing processes where the cost of production is the overriding concern, and quality and compassion are secondary. And the recycling of dead animals as feed for live ones of the same species is surely the most extreme example of this 'industrial' approach:

In 1979, **ahead** of the BSE crisis, the Royal Commission on Environmental Pollution warned against the dangers of feeding dead farm animals to live ones, highlighting the possibility of recycling disease-causing agents (22). And yet, even now, nearly 20 years after this warning from the Royal Commission and 10 years after the farming industry became aware that the BSE crisis was triggered by feeding dead animals back to live ones, we continue to make cannibals of farm animals: poultry can still be fed with hydrolysed feather meal, and the 'off-cuts' and waste blood from poultry abattoirs.

International action against *Salmonella*

A spokesman for the British Chicken Information Service has been reported as saying that "*Salmonella* is difficult to eradicate in live birds" (23). But countries outside of the UK are taking action against *Salmonella*, and it seems to work:

In Holland, at the end of 1996, the Ministry of Agriculture threatened to **ban** the sale of contaminated poultry meat if the industry could not deliver low-*Salmonella* chicken onto the market within 30 months (24). Farms will have to implement special procedures and facilities aimed at reducing *Salmonella* levels, and will be strictly monitored with tests for *Salmonella* every nine weeks. At slaughter, any birds contaminated with faecal *Salmonella* will have to be kept apart from other poultry, to avoid cross-contamination.

In the United States, the egg industry itself now funds *Salmonella* control programmes. United Egg Producers' President Albert Pope said: "The egg industry will now take over the burden of payment so we won't have to continue to ask tax-payers to foot the bill" (25).

In Japan, the incidence of *Salmonella* food poisoning fell by 39% in 1993 (26).

But it is in Sweden that the most dramatic changes have been seen, where animal husbandry has been at the centre of the improvements. Swedish poultry is now practically *Salmonella*-free. Coupled with the virtual eradication of *Salmonella*, animal welfare standards have been improved, and routine administration of antibiotics is no longer practised.

Flocks are graded, at processing time, depending on key welfare indicators such as breast-blisters, leg problems, and hock burns. If significant numbers of these problems are detected, then the producer must adopt reduced stocking densities.

The Managing Director of the Swedish Poultry Meat Association, Camilla Littorin, has said that Sweden's standards of production and animal care will **help** it to compete in Europe, and may encourage other member states to adopt similar methods of 'disease-free' production: "Consumers need to feel confident that poultry meat comes from healthy flocks" (27).

In the UK, in 1993, the Government ceased to require the monitoring of commercial egg-laying flocks for *Salmonella* (28).

In 1995, several of the UK Ministry of Agriculture's voluntary codes of practice for the control of *Salmonella* were revised and published in booklet form. These voluntary codes of practice give advice to farmers on measures to control *Salmonella* infection in their flocks, and were described by the industry at the time as "a valuable tool with which to demonstrate to consumers at large the positive safety benefits of British food" (29).

Campylobacter

According to the UK Government's Advisory Committee on the Microbiological Safety of Food, *Campylobacter* is the most common cause of infectious intestinal disease in humans in England and Wales (30) - more common even than *Salmonella*. Symptoms typically include nausea, headache, backache, fever, abdominal pain and diarrhoea. In some cases, illness can be severe and prolonged, leading to arthritis and even neurological complications: since the eradication of polio, Guillain-Barré Syndrome is the most feared cause of paralysis in the Western world, and its most common cause is *Campylobacter* infection.

The extent of contamination of poultry with *Campylobacter* is very serious.

Campylobacter has been detected in 48% of fresh chickens in the UK (31) and in 11 out of 12 turkeys following processing and before sale (32).

In Northern Ireland, a study found *Campylobacter* in 94% of fresh chickens examined (33). Another study in Northern Ireland found *Campylobacter* in 64% of chicken wings on retail sale (34).

The organism has been found to survive on chickens which have been frozen for three months (35).

Given the prevalence of *Campylobacter* in chicken carcases, it is not surprising that a survey by the Central Veterinary Laboratory found that nearly half of those broiler flocks examined were colonised with *Campylobacter* (36).

Unlike *Salmonella*, animal feedstuffs are not thought to be a significant source of *Campylobacter* infection. Instead, the environment of the intensive broiler house is believed to represent the

most significant reservoir of infection (37). The Advisory Committee on the Microbiological Safety of Food, in its 1996 Report on Poultry Meat, said: "We recommend that industry gives attention to the improvement of the broiler farm environment at the earliest opportunity" (38).

And poultry are not the only potential source of *Campylobacter*. In Holland, 85% of pigs sampled were found to be infected with *Campylobacter* (39).

A report in The Veterinary Record in 1996 noted the spread of resistance to the antibiotic erythromycin in *Campylobacter*, and suggested that "the spread of these resistance traits may be due to genetic exchange of material between strains among animals under intensive production techniques. These conditions could vastly increase the occurrence of very rare genetic events". The authors went on to note: "Modern production methods and slaughter of young susceptible animals have created a heterogeneous population of *Campylobacter* species in pigs, which may allow the transfer of genetic resistance factors ... to human pathogens" (40).

Escherichia coli (E. coli)

The possible emergence in farm animals of new, especially dangerous strains of bacteria has also been raised in connection with the recent outbreaks of *E. coli* O157 in Scotland, which killed one in twenty of the people infected (41).

The bacterium *Escherichia coli (E. coli)* is a normal inhabitant of the gastro-intestinal tract of man and animals. It colonises the newborn's colon within hours of birth, and serves important intestinal physiological functions for the remainder of the host's life.

However, there is a sub-set of strains of *E. coli* which can cause a wide variety of diseases; approximately one-third of mastitis cases in cattle, for example, are caused by *E. coli* (35% of the UK's cows contract mastitis every year - a painful disease of the udder) (42). Recently, most attention has focused on those strains which produce toxins harmful to cultured Vero cells (African green monkey kidney cells) - Verocytotoxin-producing *E. coli*, or VTEC.

E. coli O157 is one such strain which frequently (but not always) produces verocytotoxins. It is thought to have acquired the ability to produce these toxins by a process of genetic exchange with another bacterium.

The strain has received close attention because, in 1983, it was found that in humans VTEC infection can lead to haemolytic uraemic syndrome (HUS), a form of kidney failure. This can be fatal in up to 10% of cases, and those who recover may have serious long-term impairment of kidney function. Even if HUS does not develop, *E. coli* O157 commonly results in severe abdominal cramps, bloody diarrhoea, and vomiting may also occur. Symptoms can last for up to two weeks.

The infectious dose appears to be very small - illness may occur after ingestion of less than 100 of these microscopic organisms (43). No specific treatment exists, and the effectiveness of antibiotics remains unclear.

E. coli O157 survives well in frozen storage. The bacterium can be destroyed by cooking.

Outbreaks of VTEC infection in England, Scotland and Wales have shown a steady increase in recent years. There was one outbreak in 1987 affecting 26 people. By 1994, there were 6 outbreaks, affecting a total of more than 130 people. Recently, more than 20 people have died,

and over 400 others have been affected in two separate *E. coli* outbreaks in Lanarkshire and Arbroath.

E. coli outbreaks in humans have been linked to a variety of foods - turkey roll sandwiches, minced beef products, even milk. In January 1993, an outbreak occurred across four states in the USA affecting 732 people, four of whom died. One child excreted the organism for 72 days. The outbreak was linked to hamburgers consumed at multiple outlets of a single restaurant chain. The US Department of Agriculture report of the outbreak states that "insanitary slaughter and dressing procedures led to the contamination".

Cattle appear to be a reservoir of *E. coli* O157, but this strain does not seem to be associated with disease in the host animal. Studies in adult cattle have shown that a high proportion have antibodies to verocytotoxins (up to 78% in some studies), consistent with frequent carriage of VTEC by cattle.

A US National Animal Health Monitoring System study found *E. coli* O157 in faecal samples from 63% of cattle feedlots examined (44).

As well as being present in the intestinal tract of cattle, *E. coli* O157 can also be present in the udder (45), which may explain why people have become ill following the consumption of milk, or milk products.

VTEC strains of *E. coli* other than O157 are known to cause cattle illness, particularly in calves. Oedema disease in pigs (which can result in convulsions, paralysis and sudden death) is also associated with verocytotoxin-producing E. coli.

E. coli O157 has also been found in UK sheep (46). And colisepticaemia in poultry, caused by *E. coli*, has increased with the development of intensive broiler production (47).

Cattle manure can be an important vehicle for the spread of *E. coli* O157. There is a case of a lacto-ovo-vegetarian in the US who contracted *E. coli* O157: her diet consisted of vegetables from her own garden, fertilised with cow manure. Typically, one gram of faecal material may contain a billion *E. coli* cells.

According to the Advisory Committee on the Microbiological Safety of Food "carcases contaminated during the slaughtering and dressing process represent an important route by which pathogenic organisms such as *E. coli* O157 can enter the food chain. The state of cleanliness of the animals and the skill and care exercised in the slaughter ... play a significant part in the initial transfer of organisms from the hide and gastro-intestinal contents to the carcase surface ... Thereafter, various factors such as rate of throughput ... will contribute to the spread of contamination" (48).

This point was re-iterated by Professor Hugh Pennington, in his report into the outbreak of *E. coli* poisoning in Scotland which began at the end of 1996:

"It is vital that farmers are made fully aware of their responsibility to send animals to slaughter in a clean condition" (49).

Clearly, the transport of animals to slaughter over long distances, and in cramped conditions, will make this very difficult, if not impossible.

Professor Pennington went on to say: "Notwithstanding commercial considerations and the implications of, for example, piece rates of payment for workers, the speed of the production process within abattoirs needs to be controlled so as to permit the achievement of adequate food safety standards" (50).

So, abattoirs dealing with very large numbers of animals, which are contaminated externally with manure, and whose gastro-intestinal contents are dispersed during slaughter can be expected to offer a route for *E. coli* to enter the human food chain.

But as well as highlighting the possibility for the spread of *E. coli* among dirty carcases Professor Pennington, noting the possibility of a 20% rise in E. coli O157 cases in Scotland this year, has said that it is now logical to assume "that there was something happening out there in the animals" (51).

As noted earlier, evidence is emerging of surprisingly high rates of mutation in those food poisoning organisms found in large numbers in our farm animals.

A study reported in the journal Science in November 1996 found "alarmingly high" incidences of mutation in E. coli O157 and Salmonella. The study also found that these bacteria seemed to be able to mutate easily by horizontal transmission of genes. It was suggested that this could confer antibiotic resistance, from a reservoir of pathogenic bacteria in the environment, allowing the bacteria "to escape immune surveillance or elude therapeutic intervention" (52).

Obviously, with the emergence of such highly mutable food poisoning bacteria, the confinement of farm animals in crowded and unhygienic conditions is simply asking for trouble.

Listeria

Between 1987 and 1989, 26 babies in the UK died from listeriosis. In 1989, the UK Government issued a warning to vulnerable groups, such as pregnant women, to avoid certain high-risk foods, such as soft cheeses and meat patés. Listeria can cause miscarriages, still births, and serious illness in newborn babies. Listeria continues to be associated with farm animals, and a wide range of food products derived from these animals. Listeria monocytogenes has been found to contaminate up to 66% of fresh and frozen chicken products (53). Other research has found the body surfaces of pigs to be contaminated by Listeria in 58% of cases (54).

