Bacterial Foodborne Disease
Medical Costs and Productivity Losses

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Introduction

Foodborne diseases in the United States cost billions of dollars each year. The Council for Agriculture and Science Technology (CAST) concludes that microbial pathogens in food cause 6.5-33 million cases of human illnesses in the United States and up to 9,000 deaths each year (1994). Pathogens are microorganisms that cause diseases and include bacteria, fungi, parasites, and viruses. Researchers at the Economic Research Service (ERS) of the U.S. Department of Agriculture (USDA) estimate that the annual cost of human illnesses for seven of these foodborne pathogens (six bacteria and one parasite) from all food sources is $5.6-$9.4 billion (*Federal Register* Feb. 3, 1995). Of these estimated costs, meat and poultry sources account for $4.5-$7.5 billion (*Federal Register* Feb. 3, 1995). These estimates undervalue the true costs of foodborne illnesses to society, because there are over 40 different foodborne pathogens believed to cause human illnesses (CAST 1994, pp. 11-15).

This report documents ERS analyses for the six bacteria mentioned above, providing a comprehensive, detailed accounting of how the cost-of-illness (COI) estimates were calculated and updated to 1993 dollars.[1] Previously, documentation for COI studies on these bacteria were spread out over diverse sources as each individual COI analysis was completed. This report is the first to comprehensively document the ERS COI analyses in terms of a given base year (in this case, 1993 dollars). The intended audience for this report is all researchers and policymakers interested in the societal costs of food safety.

Specifically, this report presents previously estimated costs of salmonellosis (Roberts 1988), listeriosis (Roberts and Pinner 1990), *E. coli* O157:H7 disease (Roberts and Marks 1995), campylobacteriosis (Lin *et al*. 1993), *Staphylococcus aureus* illness (Roberts 1989), and *Clostridium perfringens* illness (Roberts 1989), after updating to 1993 dollars and with more recent estimates of annual cases and deaths. These six bacteria from all food sources cost the United States an estimated $2.9-$6.7 billion annually (in 1993 U.S. dollars), with $1.8-$4.8 billion attributable to meat and poultry (*Federal Register* Feb. 3, 1995).

The COI estimates reported here can be used in three main ways. First, they can be used to evaluate the economic impact of foodborne diseases on the United States. Second, they can be used to target pathogen reduction efforts toward the most costly diseases. Third, they can be used to compare benefits and costs of control efforts to determine the most cost-effective interventions.

Bacteria

Of the four pathogen types (*i.e.*, bacteria, fungi, parasites, and viruses), Bean *et al.* (1990) found that over 90 percent of confirmed foodborne human illness cases and deaths reported to the Centers for Disease Control and Prevention (CDC) are attributed to bacteria.[2] Bacteria are commonly found in soil, water,

[1]The parasite, *Toxoplasma gondii,* was not included in this document because we wanted to focus on bacterial pathogens as a group and because the cost-of-illness analysis for *T. gondii* was well documented by Roberts and Frenkel (1990).

[2]Note that bacteria are more easily cultured than viruses and therefore the percentage attributed to bacteria may be biased upward. Also note that roughly half of the foodborne outbreaks cannot be attributed to a specific bacterial, viral, parasitic, or fungal pathogen.

plants, and animals (including humans). Most bacteria do not cause human illnesses and society relies on some bacteria to make bread, alcohol, vitamins, and antibiotics. Bacteria in the human body outnumber human cells (CAST 1994, p. 24). Over 400 species of bacteria live harmlessly in the gastrointestinal tracts of humans and some live on human skin (CAST 1994, p. 24). For example, *Staphylococcus aureus* lives harmlessly on human skin and in nasal cavities of up to 50 percent of all people in the United States; present in food, however, it can produce toxins that cause human illness (Labbe 1989, p. 498).

Food sources account for most human illness cases caused by the six bacteria discussed here except for *Staphylococcus aureus*. People can also be exposed to some bacteria through inhalation, contaminated drinking water, and contact with infected pets, farm animals, and humans. Here, "food sources" is broadly defined to include all sources of exposure to pathogens in the food chain, between exposure at the farm or production level to exposure at the food consumption level. Here, food sources also include secondary sources of exposure to foodborne illnesses, such as transmission of a foodborne illness from an ill person to other family members or other children in a day care center. Therefore, "foodborne illnesses" or "foodborne diseases" can originate at any of these stages.

Table 1 lists seven pathways through which people can be exposed to pathogens found in animals. Illnesses from exposure to pathogens through any of those seven pathways are included in estimates of foodborne diseases. Illnesses in farm families or slaughterhouse workers that arise from either direct or indirect contact with live animals are categorized here as illnesses from food sources, because these illnesses would not have occurred had these people not been exposed to this occupational hazard.

Although table 1 focuses on pathogens found in meat, an abbreviated list could be developed for pathways of human exposure to pathogens found in non-animal food sources, such as on fruits and vegetables. Also, the seven pathways in table 1 do not have an equal likelihood of causing human illnesses. Most foodborne illnesses occur from consumption of food contaminated with pathogens. Less common is the inclusion of kitchen/processing plant workers who become

Table 1—Potential pathways of human exposure to pathogens found in animals

I. Direct contact with live food animal.
- ° Food animal bite.
- ° Contact with the skin, fur, tail, etc., and microorganisms found there.

II. Indirect contact with the live food animal.
- ° Aerosol contamination of the barn and air system.
- ° Contamination of the walls, floor, gates, etc.
- ° Animal waste.
- ° Bites by flies or fleas that had become disease vectors from previous contact with infected animals.

III. Direct contamination by the carcass.
- ° Penetration of the skin of the personnel handling meat by microorganisms.
- ° Entry of organisms through cuts and nicks on the hand of slaughterhouse or processing plant workers.

IV. Indirect contamination by the carcass.
- ° Aerosol contamination through pathogens released when the carcass is cut up and/or slapped onto the counter.
- ° Contact with knives, wiping clothes, sinks, etc., where pathogens have been deposited.

V. Cross contamination of other edible products from the environment, other foods, or pests.
- ° In the slaughterhouse, spreading from one contaminated carcass to others.
- ° Meat products in the processing plant.
  - ° Other raw or cooked foods in the kitchen of a private home or commercial feeding establishment.

VI. Consumption of meat, poultry, and dairy products.

VII. Person-to-person transmission.

Source: Economic Research Service, USDA, adapted from Roberts, T. "A Retrospective Assessment of Human Health Protection Benefits from Removal of Tuberculous Beef," *Journal of Food Protection* 49,4(April 1986):293-8.

ill by handling contaminated food. For example, brucellosis, psittacosis, and tuberculosis are occupational hazards among slaughterhouse workers.

Some pathogens that cause human illnesses are carried by animals but do not cause animal diseases. *Escherichia coli* O157:H7 seems to live innocuously in the intestinal tracts of some cattle, though people who eat rare hamburgers from infected animals can develop bloody diarrhea and kidney failure.

Farm livestock and poultry infected with bacterial pathogens may spread infection among the herd or flock through their excrement. Nonetheless, contamination of meat and poultry flesh does not usually occur until slaughter. For example, Martz (1994-95) quotes Stephen Knabel, a food scientist at Pennsylvania State University, as stating that "only 5 percent of live poultry are contaminated with *Salmonella*, but after processing, nearly half of the carcasses contain *Salmonella*." Defeathering, slaughtering, chilling, and processing stages all provide opportunities for cross-contamination. Accidental puncturing of the intestinal tract during slaughter can lead to widespread contamination of the packing line.

Animal products such as milk and eggs also require proper handling. For example, if pasteurization of raw milk is not done properly, some *Listeria* may survive, though injured, and recover sufficiently to grow in refrigerated milk. Proper sanitation on the farm, in fishing vessels, and in slaughter and processing plants can reduce the pathogen level in food that goes to retail.

For each of the six bacterial pathogens discussed here, table 2 provides estimates of the number of annual U.S. cases and deaths from all sources and from foodborne sources. The estimates are subject to revision as new data become available or alternative databases are used.

Table 3 presents major and minor food sources for these pathogens. Foods most likely to cause outbreaks of human illness in the United States are animal foods and their products such as meat, poultry, seafood, dairy products, and eggs (CAST 1994, p. 32). Table 3 also lists acute symptoms and chronic complications associated with infections from each of these foodborne pathogens.

Human illnesses caused by microbial pathogens are generally classified in three categories: foodborne infections, foodborne toxicoinfections, and foodborne intoxications (CAST 1994, pp. 17-20). Figure 1 shows the classification of foodborne disease causes.

- *Foodborne infections* occur when pathogens are eaten and are then established in the body. The pathogens usually multiply inside human intestinal tracts, irritate the lining of the intestines, and cause human illnesses. Sometimes, the pathogens invade other tissues causing additional infections. Of the six pathogens investigated here, *Listeria*, *Salmonella*, and *Campylobacter* cause foodborne infections.

- *Foodborne toxicoinfections* occur when the pathogens produce harmful or deadly toxins while multiplying in human intestinal tracts. It is these toxic byproducts and not the pathogens themselves that cause human illnesses. In this report, two pathogens that cause foodborne intoxication are examined: *Clostridium perfringens* and *E. coli* O157:H7.