Listeria also seems to be a significant contaminant of abattoirs. One Dutch study found Listeria in 100% of environmental samples taken from the conveyor in an abattoir (55).

Sheep are also known to harbour Listeria, where it causes increases in abortions, and nervous disease.

Despite warnings in the 1980s, Listeria continues to be a significant health-hazard. In May 1995, Brie cheese was the source of 17 cases of listeriosis in France. Nine pregnant women were infected, two of whom had miscarriages, and another two of whom had stillborn babies (56).

A contributory factor to the dangers of Listeria may be the peculiar ways in which the bacteria respond to storage and cooking. Unusually, vacuum packing seems to enhance recovery of the bacteria, compared with cells packaged in air (57). Exposure to low temperatures appears to enhance the potential for the organism to cause disease, and the bacterium replicates at a faster rate, when moved from chill (4C) to warm (37C) temperatures, than if it is kept at 37C for the

whole time (58). In addition, Listeria has been found to be able to survive microwave cooking (59).

According to the Oxford Concise Veterinary Dictionary: "The organism is widespread in nature and very resistant to physical and chemical agents. Infected materials, such as faeces and silage, can harbour the organism for many years" (60). In view of the fact that Listeria monocytogenes appears to be so widespread among intensively reared poultry, the practice of spreading untreated litter from broiler chicken sheds (often containing dead birds, many of which may be contaminated) onto fields seems highly unwise.

Poison food

With so many bugs associated with our food-producing animals, is it any surprise that, increasingly in the UK, we are being poisoned by the meat (and other animal products) that we eat? Survey after survey has found unacceptably high levels of food-poisoning organisms in our meat, and a relentless increase in reported cases of food poisoning.

Much of our meat is infected

As noted earlier, the 1996 Report on Poultry Meat by the Advisory Committee on the Microbiological Safety of Food found that one in three chilled, raw UK-produced whole chickens on retail sale was contaminated with Salmonella, and even more frozen birds (41%) were contaminated (4).

Which? magazine surveys found Salmonella in 36% of chickens in 1994, and in 20% of chickens in 1996. Campylobacter was found in 41% of chickens in 1994, and in 37% of chickens in 1996 (61).

In other research, Campylobacter has been isolated from 48% of fresh chickens examined in the UK (31), and in 94% of fresh chickens examined in Northern Ireland (33).

Campylobacter has also been found in 11 out of 12 turkeys, following processing and before point- of-sale (32).

Verocytotoxin-producing strains of E. coli have been found in 22% of raw beefburgers (62) and in 25% of raw pork sausage (63) obtained from retail outlets in north and north-west London.

Even for milk and milk-products, the levels of bacterial contamination considered 'acceptable' are surprisingly high. For ground-water drinking water, a typically acceptable level of contamination might be 10-20 bacterial cells per ml (64). But for raw cows' milk which is to be used for the manufacture of raw milk-based products whose manufacture does not involve any heat treatment, up to 100,000 bacterial cells per ml is considered an 'acceptable' level of contamination (65).

And just as the levels of food-poisoning organisms in our meat are unacceptably high, so too are the (increasing) levels of food poisoning itself.

Food poisoning is increasing relentlessly

According to figures from the Communicable Disease Surveillance Centre, laboratory reports of faecal isolates of Campylobacter in England and Wales increased from 28,761 in 1988 to 44,414

in 1994. For Salmonella, the increase was from 27,478 in 1988 to 30,411 in 1994. This represents a total increase, for the two pathogens, of 33% in six years.

Of course, not all food poisoning cases are reported. Estimates suggest that somewhere around 1 in 30 to 1 in 40 cases are reported (66, 67). This would mean that, in England and Wales in 1994, there were 1.5 - 3 million cases of food poisoning from Salmonella and Campylobacter alone -- one for every 17-34 people in the population.

One expert in the Public Health Laboratory Service has estimated that the average cost in lost production of a single Salmonella case, in 1988, was £413, and the public sector cost was £298, for investigation and treatment (67) -- a total of £711. At 1994 prices this was equivalent to £941. With between 1.5 and 3 million cases of Salmonella and Campylobacter food poisoning in England and Wales in 1994, food poisoning in England and Wales alone can reasonably be predicted now to be costing industry and the taxpayer well over £1 billion, and perhaps nearer £3 billion every year.

And there are, on average, four deaths for every 1000 reported cases of Salmonella infection in humans in England and Wales (66).

The situation in Scotland likewise shows an accelerating incidence of food poisoning. A report in The Guardian newspaper on December 31st 1996 quoted Scottish Office figures which showed that confirmed notifications of food poisoning rose from 858 in 1970 to 6,510 in 1990. By November 1st 1996 (that is, prior to the outbreaks of E. coli O157), Scotland had recorded 8,408 cases of food poisoning in 1996, compared with 7,575 for the comparable period in 1995 (68).

Most recently, a report in The Financial Times on March 5th 1997 noted that, according to the Office of National Statistics, food poisoning in England and Wales has increased six-fold over the past 15 years (69). One of the reasons given for this huge increase was: "more intensive rearing of chickens and farm animals under conditions that can spread germs".

Whichever way one looks at it, the current high levels of food poisoning in the UK are completely unacceptable.

Bad animal welfare produces bad food

Attempts are often made to dismiss the widespread contamination of our food-producing animals with disease-causing organisms, by saying that these toxic agents can easily be destroyed by cooking. However, the massive rise in food poisoning in recent years highlights the inadequacy of this approach (especially when, for example, a recent survey found that three-quarters of the UK's brands of frozen burgers fail to carry the Chief Medical Officer's recommended cooking advice on their labels (70)). In the face of the rapid increase in food poisoning, and with the loss of life involved, it is not good enough just to accept implicitly that our food is contaminated.

And there is plenty of research which clearly points the finger of responsibility away from the consumer, and back to the point of production.

Scientists at the University of East Anglia found that when the average monthly temperature exceeded 7C, the number of food poisoning cases increased -- a 7% increase in food poisoning for each degree rise in temperature. However, food poisoning did not increase during the rise in temperature (as one might have expected if contamination occurred close to the time of consumption), but a month later (suggesting that contamination occurred about a month prior to

consumption) (71). As one of the researchers pointed out: "This shifts the emphasis of responsibility for food poisoning away from the consumer and on to the producer" (72). Another of the researchers said that further investigations had shown the infections began on the farms, usually those that employed intensive methods (73).

Other researchers have noted that because poultry have a habit of pecking the ground, pathogens shed in faeces and present in litter will be ingested (74). Also, feet become contaminated from faeces, and skin and feathers can readily become contaminated in the dust-laden environment (75). A study of Salmonella contamination in poultry houses in France found samples of the pathogen on walls, drinkers, feeders, floor, insects, in water, in feed, and of course on the chickens themselves (76). Similarly, Campylobacter was found in the air, litter and drinking water containers (77, 78). It was stated that:

"Intensive rearing of fowl where thousands of birds are kept together is conducive to the spread of Salmonellae, and likely, other pathogens (79). Under such conditions, one infected bird, a contaminated lot of feed or other sources of contamination in the environment can easily spread pathogens to many birds" (74).

Further evidence for the on-farm origin of food-poisoning organisms comes from a large study conducted in England in 1985 and 1986. This research identified similar strains of Campylobacter, from a broiler farm, through a processing plant to wholesaler, catering college and retail outlet to consumers. About 31% of Campylobacter isolates from patients were the same strain as 91% of the chicken isolates from the affected farm. Elimination of infection at the farm was reflected in a major reduction in prevalence of the Campylobacter strain in the local population (78, 80, 81).

Furthermore, a higher incidence of Campylobacter infection occurs in people who have occupational exposure to poultry and cattle than in others in farming communities (82), and employment in poultry processing may predispose workers to campylobacteriosis (83). A serological survey has found that 27-68% of personnel in poultry and red meat plants have antibodies to Campylobacter jejuni, compared to 3% of arable workers. And a higher incidence of campylobacteriosis was observed among holiday workers (71%) than in permanent staff (29%) during an outbreak among Swedish poultry abattoir workers (84). The fact that people who work in abattoirs have increased levels of antibodies, and apparently an increased resistance to campylobacteriosis, suggests that the carcases being processed are significantly contaminated by the time they arrive at the abattoir.

Work by the Food Microbiology Research Division of the Department of Agriculture for Northern Ireland in 1994 found that: "The manner in which animals are housed will directly affect the microflora of their exterior, whilst the age at slaughter will have a major effect on the microflora of the GI [gastro-intestinal] tract ... Those animals which are intensively reared and slaughtered young will have the greatest potential for carrying pathogens" (85) (broiler chickens are slaughtered at six weeks of age). As an example, the Department of Agriculture compared the microbial count of hides from cattle in summer (cattle slaughtered in summer will have spent some months in open fields) with hides from cattle in winter (which will have spent months indoors in pens), and found that the winter hides showed around 100,000 times more microbial contamination than the summer hides.

The same author considered the effect of long journeys to abattoirs, and noted: "The travel can be stressful ... Further stress can be applied when animals with a social hierarchy, e.g. cattle, are mixed. These animals will then attempt to reassert their social position and the consequent

fighting causes stress which can increase susceptibility to GI [gastro-intestinal] infections ... Whilst the time period required for full-scale infection will not be available, the animals will be able to contaminate their exterior surfaces. This is especially so where tiered transport systems are used, e.g. the 'double decker' carriers for cattle and 'triple-decker' carriers for sheep."

All of this strongly bears out comments by David Statham, Chairman of the Chartered Institute of Environmental Health's (CIEH) Food and General Health Committee, at the CIEH's Annual Congress in 1995, when he said: "Far too many of our meat animals are infected with the organisms that cause food-borne disease". He also said that a concerted effort is needed at farm level to attempt to eradicate the infective organisms from food animals: "This requires careful control of feedstuffs, and good animal husbandry to attempt to eliminate infection at source". He added: "Further controls must not stop at the farm gate. Strict controls must be implemented to prevent animals being crammed together in dirty, overcrowded conditions during transportation. This is more than just a hygiene problem. It is an important animal welfare issue, too" (86, 87, 88).

And, in 1995, at the launch of the Food and Drink Federation's National Food Safety Week, the then Junior Agriculture Minister Angela Browning said: "The need for hygiene practice in the food chain starts with the producer, and they need to pay attention to good practice and need to recognise that they are an integral part in the chain" (89). Farm animals would be infected with fewer disease-causing organisms if, for example, they were reared in less overcrowded, more open environments -- where broiler chickens, for instance, were not pressed up one against the other, where they were not forced to trample on the dead bodies of their companions, where they had access to direct sunlight, and were not condemned to spend their brief lives blistering their feet by standing in their own excrement, and blistering their lungs by breathing an atmosphere heavy with ammonia fumes, dust and bacteria.

Antibiotics -- saviours or suicide?

With disease-causing organisms so prevalent on our factory farms, it is not surprising that farmers have resorted to the widespread use of antibiotics. The routine use of antibiotics is particularly attractive to farmers as many of these substances have an as-yet poorly understood ability to promote the growth-rate of the animals to which they are administered.

So farmers, in a desperate battle to contain the bug-explosion on factory farms, and in order to maximise profits by pushing growth-rates ever faster and faster, are routinely dosing our food-animals with a whole range of different types of antibiotic.

And it's not just factory-farmed pigs and poultry which are dosed with antibiotics. In a Parliamentary answer given in December 1996, the then Junior Agriculture Minister Angela Browning revealed that no less than 62 different antibiotics and other antimicrobial substances were, at that time, licensed for use in the feed and water of dairy cows and other lactating animals (90).

It is important to understand the scale of the use of antibiotics in animal feed. According to an industry report published in 1995, 90% of feed given to broiler chickens worldwide is supplemented with antibacterial additives, and 60% of feed that is given to pigs (91).

Antibiotics are active in very small amounts. Yet, world-wide, 2700 tonnes of antibacterials were incorporated into broiler chicken feed alone; 5400 tonnes were added to pig feed (91). For

substances which are biologically active in very small amounts, the quantity of material being added to farm animals' feed is vast.

And the discontinued use of one specific antibiotic is no guarantee that the total amount or numbers of antibiotics used will be reduced -- take the case of chloramphenicol, for example. This substance was banned from use in food-producing animals in the United States over ten years ago. Chloramphenicol is the only effective treatment in humans against typhoid. But research suggested that its over-use in animal husbandry had led to increased resistance to this antibiotic in the typhoid bacterium (a member of the Salmonella family). Also, Dr Lester Crawford, director of veterinary medicines for the US Food and Drug Administration warned, in 1985, that the slightest residue of the antibiotic in food, such as meat, could cause life-threatening diseases in susceptible people: "This is because chloramphenicol can cause fatal aplastic anaemia and/or leukaemia in susceptible humans at any dose by any rate of administration" (92). Chloramphenicol was eventually banned from use in food-producing animals in the EU in 1994. But it has been swiftly replaced by at least three other products: florfenicol, ceftiofur and cefquinome (93).

Antibiotic resistance

The example given above, of chloramphenicol, illustrates one of the greatest potential dangers for human health which may result from the over-use of antibiotics in factory farming.

Antibiotics are designed to kill bacteria. But not all bacteria are the same, and they also change, or mutate, over time. Some bacteria are (or become) resistant to particular antibiotics. So eventually, as an antibiotic is used more and more, the population of bacteria which is susceptible to an antibiotic will gradually be replaced by a population of bacteria which the antibiotic cannot kill.

This is why the use of antibiotics in human medicine is so strictly limited. And this was one of the reasons for the discontinued use of chloramphenicol in animal husbandry -- it was thought that its use on farms might be compromising its effectiveness in the treatment of typhoid in humans.

The principal potential human health problem with the huge use of antibiotics in farming stems from the similarities between different kinds of antibiotics. Different kinds of antibiotics can work in very similar ways, so bacteria which develop a resistance to one particular antibiotic may co-incidentally become resistant to other antibiotics -- this is known as multi-resistance (so the restriction in the UK, that no antibiotic which is used to treat infection in humans can be used as a growth promoter in animals may be of limited benefit).

In addition, it is thought that bacteria may be able to pass antibiotic resistance between each other ('transferable drug resistance').

The result is that, by over-use of a particular antibiotic in one area (say, broiler chicken farming), bacteria may proliferate which are resistant to antibiotics used in an entirely different area (for example, human medicine).

The World Health Organisation, in its 1996 report, 'Fighting Disease, Fostering Development', made specific mention of the potential problems for human health of the emergence of antibiotic-resistant bacteria:

"More than half of the total production of antimicrobials worldwide is currently used in farm animals, with a large proportion of antibiotics being administered in subtherapeutic doses, not to treat disease but to promote growth ..... As a result, two important human pathogens of animal origin, E. coli and Salmonella, are today highly resistant to antibiotics in both industrialized and developing countries. For instance, in the United Kingdom, the increase in multi-resistant strains of S. typhimurium isolated from cattle is paralleled by increasing resistance among strains of human origin. In Thailand, Salmonellae isolated from food animals are also highly resistant to the common antibiotics. These bacteria cause diarrhoeal disease and can lead to life-threatening complications. Due to the globalization of food supply and international travel, antimicrobial resistance among animal bacteria can affect consumers anywhere in the world" (94).

And these concerns are not new -- it is almost 30 years since the Swann Report to the UK Government on the use of antibiotics in animal husbandry warned, in 1969:

" ... it is certain that the use of an antibiotic in animal feed produces large numbers of resistant organisms, including organisms with transferable resistance, and that these resistant organisms may be transmitted to man" (95), and:

" ... this resistance ... could conceivably be transferred either directly or indirectly to such highly dangerous organisms as the typhoid bacillus (Salmonella typhi). Such a chance meeting between resistant organisms and highly dangerous (pathogenic) ones could give rise to a potentially explosive situation" (96).

Remember that, on page 11, a study reported in the journal Science in November 1996 was described which found "alarmingly high" incidences of mutation in E. coli O157 and Salmonella, and also that these bacteria seemed to be able to mutate easily by horizontal transmission of genes. It was suggested that this could confer antibiotic resistance, from a reservoir of pathogenic bacteria in the environment, allowing the bacteria "to escape immune surveillance or elude therapeutic intervention" (52).

Is this what Professor Pennington meant, when he said that it is now logical to assume "that there was something happening out there in the animals" (51)?

Certainly, there is ample evidence that bacteria present in farm animals are widely resistant to commonly used antibiotics. Research in Holland, reported in 1993, found that, in pigs, "the prevalence of resistance to the most commonly used antimicrobial agents in veterinary medicine was high ... despite the absence of mass medication during the observation period". Resistance to amoxicillin was found at levels up to 97%, and resistance to oxytetracycline, sulfamethoxazole and trimethroprim were all found at levels up to 100%. Up to 68% of isolated E. coli strains were multi-resistant to oxytetracyline, streptomycin and sulfamethoxazole. 43% of isolates were resistant to three or more antibiotics (97).

Dr Bernard Rowe, head of the Laboratory of Enteric Pathogens at the Central Public Health Laboratory, London, speaking at the European Congress of Chemotherapy in 1996, said: "The use, misuse or overuse of antibiotics in animal husbandry is creating a selective pressure which is encouraging the emergence and persistence of resistant, multi-resistant strains of Salmonella in food animals which then go into the food, causing epidemic spread not only on the farm but also eventually in us" (98).

Lord Hylton, in June 1996, asked the then UK Government: "What research has been done or is planned on the effects of including antibiotics in the food fed to animals, poultry and fish; and in

particular whether such practices could increase the number of organisms present in humans." Surprisingly, Lord Lucas, replying on behalf of the Government, answered: "No research has been conducted or is planned." (99).

The same month, the then Junior Agriculture Minister Angela Browning gave a more reassuring reply to a question from Elliot Morley MP about the routine use of antibiotics in farm animal feed; she said: " ... many antibiotics are effective against both human and animal disease and a number licensed in human medicine are also authorised for therapeutic use in animals. Their use in human medicine, however, far outweighs any animal use" (100).

But this reply is not nearly so reassuring as it seems. The threat to human health from the over-use of antibiotics in farming is less likely to come from antibiotics used selectively for therapeutic purposes, but much more likely to result from the huge amounts of antibiotics supplied routinely to farm animals to make them grow faster, specifically where the farming antibiotic closely resembles an antibiotic used in human medicine.

The controversy surrounding the farm animal growth promoter avoparcin, and the antibiotic used in human medicine vancomycin, offers an important example:

Avoparcin and vancomycin

The antibiotic avoparcin was first approved in the EU in 1976, as a growth promoter for chickens. Approval for its use was subsequently extended to turkeys, pigs, lambs and cattle. It has been used to increase milk yield in dairy cattle. In 1995, global sales of the product were estimated as $50 million (101).

Avoparcin is closely similar in chemical structure to an antibiotic called vancomycin, used in human medicine. Vancomycin is very important for humans -- it is a drug of 'last resort'. In the United States, for example, up to 12% of the bacteria which cause two million cases of pneumonia and 3,000 cases of meningitis each year are multi-drug resistant, and patients have to be given vancomycin. Professor Stuart Levy, Director for The Centre for Drug Resistance at Tufts University, Boston has said of these patients: "We are one antibiotic short of being unable to treat them" (102).

The controversy concerning these two antibiotics has arisen because of research suggesting that bacteria have emerged which are resistant to vancomycin, and that this resistance is the result of the over-use of avoparcin on farm animals.

To put the use of these two antibiotics into context, one should note that in Denmark alone, in 1993, 22kg of vancomycin was used in human medicine, and 19,000kg of avoparcin was administered to animals (103).

In 1994, research was published in the UK following isolation of vancomycin-resistant bacteria from an Oxford hospital, which found that pigs, a sewage inlet and human faecal material contained vancomycin-resistant bacteria which genetic fingerprinting showed to be identical. Similarly, uncooked chicken yielded resistant bacteria indistinguishable from sewage isolates. The authors concluded: "It is therefore interesting that in this study vancomycin-resistant E. faecium [Enterococcus faecium] were isolated from a selection of farm animals and the carcasses of uncooked chickens. Hence VRE [vancomycin-resistant enterococci] may be entering the community via the foodchain" (104).

In 1995, research was published which investigated the occurrence of vancomycin-resistant bacteria on farms in Germany. Vancomycin-resistant bacteria were found in pig and poultry farms which used avoparcin, but could not be found where avoparcin was not used. The authors concluded that farms which use avoparcin are "an important reservoir" for vancomycin-resistant bacteria. In addition, the same authors found vancomycin-resistant bacteria present in frozen broiler chickens from the kitchen of a hospital (105).

And in April 1997, a paper in the medical journal The Lancet described the case of a UK truck driver who sustained a broken femur while working at a factory packaging chickens. The wound became infected with vancomycin-resistant Enterococcus faecalis. Specimens taken from chicken carcases at the factory revealed the presence of VRE, with antibiotic resistance profiles very similar to the bacteria infecting the patient. The authors noted: " ... this case shows that animal use of glycopeptides may represent a risk to patients" (106).

In the same month that the case of the truck driver infected with VRE was reported in The Lancet, the European Commission introduced a ban on all use of avoparcin in animal feed in the European Union. This followed unilateral bans by Denmark in May 1995 and Germany in January 1996. The EU-wide ban was introduced as a precautionary protective measure because, according to the EC, "current information does not allow the risk [that resistance can develop to glycopeptide-based antibiotics used in human medicine] to be dismissed with any certainty" (107).

The announcement of a ban on avoparcin in the EU has since been followed by bans in Switzerland (108) and Japan. The Japanese ban follows tests by their Veterinary Assay Laboratory and Fertiliser and Feed Inspection Centre, which examined chicken faeces from farms where avoparcin has been used. The tests found bacteria which were resistant to avoparcin and also to vancomycin (109).

The evidence for a link between bacterial resistance to vancomycin as a result of the widespread use of avoparcin as a growth promoter in farm animals might, at present, be considered incomplete. Nevertheless, the consequences of widespread bacterial resistance to vancomycin for human medicine are so serious that the adoption by the EC of a precautionary protective ban is to be welcomed.

Compassion in World Farming Trust would urge that the breathing space offered by this ban is used to reconsider radically the use of antibiotics for growth promotion in farm animals. Today's farm animals are already pushed far beyond their natural growth capabilities by selective breeding so that, for example, most turkeys are physically incapable of mating, and pigs and broiler chickens suffer widespread cardiovascular and leg problems because their hearts and skeletons cannot provide for the demands of their over-rapid muscle development.

Routinely to dose these animals, already suffering from unsustainable growth rates, with antibiotics simply to push them further beyond their natural metabolic limits is literally adding insult to injury.

And there are other problems associated with the over-use of antibiotics in farming, in addition to the emergence of multi-resistant strains of bacteria.

Competitive exclusion

Even in a healthy animal, the gastro-intestinal tract contains a diverse variety of micro-organisms, which usually colonise the individual shortly after birth, which exist in a natural balance, and without which the animal's healthy development would be impaired.

The introduction of growth-promoting antibiotics into this delicate internal ecosystem can have unpredictable, and maybe unknown, consequences.

For example, the antibiotic avoparcin has been found to increase Salmonella excretion by turkeys and chickens. It also makes chickens more susceptible to Salmonella infection (110). Since avoparcin is not absorbed into the bodies of the animals to which it is administered, this unexpected effect of the antibiotic on Salmonella is presumed to occur by disturbing the balance of competitive exclusion -- by knocking out a naturally-occurring organism in the gastro-intestinal tract of poultry which normally holds Salmonella bacteria in check.

This explanation is supported by the observation that administration of a mixture containing the normal intestinal bacteria from a healthy adult hen will reduce Salmonella in newly hatched chicks.

Now, the potential for unpredictable and adverse effects of routine, non-therapeutic antibiotic administration to farm animals starts to become clear with the following piece of research:

The same mixture derived from the normal intestinal bacteria of a healthy adult hen, as well as reducing Salmonella in newly hatched chicks, has also been found to prevent colonisation of chicks by the human pathogenic E. coli O157 (111).

Summarising these findings:

administration of normal intestinal bacteria from healthy adult hens reduces Salmonella in newly hatched chicks;

administration of normal intestinal bacteria from healthy adult hens also prevents colonisation of chicks by E. coli O157;

administration of a growth-promoting antibiotic to poultry increases Salmonella excretion, it is thought by knocking out naturally occurring intestinal bacteria which hold Salmonella in check.

Is it possible that, by manipulating the natural internal micro-flora of our food animals with growth-promoting antibiotics, we are inadvertently risking an explosion of potentially pathogenic bacteria, such as E. coli O157? The evidence that we have so far suggests we should investigate this possibility very carefully.

Particularly since we already know that there are naturally-occurring micro-organisms, called bifidobacteria, which live in the large intestine and which produce a substance that kills E. coli O157 (112). Any adverse effect of antibiotics on bifidobacteria could clearly have serious consequences.

Antibiotic residues in meat

The development of antibiotic resistance and disturbances in the balance of competitive exclusion are not the only aspects of the widespread use of antibiotics in factory farming which could have implications for human health.

As noted earlier, in connection with chloramphenicol, the presence of some antibiotics as residues in meat may pose a risk to human health. Some people experience severe, even fatal, adverse reactions to antibiotics (presumably, the same is also true of the reaction of some farm animals to these substances).

And yet, certain drugs are detected again and again in farm animals in the UK in quantities which exceed the maximum permitted levels. According to a report in the journal International Food Hygiene in 1997, the sulphonamide antimicrobial, sulphadimidine, is the residue most frequently detected in meat (113). The same report notes that "sulphonamides in high doses can impair kidney function and a study in the USA has reported a link with thyroid cancer".

In the 1995/96 Annual Report of the UK Veterinary Medicines Directorate (VMD), the presence of sulphonamides above the permitted Maximum Residue Level (MRL) was noted in cattle kidney, chicken liver, eggs, and pig kidney (114). Interestingly, one of the cattle samples which contained sulphonamide residues above the permitted MRL contained sulphathiazole; the licence for use of this product in cattle was withdrawn several years ago (115).

A report in Farmers Weekly in December 1996 noted that an investigation by the VMD found that excessive drug residues in pigs at slaughter could result from, among other things: "increase of sulphonamide feed due to the rise in respiratory disease in intensively-reared pigs" (116). And research on breeding-pigs published in Sweden in 1994 found that: "Consumption of medicated feed was negatively correlated with good hygiene. Herds using deep straw bedding systems used three to four times less medicated feed than those with other weaning systems" (117).

The situation with sulphonamide residues is clearly considered by the VMD to be serious, since they produced a special leaflet for pig farmers in 1996 giving guidelines on the use of sulphonamide in pig feed. The leaflet reminds farmers that "It is an offence, under The Animals, Meat and Meat Products (Examination for Residues and Maximum Residue Limits) Regulations 1991 as amended, to sell, or supply for slaughter, any pig for human consumption in GB if it contains residues of medicines in excess of the Maximum Residue Limit" (118).

The possibility for inadvertent contamination with substances such as sulphonamides appears to be high, according to the VMD's leaflet. It notes that "sulphonamide is present in the urine and dung from pigs eating sulphonamide medicated food. Pigs will eat and drink other pig's urine and faeces" (up to 89% of a dose of sulphonamide is excreted in dung and urine within 10 days of administration), and also: "barrows used to move pig food should not be used to move pigs. The potential for contamination is high" (118).

Clearly, there is a real problem of contamination of pig carcases with sulphonamide residues. Every year between 1990 and 1996, sulphonamides above maximum permitted levels were detected in pig carcases in the UK (119-125). As yet, there have been no prosecutions (126).

Sulphonamide use may also be a problem for pigs. According to a report in The Farmers Guardian, medication of sow feeds, especially with sulphonamide-based products, can compromise natural biotin production. And a lack of biotin can lead to claw distortion and cracking, in breeding pigs (127).

So, once again, a 'technical fix' to circumvent the poor hygiene of intensive farming (the administration of antimicrobials to control respiratory disease in intensively farmed pigs) appears to have an unforeseen and adverse welfare impact on the factory-farmed animals.

The presence of antibiotic residues in meat, even if these antibiotics are believed to pose a threat if administered to humans, does not necessarily imply a health risk -- the residues will be exposed to cooking and the digestive process before the possibility of their being taken up internally by humans.

Unfortunately, there is evidence that certain antimicrobial residues offer resistance to the cooking process. An investigation into the stability of sulphamethazine (sulphadimidine) residues in cooked meat found that:

the drug is stable in boiling water at 100C;

in cooking oil, at 180C, half of the drug's activity is still present after two hours, and it takes a further two hours of cooking to deplete the activity of the drug to 25% of its original level;

migration from the contaminated tissue into the surrounding liquid or meat juices was observed during cooking;

during frozen storage, sulphamethazine residues were stable over a period of three months (128).

Some properties of sulphadimidine and its break-down products also raise concerns about the significance of measured residue levels, and behaviour of these substances during the digestive process.

It has been suggested that the break-down products of sulphadimidine can convert back to their parent compound under acidic conditions. Thus, although measured levels of the parent product sulphadimidine might be below maximum permitted levels, "since gastric conditions are highly acidic, a similar conversion could be expected in consumers during digestion, thereby exposing them to a quantity equivalent to the sum of the metabolite and parent drug" (113).

Antibiotic residues are not just a UK problem

Recent research into levels of antibiotic residues in meat has shown the problem to be widespread throughout the EU.

A report in Which? magazine in March 1997 gave details of research carried out with financial support from the EC, which showed that only four of the fifteen EU countries have meat which is free of antibiotic residues (Denmark, Sweden, Finland and Portugal). The Republic of Ireland recorded the most frequent contamination of any one category of meat: 17% of Irish pigmeat samples were contaminated with antibiotics (129). When the Irish results first became known, in October 1996, former chief executive officer of the Irish Veterinary Union Peter Dargan, commenting in The Irish Sunday Independent, said that the results of the tests suggest that part of the pigmeat sectors may be using antibiotics to cover up bad management (130). The Irish Farmers' Association described the levels of residue as "totally unacceptable"(131).

And a subsequent report in the Irish national newspaper The Examiner, in November 1996, revealed the existence of an Irish Department of Agriculture report which showed the situation to be even worse. Over 25% of bargain pork cuts examined were found to exceed permitted antibiotic residue levels; over 28% of pork chops surveyed contained excessive levels of antibiotic residues; and in some sample areas the amount of antibiotic residue ran as much as 45% above the permitted EU levels (132).

In Germany, the BgVV (the federal institute for consumer health and veterinary medicines) reported that 18% of calf units surveyed in 1995 showed positive readings for chloramphenicol in calves' blood or urine (133). Chloramphenicol was banned from use in food-producing animals in the EU in 1994.

And in Australia, in 1995, antimicrobials were the substances in meat most frequently found to be present at levels exceeding maximum permitted limits (134).

The reason most frequently given for the existence in meat of antibiotics at levels exceeding permitted maxima is a failure to adhere to recommended withdrawal times -- the minimum number of days before slaughter during which the antibiotic must not be administered to the animal.

In 1994, for instance, the main cause of residue violations in the US (43.4%) was failure to adhere to approved withdrawal times (135).

Indeed, Ireland's former farm minister Ivan Yates has suggested that his country's poor record on antibiotic residue contamination in pigmeat may be the result of farmers' efforts to respond to high pig prices by rushing stock to finish earlier (136).

In an industry where prices are volatile, and where only a small minority of carcases are tested for residues, there will always be a strong temptation for farmers to depart from approved withdrawal times. We shall return to this problem in the section of this report about anabolic hormones, in the context of moves to lift the current EU ban on these substances.

Compassion in World Farming Trust believes that the over-reliance of factory farming on the widespread use of antibiotics poses a potentially serious threat to human health, and is pushing farm animals beyond their limits, so that they are suffering as a result of their unbalanced metabolisms, and their unnaturally accelerated growth rates.

Compassion in World Farming Trust considers it wrong that substances which are thought to be directly threatening to human health are allowed to be administered to food animals. For example, the non-antibiotic growth promoter carbadox, approved in the EU as a feed additive for piglets, must now be formulated so as to preclude dust inhalation by mill operatives (137).

We note the statement by the Federation of Swedish Farmers, in August 1996: .

"Since 1986 no antibiotics or chemotherapeutics are added generally to the feed for growth promotion in Sweden. Antibiotics to farm animals are only used for curing disease and after a veterinary prescription.

The total consumption of antibiotics to farm animals since 1986 has reduced by over 40%. The amount added to animal feed has reduced by 90% from 30 tonnes in 1984 to 3 tonnes in 1995. There is no sign of a black market.

Swedish research has shown that less use of antibiotics has led to less risk of residues in food and less risk of building up antibiotic resistance.

The restrictions on antibiotic use have led to improved animal welfare, animal environment and management. To maintain production efficiency, Swedish producers have improved their skills and knowledge." (138).

Compassion in World Farming Trust concurs with Germany's federal chamber of veterinarians, the Bundestierarztekammer, which in November 1996 voted in favour of a ban on the use of in-feed antibiotic growth promoters, on the grounds that in-feed antibiotic growth promoters are not necessary in animals reared properly and in hygienic conditions (139).

Non-antibiotic growth promoters

There is a range of substances which increase the growth of farm animals and which are not antibiotics. The two most controversial groups are the beta-agonists (including clenbuterol, or 'Angel Dust') and the anabolic hormones.

Beta-agonists

Every cell in an animal carries membrane receptors ('alpha' or 'beta' receptors) which control the biochemical reactions that take place in that cell. Different active agents (or 'agonists') can stimulate these receptors in different ways.

Beta-agonists have dramatic effects on fat metabolism and muscle-cell activity. The breakdown of fat is stimulated, and the replenishment of fat stores is inhibited. Protein breakdown is reduced, and the production of muscle proteins is enhanced. The result is a leaner, more heavily muscled animal.

In 1992, an Animal Pharm report identified five beta-agonists as having been developed, or being in the process of development for use on farm animals: cimaterol; ractopamine; L-668,488; salbutamol; and clenbuterol (140). None of these substances has yet been approved for non-therapeutic animal use in the EU. The reason for this is the risk to human health from residues of the substances.

Physiological effects of beta-agonists

Beta-agonists are active in extremely low doses, and are known to affect human metabolism. They can cause bronchodilation -- salbutamol and clenbuterol have been used in human medicine, for the treatment of asthmatics. They can also induce uterine relaxation, increased heart rate, and heart tremors.

These side-effects can also be seen in livestock. In addition, some strains of pigs are reported to be sensitive to the effects of beta-agonists on the central nervous system, resulting in hind-limb paralysis (141).

Increased incidences of lameness and hoof lesions, and increased severity of hoof lesions have been reported in pigs treated with cimaterol (142), clenbuterol (143) and the Merck analogue L-644,969 (144).

Some derivatives of clenbuterol have up to 10 times the potency of clenbuterol itself, for example in reducing urinary excretion.

A veterinary expert at University College, Dublin has warned that residues of clenbuterol in meat might nullify the effects of medication taken by people suffering from blood pressure disorders, and would be deleterious to people with heart problems (145).

Increased heart rate, and tremors, have been reported in people in France and Spain, following consumption of calf and beef liver containing residues of clenbuterol. The French outbreak involved 22 people, and the adverse effects persisted for 2-3 days. The Spanish outbreak, in 1990, affected 135 people (146).

And in Ireland, it is alleged that three farmers have died after inhalation of clenbuterol powder while adding quantities to animal feed (147).

Despite the serious risks there can be from beta-agonists for both human and animal health, there is a long and persistent history of the unlawful administration of these substances to farm animals, right up to the present.

Widespread detection of beta-agonists in farm animals

In December 1992, a series of police raids in the Irish Republic resulted in the seizure of large quantities of clenbuterol. Banned substances were even reported to have been found at the home of an official in the Irish Department of Agriculture (148).

In the Spring of 1995, an investigation in Belgium revealed that up to 25% of samples of meat tested positive for clenbuterol (149). At around the same time, Belgian veterinary inspector Karel van Noppen was murdered, just ten days after detecting carcases containing banned substances at an abattoir in Rekken, West Flanders (150). In April 1995, a letter in The Veterinary Record revealed how Karel van Noppen had spoken of "the widespread use of illegal substances to enhance animal growth and the huge well-organised black market that supplied them ... huge sums of money were involved with widespread corruption among officials ... The black market, he said, was so well organised that it was able to compensate farmers whose animals were found to contain residues" (151).

Also in April 1995, Irish Department of Agriculture scientists discovered a new, modified form of clenbuterol in carcases at a meat processing plant. The scientists claimed that the chemical make-up of the substance had been altered in order to beat existing laboratory tests for beta-agonists (152).

In May and July 1995, reports revealed that meat and paté imported into the UK had been found to contain clenbuterol (153, 154).

In September 1995, the industry journal Animal Pharm reported that veal calves in Holland had been illegally treated with clenbuterol (155). And in December 1995 a federal grand jury in Milwaukee charged the Vitek Supply Corporation, and members of the company's staff, including its president Jannes Doppenberg with 12 counts of conspiracy, smuggling unapproved drugs into the US, and illegally adding the drugs to feed mixtures sold to veal and lamb producers throughout the United States (156). One of the drugs involved was clenbuterol, used to make confined calves and lambs put on muscle even though they get no exercise. Vitek has been linked with a veterinary pharmaceuticals firm, Pricor Inc., based in Holland. As a result of the investigation, charges were upheld of criminal conspiracy and of violating US food and drug laws (157).

Bradley Miller, national director of the San Francisco based Humane Farming Association which was instrumental in bringing the Vitek case to court, said: "The veal industry's disregard for animal suffering is only surpassed by its disregard for the health and safety of consumers" (156).

In January 1996, Irish Department of Agriculture officials seized a substantial quantity of clenbuterol during inspections of farms in the North Kerry area (158). And in February 1996, Farming News reported: "Irish police are investigating the murder of a farmer suspected of a second offence of using the illegal growth promoter clenbuterol ... samples from his farm were allegedly stolen in a raid on a meat factory last summer. According to Irish press reports, the murder may have resulted from the farmer breaking an agreement to pay for the results to be stolen. It is claimed his killers shot him in the leg, leaving him to bleed to death while his wife and daughter were tied up in an adjoining room" (159).

In February 1996, a Northern Ireland farmer was convicted of presenting for slaughter animals containing clenbuterol. The farmer was fined £500 (160).

In May 1996, Farming News noted that German police had found traces of clenbuterol in German beef herds (161). Subsequently, an article in The Guardian newspaper, in August 1996, detailed the use of clenbuterol by veal farmers in Germany (162). Animal Pharm, in September 1996, reported that 80-90% of all positive analyses of clenbuterol in Germany in 1995 were in veal calves (163).

In December 1996, Animal Pharm reported that an Irish farmer was jailed for ten months and fined IR£1000 for using clenbuterol in animal feed; the same report noted that a further 47 cases remained pending in Ireland at that time, involving clenbuterol, or clenbuterol/hormone 'cocktails' (164). In January 1997, a man in Ireland was sentenced to eight months imprisonment, and fined IR£1000 after admitting preparing and helping to distribute four tons of clenbuterol (165).

Most recently, in May 1997, the German regulatory authority BgVV revealed that residues of the unlicensed beta-agonist brombuterol had been detected in livestock. It is suspected that certain producers had resorted to using the drug in cattle and calves as a substitute for clenbuterol (166), in advance of the tightening of the licensing of clenbuterol for therapeutic purposes in the EU from July 1st 1997.

Beta-agonists -- reasons for taking the risk

The unlawful administration of beta-agonists to promote muscle development is particularly worrying in terms of the potential for residue-contamination of meat, because for maximum effect, these substances have to continue to be used as close as possible to the time of slaughter. This is because the withdrawal of the substance leads to a rapid compensatory deposition of fat (167). For example, treatment of broiler chickens with cimaterol for 14 days, followed by seven days withdrawal, resulted in the loss of most of the 'beneficial' effects (168).

And yet, residues of beta-agonists can be detected in animal tissues up to six days after the final dose (169).

The likely pattern of unlawful administration, therefore, is to dose the animals with the substance right up to the day of sale, observing no withdrawal period; this pattern of dosage is the one most likely to maximise financial return on the use of the drug, but it is also the pattern most likely to endanger human health from the presence of residues in the meat.

Perhaps this might explain why targeted testing for beta-agonists in Spain in 1994 revealed an incidence in slaughterhouses (22.02%) which was over ten times the level found on farms (1.92%) (170).

Clearly, then, the administration of beta-agonists such as clenbuterol to farm animals has been widespread and persistent, despite evidence of the harmful effects on human health from residues of these substances in meat.

Now, there is worrying evidence that, as analytical methods improve for known beta-agonists, farmers are prepared to stay one step ahead of detection by administering new varieties of these drugs.

There have even been suggestions that farmers may be administering cocktails of these drugs plus masking agents, in the same way that unscrupulous athletes are alleged to have done, in an attempt to avoid detection of illegal substances (171, 172).

Beta-agonists would appear to be considered, by certain farmers, as an important productivity tool, particularly for animals closely confined in factory farming conditions. How else could a farmer, so easily and so cheaply, put meat on a malnourished veal calf confined, without exercise, in a tiny veal crate? And in Ireland, at a cost of £30 per treatment, clenbuterol fed to a bullock for 18 days before slaughter will add around 110 lbs in weight, and around £100 in value to the animal, without extra feeding, putting farmers who use the drug illegally at a considerable competitive advantage over those who do not (172, 173, 174).

Anabolic hormones

A debate has been raging, particularly fiercely during 1995 and 1996, between the EU and the US, over the EU ban on treatment of food animals with hormones for growth promotion, and a similar ban on the importation into the EU of meat from food animals treated with such hormones.

Particular attention has been paid to the anabolic hormones, of which there are five main types:
testosterone: a naturally-occurring male hormone;

oestradiol: a naturally-occurring female hormone;

progesterone: a naturally-occurring female hormone;

trenbolone: a synthetic male hormone;

zeranol: a synthetic female hormone.

In 1988, European Council Directive 88/146/EEC prohibited the importation into the EU from third countries of animals, and meat from animals, which have been administered hormones as growth promoters (175). The administration of these hormones as growth promoters to farm animals in the EU was also banned, following concern from consumers about possible adverse effects on their health from eating meat from animals treated with these substances. No such ban was implemented in the United States. The ban in the EU is said to be costing the US beef industry $100 million a year in lost exports (176).

The battle against the EU ban

The EU ban began to be undermined most seriously in the summer of 1995. On 1st July 1995, the new trade rules agreed in the Uruguay Round of negotiations on the General Agreement on Tariffs and Trade (GATT) came into force, which strongly tightened up the circumstances under which the import of goods into a country could be blocked.

Then, on 6th July 1995, the Codex Alimentarius Commission (the UN body, supported by the World Health Organisation and the Food and Agriculture Organisation, responsible for setting safety standards for residues in food) voted by 33 countries to 29 (with seven abstentions) in favour of a resolution to adopt maximum residue levels for hormones -- implying that some degree of contamination of meat with hormones could be considered safe (177).

The Codex decision drew a swift response from the State Secretary Dr Baldur Wagner, at the German Ministry of Health: "The unanimous opinion in Germany is that these products are not only superfluous, but are also problematic ... Consumer and animal protection must also be considered. What is the point of artificially stimulating animal muscle growth with drugs, and how would such questionable practices be explained to responsible consumers?" (178).

The EC Agriculture Commissioner Franz Fischler also attacked the Codex decision as being "totally unacceptable", and said that it would "have no bearing on the EU's policy on hormones" (179).

But the introduction of the new GATT rules provided powerful support for the Codex Commission's decision. In August 1995, the journal Animal Pharm reported that João Mahalhães, counsellor of the World Trade Organisation's Agriculture and Commodities Division, felt that the Codex vote would contribute to a resumption of imports into the EU of US hormone-treated meat (the World Trade Organisation, or WTO, is the body which ensures implementation of GATT rules). Mr Mahalhães suggested that labelling could be used to allow consumers to choose between beef produced using hormones, and that which is not (180).

In September 1995, in an all-party resolution, the European Parliament backed existing import restrictions; the resolution noted that a reintroduction of hormone growth promoters would jeopardise animal and human health (181).

With the US hardening its stance (the US Department of Agriculture was reported, in September 1995, as being adamant that the EC's ban on imports of hormone-treated beef was not based on science, and was therefore contrary to WTO rules (182)), an EC conference was scheduled for the end of November -- the 'Scientific Conference on Growth Promotion in Meat Production' -- aimed at providing an up-to-date scientific analysis of the safety implications for humans, and effects on the health and well-being of animals treated with these substances.

In hindsight, the conference was a lost cause from the start, if it was felt that it could protect the EC's ban from the weight of the WTO. True, it provided a partial update on health and welfare issues (though its conclusions on the animal welfare effects of hormones were seriously misinformed -- see next section), but in reality the conference was being written off even before it began. Some commentators felt that the final arbiters would not be the scientists, but consumers. The Genetics Forum, for example, commented: "....consumers have to tell the supermarkets that whatever the law, they don't want hormones in their meat. If neither our Government nor the European Union can or will protect us, we will have to rely on the market to kill hormoned meat dead" (183).

A week before the conference, the UK National Farmers Union expressed its support for the EC's ban: "We are against additives and want the ban to stay. The consumer's wishes must be respected" (184). And Don Curry, chairman of the Meat and Livestock Commission, said: "... I was delighted that the whole industry, from farmer, processor, through to retailer and consumer, were all agreed that we should resist the American pressure", and "we within the EU should maintain the status quo internally within Europe and resist the introduction of the use of growth promoting hormones" (185).

Of course, it's not surprising that the meat industry in the UK is opposed to a lifting of the EC's hormone ban. If the ban were to be lifted, a significant quantity of US beef could come onto the UK market. In addition, if hormones began to be used in the EU, more EU beef would be produced, because the carcase weights of the animals would be increased. With the UK unable to export its own beef because of the BSE ban, beef prices in the UK could fall dramatically.

The EC conference at the end of 1995 broadly found no indications of possible human health risk from anabolic hormones in farm animals when used 'correctly' (186) (but see page 32 for the issues around 'correct' use). Nevertheless, the EC's Agriculture Commissioner Franz Fischler insisted that he would do everything in his power to prevent a lifting of the hormone ban (187). He also said: "What is clear is that we don't have all the research we need; we need to know more about abuse and fraud" (188). His position was supported by an editorial in Farmers Weekly, on 8th December 1995, which asked: "What guarantee could anyone offer that such treatments had been used responsibly?", and: "Who would benefit from allowing hormone-treated beef on to the market? Hardly livestock, not consumers and certainly not farmers in the long run" (189).

The UK's then Agriculture Minister Douglas Hogg was the only EU farm minister to support the United States' case for a lifting of the hormone ban, for which he was condemned by the National Federation of Meat and Food Traders (190) and the Farmers Union of Wales (191).

On 18th January 1996, the European Parliament unanimously adopted a resolution calling on the Commission and Council to oppose the import of hormone-treated meat into the EU (and, incidentally, regretting that too little attention was given at the EC conference to the environmental impact of the use of growth-promoters and to animal welfare) (192, 193).

Then, at the end of January 1996, the United States carried out its threat to lodge a formal complaint with the WTO over the EC's hormone ban (194). The next month, the US got support from Australia and New Zealand, when those two countries also lodged complaints with the WTO (195). Canada also lodged a complaint.

On 18th March 1996, EU agriculture ministers voted to tighten the ban on hormones in beef, by introducing stricter controls, checks and penalties -- one week before the EU entered negotiations with the US over the ban at the WTO; once again, the UK was the only country to oppose the tightening of the ban (196).

At this point, it began to seem that EC officials had perhaps made a calculated decision about the possible impact of losing the adjudication at the WTO. The Times, on 19th March 1996, noted: "The WTO can impose penalties equal to the estimated losses incurred from an illegal trade barrier" (197). The US is believed to be losing the opportunity of exporting 10,000 tonnes of beef each year to the EU as a result of the hormone ban. The estimated value is £60 million a year. Perhaps the EC had decided that a penalty of £60 million was a price worth paying,

compared with the possible impact on the EU beef market of lifting the ban on anabolic hormones?

The 60-days consultation period between the US, Canada, Australia, New Zealand and the EU at the WTO unsurprisingly did not achieve agreement on the hormone ban, so in May 1996, the US called for a dispute settlement panel to be established by the WTO (198). And in July 1997, the WTO dispute settlement panel produced its report , which found the EU's import ban illegal, because it lacks scientific justification. The EC is expected to appeal against the ruling (199).

Anabolic hormones and animal welfare

The EC's Scientific Conference on Growth Promotion in Meat Production, at the end of 1995, claimed that there was insufficient evidence of a negative impact of anabolic hormones on animal welfare; presumably, this played a part in the WTO's decision that there was no scientific justification for the hormone ban.

But this view is seriously flawed. There is ample evidence of adverse effects of anabolic hormones on animals' well-being.

Compassion in World Farming Trust has conducted a review of the scientific literature, and has found the following evidence, published during the period 1991-1995:

In 1991, researchers in Germany found that "Trenbolone and several steroid hormones ... were strong promoters of melanoma [tumours]" (200). In the same year, French research found that both "trenbolone and testosterone exhibit a weak transforming [mutagenic] effect" on cells in vitro (201). Also in 1991, van Leeuwen in Holland found that "increased tumour incidence observed in long-term studies in mice and rats arose as a consequence of the hormonal activity of TBA [trenbolone acetate] ... histopathological abnormalities (particularly in testes, ovaries and uteri) were observed in male and female pigs fed with high doses of TBA" (202). And in UK research, also published in 1991, it was found that "signs of abnormality were seen in the liver of animals receiving trenbolone acetate or testosterone" (203).

In 1992, American researchers showed that, for lambs implanted with zeranol, "There were structural changes in metacarpal bones from implanted lambs" (204). Also in 1992, UK researchers found that, following administration of trenbolone to pigs, "embryo survival in Regumate[trenbolone]-treated Meishan gilts was 14 percentage units lower than in control Meishan gilts" (205). The same year, in Germany, scientists found that calves given oestradiol, testosterone and trenbolone showed "cysts in two-thirds of cases" (206).

In 1994, American researchers showed that oestradiol plus progesterone "tended to reduce heifer fertility" (207). The same year, researchers in Spain found that, following administration of trenbolone acetate to pigs, "Histological study of prostate demonstrated structural modifications. As a consequence of the treatment glandular lumen was increased and most parenchyma was occupied by poly-morphous cysts" (208). In other words, there was widespread disruption of the internal prostate tissue.

In 1995, King et al in Canada found that "Pregnancy loss in zeranol-implanted heifers was 37.5% compared with 0% for control heifers" (209). And also in 1995, US scientists found that feedlot steers given zeranol showed "a significant decrease in bone Ca [calcium] concentration ... Loads withstood by the bones up to flexure and the strain at flexure were inversely related to the

quadratic of zeranol dose [i.e. as zeranol is increased, the load-bearing capability before the bones start to bend is decreased]" (210).

Tumour-promotion, liver abnormalities, cysts, increased pregnancy-loss, bone weakness .... throughout a period of several years, in research groups all around the world, evidence has been accumulating of detrimental effects on animals of anabolic hormones.

Compassion in World Farming Trust believes that, far from the EU hormone ban being lifted, the administration of anabolic hormones to farm animals for growth promoting purposes should be banned world-wide.

Anabolic hormones and human health

With so many abnormal effects being discovered when anabolic hormones are administered to animals, it is not surprising that people should be apprehensive about eating meat from animals treated with these substances. Remember, these substances are generally implanted as pellets into farm animals, and are released via the bloodstream to all parts of the animal. Two of the substances (trenbolone and zeranol) are not naturally-occurring, so one might expect that humans and cattle do not have the capacity to regulate the levels of these substances once they are released into the body. Perhaps this is why a European Commission document, in 1984, noted that a study of trenbolone had failed to discover a minimum dose at which no hormonal activity, in several species of test animals, could be detected (211). The results for zeranol were even more alarming -- they showed "evidence of gross hormonal effects at all levels tested. A no-hormonal effect level was not determined" (212).

Of course, the manufacturers of each of these substances claim that they are safe, if used according to their instructions, and provided that the manufacturer's withdrawal period is strictly observed. But we have already seen, in this report, that a frequent reason for the detection of residues in meat in quantities above maximum permitted levels is the failure to adhere to recommended withdrawal times. In 1994, the main cause of residue violations in the US (43.4%) was failure to adhere to approved withdrawal times (135).

And even when residues of any one substance are below maximum permitted levels, this may be no guarantee of safety, if the farmer is using substances from different manufacturers, in combination with one another:

There is evidence that combinations of these kinds of chemicals can be powerfully synergistic in their activity. Researchers at Tulane University, in 1996, presented evidence that oestrogen-mimicking chemicals can be up to 1,600 times more potent, in combination, than separately (213).

What will be the effect on human health of consuming meat containing traces of combinations of these substances? We simply do not know.

Why some countries want to use anabolic hormones

So, in the face of opposition from European farmers, evidence for adverse effects on animal well-being, and unquantified but potentially serious risks to human health, why is the US farming industry so committed to the use of anabolic hormones? Yet again, the reason is that these substances provide a 'technical fix' required to support an unnatural and inherently

unsustainable intensive farming regime -- the vast 'feedlots' of the United States, Australia and Canada.

As a Canadian beef farmer explained, on BBC Radio 4's 'Farming Today' programme, in November 1995:

"Hormones are used quite commonly in the raising of beef cattle in Canada. We implant about 40% of the animals that are raised for meat purposes. These implants are used mostly in the larger commercial feedlots, perhaps slatted floor barns that are feeding out thousands of head of cattle each year ... All our male calves are traditionally castrated in Canada at approximately three months of age ... we find that we have to have these animals castrated so we can mix them easily with cattle from other farms. Because these animals are castrated we lose some of the production efficiency. To regain this efficiency we implant growth hormones and find that we can improve weight gain by roughly 10% through the use of hormones" (214).

So there it is:

The intensive farming of thousands of head of cattle in the feedlots of the US, Canada and Australia brings together cattle from many different farms.

In order for these cattle to 'mix easily', they are castrated.

But castrated cattle do not grow quickly enough for the feedlot operators.

So, to make them put on 10% more weight, the cattle are implanted with anabolic hormones.

And because this is what the feedlot operators of America are doing to their cattle, the EU is being told by the WTO to lift its ban on the import of meat from these hormone-implanted steers -- in spite of resistance from European farmers, uncertainty about the risks to human health, and widespread scientific evidence of animal health problems associated with the use of these substances.

Compassion in World Farming Trust believes that it is unacceptable potentially to jeopardise human health and animal welfare, simply to allow the farming industry to reap maximum profit by chemically boosting productivity of animals which naturally do not thrive in a fundamentally flawed intensive farming regime.

Regulating food safety

Unpicking the safety harness

The evidence cited in this report clearly shows that animal welfare and food safety are two sides of the same coin. When animal welfare is jeopardised by overcrowded and unhygienic rearing, transport and slaughter conditions, food safety is compromised.

Loosened regulations and inadequate enforcement regimes have allowed the farming and food industry in the UK to put at risk the well-being of millions of farm animals, and also millions of consumers.

Stephen Dorrell's official announcement on March 20th 1996 of a probable link between BSE and nvCJD lit the fuse which has blown apart the confidence of meat-eaters in the UK, but

looking back at events during the year prior to Dorrell's announcement, the relentless and reckless deregulation of the food and farming industry is apparent:

On 15th July 1995, the UK Government relaxed rules of food hygiene, just as new figures were released showing record high levels of food poisoning. 12 sets of temperature regulations for food-storage were revoked, and replaced by just two. The maximum temperature allowed for certain cook-chill foods was increased from 5C to 8C. Announcing the change, Baroness Cumberlege said: "We are determined to reduce bureaucracy whilst maintaining the best hygiene standards" (215).

Later the same month, Farming News reported the concerns of veterinarians and animal health consultants about the loosening of regulations on the import of veterinary medicines into the UK. Bob Stevenson, vice-president of the British Veterinary Association, said: "At present the UK vet is being by-passed in many cases, and we feel somewhat castrated if we are not in touch with what is happening on farms". Mr Stevenson was reportedly worried that, if farmers misunderstood directions on the foreign labels, residues could leak into the human food chain. Roger Cook, from the National Office of Animal Health (NOAH), said: "Livestock farmers are risking their industry which has been under a lot of pressure from consumer groups and supermarkets lately for the sake of a few pounds" (216).

Then, in October 1995, the Government's advisory panel on healthy eating, the Nutrition Task Force, was axed. Bill Shannon, general manager of the Co-op, attacked the abolition of the panel: "To stop now is ludicrous; urgent action is clearly needed ... consumer groups accuse the Government of bowing to pressure from vested interests in the food industry which, they believe, make profits from unhealthy eating" (217).

In November 1995, MAFF's Steering Group on Chemical Aspects of Food Surveillance was disbanded. The group had investigated, among other things, veterinary residues in food. Writing in The Food Magazine about how the steering group was disbanded, Suzi Leather (the first and only consumer representative on the committee) said: "The committee was axed without public consultation or even a warning to the committee members. We simply got a letter thanking us for our work" (218).

And in December 1995, the former Agriculture Minister Douglas Hogg boasted a series of "food deregulation measures ... a major package of measures to lighten the burden of food law on business ... These measures will take effect on 1 January 1996 -- a good start to the New Year" (219). Within a few months, this "good start" had been swept away, with a world-wide ban on British beef, mass slaughter of (for the most part) healthy animals, and a collapse in consumer confidence -- brought on, most probably, by an apparently harmless deregulation of the processes involved in animal feed production, fifteen years before, resulting in a lowering of the temperature at which the feed was processed.

And with the world's worst E. coli O157 outbreak occurring in the UK at the end of 1996, the pressure was on for a tightening of all the regulations that had previously been discarded. Christopher Haskins, head of Northern Foods, interviewed in The Guardian, said: "While I thought I knew most of what was going on I was taken aback by the lack of regulation. I thought it was bad, but not as bad as it turned out to be" (220).

Professor Hugh Pennington, commenting in the wake of the Scottish E. coli outbreak, noted that enforcement of legislation on food safety "has, at the request of the Government, been pursued generally with a light touch ... We believe that approach can no longer be considered appropriate

or acceptable" (221). He also said: "The Government was quite relaxed about the implementation because clearly it was technically difficult and it cost money ... Targets were set and were then relaxed ... We are unhappy with that" (222).

The president and vice-presidents of the Veterinary Public Health Association, writing in The Veterinary Record in November 1996, emphasised the central importance of animal welfare in food safety, noting that: "Safe food can only be produced if a healthy, clean, residue and stress free animal is delivered to the slaughterhouse" (223).

The President of the European Commission, addressing the European Parliament in February 1997, asked: "Can we really go on claiming that BSE is an accident of nature? Is it not actually the consequence of a model of agricultural production which pushes productivity at whatever cost?" (224).

And The Veterinary Record in April 1997 reported a paper by BVA vice-president Mr Keith Baker, which highlighted the need for an integrated system, taking into account animal health, animal welfare and public health in food production (225).

By May 1997, with the publication of a Eurobarometer poll showing that 35% of EU citizens think that food is not safe (226), the pressure was on the new UK Government to deliver structural reforms in the farming and food industry, to restore consumer confidence.

Animal welfare and the new food agency

The 'front runner' for the long, slow job of restoring consumer confidence seems to be the creation of an independent food safety agency. Consultation on the structure and remit for the agency was completed on June 20th 1997, and the process of implementation is now underway.

This report has demonstrated that farm animal welfare and food safety are indivisible, so one might question the wisdom of taking responsibility for food safety out of the Ministry of Agriculture, which retains its responsibility for animal welfare.

Indeed, in Denmark, where there was previously an independent National Food Agency, this body was merged, on January 1st 1997, into a new Ministry of Food, Agriculture and Fisheries -- quite the opposite to the action being undertaken in the UK. The working group which recommended this course of action in Denmark had concluded that problems with public health, food poisoning, contaminants and additives stemmed mainly from primary production, and so the system for controlling food safety had to include responsibility for what happened on the farm (227).

But Tim Lang, Erik Millstone and Mike Rayner, in their seminal paper "Food Standards and the State: A Fresh Start" have rightly suggested that the benefits to be obtained in the UK by bringing into the open the tensions which exist between production and consumption would outweigh the disadvantages (228).

So, in the UK the split could be achieved by making the Food Standards Agency responsible for auditing standards of animal welfare agreed with the Ministry of Agriculture (the Ministry of course would retain its duty to enforce the relevant legislation).

Lang et al are exactly right in advocating that the new food agency should seek standards-driven improvements (and Compassion in World Farming Trust believes that these standards must encompass animal welfare, if food safety and farm animals are properly to be protected).

In many other areas of commerce, consumer-driven quality-standards are already in place, right through the 'supply-chain'. And in the area of environmental protection, EU-wide Environmental Management Standards exist, by which organisations commit themselves to a policy of environmental protection.

In the same way, it should be a responsibility of the new Food Standards Agency, working together with the Ministry of Agriculture, to develop the highest standards of animal welfare, on which farms can be publicly audited, to allow them to demonstrate to their customers in the supply-chain that their meat is both safe and humanely produced.

Naturally, the Food Standards Agency would be expected to take the lead in driving these standards upwards over time.

The UK farming industry, in order to remain competitive, has no choice but to adopt such a standards-driven approach to improved farm animal welfare. It is only a matter of time before consumers expect products to be humanely produced, and they will not expect to pay a price-premium for the privilege. And if the UK farming industry cannot deliver, then the consumer will look elsewhere.

Summary and conclusions

Much of our meat is infected

There is evidence that the meat from farm animals is frequently contaminated with disease-causing organisms:

up to 41% of chickens on retail sale in the UK have been found to be contaminated with Salmonella;

up to 48% of fresh chickens on sale in the UK have been found to be contaminated with Campylobacter;

in one survey, 11 out of 12 turkeys examined contained Campylobacter;

surveys of retail outlets in London have found VTEC E. coli in 22% of raw beefburgers and 25% of raw pork sausage.

Compassion in World Farming Trust believes that levels of contamination of UK meat with disease causing bacteria are unacceptably high.

Bacterial contamination seems to originate on farms

There is evidence for the origin of much of the contamination on the farm:

research by the Food Microbiology Research Division of the Department of Agriculture for Northern Ireland has found that "those animals which are intensively reared and slaughtered young will have the greatest potential for carrying pathogens";

Salmonella and Campylobacter contamination has been found on walls, feeders, drinkers, floors, insects, water and feed at intensive poultry farms;

elimination of infection of a specific broiler farm by a particular Campylobacter strain resulted in a major reduction in the same Campylobacter strain in the local human population;

scientists have found a one month lag between warm weather and increased incidence of food poisoning;

hides from cattle in winter (winter cattle spend months indoors) show around 100,000 times more microbial contamination than hides from cattle in summer (summer cattle spend more time in the open);

the Advisory Committee on the Microbiological Safety of Food has said: "carcases contaminated during the slaughtering and dressing process represent an important route by which pathogenic organisms such as E. coli O157 can enter the food chain;

serological studies have found higher than expected levels of antibodies to Campylobacter in personnel in poultry and red meat plants, suggesting that the carcases being processed are significantly contaminated by the time the animals arrive at the abattoir.

Compassion in World Farming Trust believes that urgent actions must be taken, on UK farms, to bring down the levels of disease-causing bacteria. These actions need to include improved hygiene (so that animals, for example, are not forced to lie or stand in their own excrement), more access for animals to natural daylight and the outdoors, and significant reductions in overcrowding.

Food poisoning is increasing relentlessly

Advice on kitchen hygiene, and thorough cooking, is clearly proving inadequate in the face of contamination "upstream" of retail outlets; this is manifest in huge rises in the incidence of food poisoning:

according to the Office of National Statistics, food poisoning in England and Wales has increased six-fold over the past 15 years; . one of the reasons given for this huge increase was "more intensive rearing of chickens and farm animals under conditions that can spread germs";

according to Scottish Office figures, there were 858 food poisoning cases in Scotland in 1970; in the first 10 months of 1996 (that is, prior to the major outbreaks of E. coli O157) there were 8,408 cases;

food poisoning, in England and Wales alone, is estimated to be costing industry and the taxpayer somewhere between £1 billion and £3 billion every year, based on figures from the Public Health Laboratory Service.

Compassion in World Farming Trust believes that significant reductions in the levels of food poisoning in the UK will only be made when hygiene and overcrowding are improved on farms, allowing less highly contaminated meat to be made available to customers.

Problems with antibiotic use

According to the World Health Organisation, more than half of the total production of antimicrobials world-wide is currently used on farm animals, with a large proportion being administered not to treat disease but to promote growth. There are a number of potential human health risks:

a senior scientist at the Central Public Health Laboratory has said: "The use, misuse, or overuse of antibiotics in animal husbandry is creating a selective pressure which is encouraging the emergence and persistence of resistant, multi-resistant strains of Salmonella in food animals which then go into the food, causing epidemic spread not only on the farm but also eventually in us";

there is evidence which suggests that the use of some antibiotics to promote the growth of farm animals may be leading to a build up of bacteria resistant to human medicines;

administration of an antibiotic to farm animals to promote growth can increase the animals' susceptibility to Salmonella infection;

scientists should investigate as a matter of urgency the possibility that administration of growth promoting antibiotics to farm animals might be risking an explosion of disease-causing bacteria such as E. coli O157, by knocking out naturally-occurring bacteria which normally hold these pathogens in check;

residues of sulphonamide antimicrobials have been detected in UK pigmeat, above maximum permitted levels, every year between 1990 and 1996; there have been reports that sulphonamides can impair kidney function, and a study in the USA has reported a link with thyroid cancer;

scientists in 1996 discovered "alarmingly high" levels of mutation in E. coli O157 and Salmonella; some mutation seemed to be via horizontal transmission of genes between bacteria. Compassion in World Farming Trust is concerned that the overcrowding of animals in unhygienic factory farms, coupled with very high levels of antibiotic use, may constitute an uncontrolled experiment in bacterial genetic engineering on an enormous scale. We strongly recommend that the implications for human health are investigated as a matter of urgency.

Compassion in World Farming Trust believes that it is unethical to administer growth promoting antibiotics to farm animals which are already suffering as a result of unnaturally high growth rates brought about by selective breeding. The human health consequences of the widespread use of growth promoting antibiotics are also poorly understood, and their use should be discontinued.

The widespread misuse of beta-agonists

Beta-agonists (such as clenbuterol -- 'Angel Dust') are being widely misused to increase the amount of muscle in farm animals. These substances can have damaging effects on human and animal health:

beta-agonists can induce uterine relaxation, increased heart rate and heart tremors in humans and livestock;

increased heart rate and tremors have been reported in people in France and Spain, following consumption of calf and beef liver containing clenbuterol residues; the Spanish outbreak involved 135 people;

an Irish veterinary expert has warned that residues of clenbuterol in meat might nullify the effects of medication taken by people suffering from blood pressure disorders, and would be deleterious to people with heart problems;

some beta-agonists can cause increased incidences of lameness and hoof lesions in pigs;

some strains of pigs can develop hind-limb paralysis in response to beta-agonists;

meat and paté, imported into the UK have been found to contain clenbuterol;

offences linked to the misuse of clenbuterol and related substances have been reported in the Unites States, Germany, Holland, the Republic of Ireland and Northern Ireland.

Compassion in World Farming Trust believes that the potential for adverse effects of beta-agonists on animal and human health means that their use for growth-promoting purposes should continue to be prohibited.

The EU hormone ban

In 1988, the EC prohibited the importation into the EU of meat from animals administered hormones as growth promoters. Several countries, including the US, are currently seeking that the WTO overturns this ban. Yet there is ample evidence of adverse effects of anabolic hormones on animals' well-being:

trenbolone and several steroid hormones have been found to be strong promoters of melanoma (tumours);

trenbolone and testosterone have been shown to be mutagenic;

trenbolone acetate has been associated with testis, ovary and uterus abnormalities in pigs, and with liver abnormalities in other animals;

calves given oestradiol, testosterone and trenbolone develop cysts in two-thirds of cases;

zeranol-implanted heifers have increased rates of pregnancy-loss;

the bones of feedlot steers given zeranol have decreased load-bearing capacity.

Anabolic hormones are used in the feedlots of the United States, Canada and Australia to make cattle put on more weight following castration. Castration is used to allow cattle from many different farms to 'mix easily' in the feedlots.

Compassion in World Farming Trust does not believe that animals' health should be jeopardised by the administration of anabolic hormones, in order to facilitate the imposition of an unnatural, intensive farming regime. Far from the EU hormone ban being lifted, Compassion in World Farming Trust would like to see the administration of anabolic hormones to farm animals for growth promoting purposes banned world-wide.

Regulating food safety in the UK

Farm animal welfare and food safety are indivisible. Whilst the UK Ministry of Agriculture should retain its responsibilities for setting animal welfare policy, and for enforcement, Compassion in World Farming Trust also believes that the new Food Standards Agency should have farm animal welfare within its remit:

the Food Standards Agency should seek standards-driven improvements to food safety, and these standards must encompass farm animal welfare, if food safety and farm animals are properly to be protected;

the Food Standards Agency should be responsible for publicly auditing standards of animal welfare agreed with the Ministry of Agriculture, so that farms can demonstrate to their customers in the supply-chain that they are producing food which is both safe and humanely produced;

the Food Standards Agency should be active in driving farm animal welfare standards upwards over time.

Compassion in World Farming Trust believes that it is only a matter of time before consumers expect products to be humanely produced, and they will not expect to pay a price-premium for the privilege. If the UK farming industry cannot deliver, then the consumer will simply look elsewhere. It is no longer acceptable for the farming industry to risk the health of its customers, and cause suffering to the animals in its care, by rearing them in overcrowded and dirty conditions, and by administering to them artificial substances which push them ever further beyond their natural growth rates, simply to maximise the industry's profit.

Dr Tim O'Brien
August 1997

Glossary

Anabolic hormones: Natural or synthetic hormones affecting metabolic processes leading to an increase in muscle production.
Antibiotics: Substances, originally produced by bacteria or fungi but now commonly synthesised, used to prevent the growth of other species of micro-organism (bacteria, fungi or viruses -- though frequently applied in a restricted way, to mean only those substances preventing growth of bacteria). Some are used to make animals grow more quickly.
Antibiotic resistance: The ability of specific micro-organisms to withstand antibiotics. Over-use of particular antibiotics over time will lead to a build up of such resistant micro-organisms.
Antimicrobial: Any substance which prevents the growth of bacteria, fungi, viruses or protozoa (such as the protozoa which cause coccidiosis in poultry).
Aplastic anaemia: A deficiency of haemoglobin (the oxygen-carrying component of blood) caused by a failure to produce sufficient red blood cells.
Beta-agonists: Substances (such as clenbuterol) which stimulate the beta-receptors on cell membranes, increasing fat break-down and muscle production.
Broiler chickens: Those chickens which are reared for meat.
BSE: Bovine Spongiform Encephalopathy ('Mad Cow' Disease). One of a group of transmissible spongiform encephalopathies which cause brain degeneration in animals, including humans.
Campylobacter: Bacteria which are the major cause of food-borne gastro-intestinal infection in humans in the UK. Symptoms include headache, nausea, diarrhoea and vomiting.
Campylobacteriosis: An infectious disease caused by Campylobacter bacteria.

CJD: Creutzveldt-Jacob Disease. A transmissible spongiform encephalopathy affecting humans. sometimes referred to as the human equivalent of BSE.
Clenbuterol: A beta-agonist, sometimes referred to as 'Angel Dust'. Used illegally to increase muscle-production in farm animals.
Codex Alimentarius Commission: Established by the United Nations FAO Conference and World Health Assembly of 1961/62. Works to implement the UN's Joint FAO/WHO Food Standards Programme to "protect the health of consumers and ensure fair practices in the food trade".
Colisepticaemia: Blood poisoning resulting from infection by certain strains of E. coli.
Competitive exclusion: The exclusion of certain types of micro-organism from an animal by micro-organisms of a different type or species.
E. coli: Escherichia coli. The most common species of intestinal bacterium. Certain strains are capable of producing enterotoxins responsible for serious disease in animals including humans.
E. coli O157: A strain of the E. coli bacterium responsible for deaths and serious illness in humans. E. coli O157 frequently produces verocytotoxins.
FAO: The United Nations Food and Agriculture Organisation. Founded in 1945, with a mandate to raise levels of nutrition and standards of living, to improve agricultural productivity, and to better the condition of rural populations.
Feedlots: A system of housing large numbers of cattle in a pen, or groups of pens. tens of thousands of cattle may be housed in this way, on a single feedlot.
GATT: The General Agreement on Tariffs and Trade. The forerunner of the World Trade Organisation, it defined rules for trade in goods. Now amended and incorporated into the WTO Agreements (which also cover services and intellectual property rights).
GI: Gastro-intestinal. That part of the alimentary canal extending from the stomach to the anus.
Growth hormones: Generally used to mean natural or synthetic anabolic hormones, including steroids, administered to animals to make them grow more quickly. Does not include beta-agonists or antibiotics.
Guillain-Barré Syndrome: A human illness which results in paralysis. Most commonly caused by Campylobacter infection.
Hock burns: Damage to skin around the 'ankle' region in poultry. Frequently caused by prolonged contact with ammonia-soiled litter in broiler sheds.
HUS: Haemolytic Uraemic Syndrome. A form of kidney failure which can be fatal in up to 10% of cases. This can follow infection by verocytotoxin-producing E. coli.
Listeria: Bacteria found in faeces and animal tissues. Can cause abortions and nervous diseases in sheep, miscarriages and still-births in pregnant women, and serious illness in newborn babies.
Listeriosis: An infectious disease caused by the bacterium Listeria monocytogenes.
Metabolites: Most commonly, the break-down products of material taken into the body, such as food, antibiotics, or implanted growth promoters.
Microflora: A population of micro-organisms. Often used to describe the collection of different types of micro-organisms which inhabit an animal's gastro-intestinal tract.
MRL: Maximum Residue Limit. The maximum concentration of a residue of a specific substance that is legally permitted or recognised as acceptable in or on a food.
Multi-resistant bacteria: Bacteria resistant to more than one antibiotic.
Mutagenic: Able to cause a change (a mutation) in the genetic material DNA.
Oedema disease: An infectious disease of young pigs, caused by certain strains of E. coli. Symptoms include convulsions, paralysis, accumulation of excess fluid, and sudden death.
Oestradiol: The major anabolic steroid female sex hormone. Triggers the onset of oestrus. Because of its anabolic properties, it is sometimes administered to animals to increase their weight.
Oestrogen: Any of a group of female sex hormones that promote the development of secondary sexual characteristics and control the oestrus cycle.

Pathogenic: Capable of causing disease.
Progesterone: An anabolic steroid female sex hormone which prepares the reproductive tract for pregnancy. Because of its anabolic properties, it is sometimes administered to animals to increase their weight.
Salmonella: Bacteria causing serious diseases (diarrhoea, dysentery, fever, septicaemia and abortions) in animals including humans. Primarily inhabit the intestine, but can survive outside the host for prolonged periods.
Serotypes: Categories into which species of bacteria are placed, based on the types of antibodies they elicit, or the antigens that they produce.
Sulphonamides: A group of chemically-similar antimicrobial substances, discovered before antibiotics, with a broad spectrum of activity against numerous micro-organisms.
Testosterone: An anabolic steroid male sex hormone which causes development of male secondary sexual characteristics. Because of its anabolic properties, it is sometimes administered to animals to increase their weight.
Transferable drug resistance: The capacity of bacteria to pass among themselves the ability to resist the effects of certain antibiotics.
Trenbolone: A synthetic anabolic steroid growth promoter. Weaker male sex hormone activity than testosterone.
Verocytotoxins: Toxins harmful to cultured Vero cells (African green monkey kidney cells). Produced by certain strains of E. coli (VTEC). These toxins can produce illness, sometimes death, in humans.
VTEC: Verocytotoxin-producing E. coli; that is, those strains of the E. coli bacterium capable of producing verocytotoxins.
WHO: The United Nations World Health Organisation. Founded in 1948, the objective of the World Health Organisation is the attainment by all peoples of the highest possible level of health.
WTO: World Trade Organisation. Established in 1995, as a successor to GATT. The WTO Agreements are the legal ground-rules for international commerce and for trade policy.
Zeranol: A synthetic non-steroidal growth promoter. Thought to act by stimulating the pituitary to increase production of somatotropin. Weaker female sex hormone activity than oestradiol.

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