- *Foodborne intoxications* are caused by consuming food that contains either toxins released during the growth stages of specific bacteria (*e.g.*, enterotoxins produced by *Staphylococcus aureus*, a pathogen included here) or mycotoxins produced by molds. Illnesses from foodborne intoxications tend to occur quickly after consumption, because they do not involve any establishment or growth stage in the human body.

The CAST report (1994, p. 27) identifies four main categories of factors that increase the risk or severity of a foodborne illness: **microbial factors** such as the type, strain, and quantity of pathogens or toxins ingested; **host factors** such as age, stress, health of the individual's immune system, and personal hygiene; **diet-related factors** such as consumption of antacids and nutritional deficiencies; and **other factors** such as geographical location.

Most cases of foodborne illnesses are classified as acute, because they have a rapid onset and are self-limiting. Acute foodborne illnesses can be mild or severe and may result in premature death. Common acute symptoms of foodborne illnesses are gastrointestinal problems and vomiting.

Table 2—Estimated annual U.S. cases, deaths, and percentage foodborne for selected bacterial pathogens, 1993

Pathogen	Total cases	Total deaths	Percent foodborne	Foodborne cases	Foodborne deaths
	- - - - - - - -Number- - - - - - - -		*Percent*	- - - - - - - -Number- - - - - - - -	
Campylobacter jejuni or *coli*	2,500,000[f]	200-730[f]	55-70[h]	1,375,000 - 1,750,000	110-511
Clostridium perfringens	10,000[b]	100[b]	100[b]	10,000	100
Escherichia coli O157:H7	10,000-20,000[a]	200-500[a]	80[a]	8,000 - 16,000	160-400
Listeria monocytogenes	1,795-1,860[d]	445-510[d]	85-95[e]	1,526-1,767	378-485
Salmonella (non-typhoid)	800,000-4,000,000[bc]	800-4,000[bc]	87-96[bg]	696,000 - 3,840,000	696-3,840
Staphylococcus aureus	8,900,000[b]	7,120[b]	17[b]	1,513,000	1,210
Total	12,221,795-15,431,860	8,865-12,960	N/A	3,603,526 - 7,130,767	2,654-6,546

N/A = Not applicable.

Sources: USDA, Economic Research Service, based on:

[a] Personal communication with researchers at the U.S. Centers for Disease Control and Prevention, and from the article: American Gastroenterological Association, "Consensus Conference Statement on *E. coli* O157:H7 Infections, An Emerging National Health Crisis." July 11-13, 1994. *Gastroenterology* 108(1995):1923-1934.

[b] Bennett, J.V., S.D. Holmberg, M.F. Rogers, and S.L. Solomon. "Infectious and Parasitic Diseases." R.W. Amler and H.B. Dull (eds.), *Closing the Gap: The Burden of Unnecessary Illness*. New York: Oxford University Press, 1987.

[c] Helmick, C.G., P.M. Griffin, D.G. Addiss, R.V. Tauxe, and D.D. Juranek. "Infectious Diarrheas." Chapter 3 in Everhart, J.E. (ed.), *Digestive Diseases in the United States: Epidemiology and Impact*. U.S. Dept. Health and Human Serv., NIH, NIDDKD, NIH Pub. No. 94-1447, 1994, pp. 85-123.

[d] Roberts, T., and R. Pinner. "Economic Impact of Disease Caused by *Listeria monocytogenes*." In Miller, A.J., J.L. Smith, and G.A. Somkuti (eds.), *Foodborne Listeriosis*. Amsterdam, The Netherlands: Elsevier Science Publishing Co., Inc. 1990, pp. 137-149.

[e] Schuchat, Anne. CDC, personal communication with T. Roberts at the FDA Science Forum on Regulatory Sciences, Washington, DC, Sept. 29, 1994.

[f] Tauxe, R.V. "Epidemiology of *Campylobacter jejuni* Infections in the United States and other Industrialized Nations." Chapter 2 in Nachamkin, Blaser, and Tompkins (eds.), *Campylobacter jejuni*: Current Status and Future Trends, Washington, DC: American Assoc. of Microbiology, 1992, pages 9-19.

[g] Tauxe, R.V. and P.A. Blake. 1992. "Salmonellosis." Chapter 12 in Last, J.M., R.B. Wallace, and E. Barrett-Conner (eds.), *Public Health & Preventive Medicine*, 13th ed., Norwalk, Connecticut: Appleton & Lange, pp. 266-268.

[h] Tauxe, R.V., N. Hargrett-Bean, C.M. Patton, and I.K. Wachsmuth. "*Campylobacter* Isolates in the United States, 1982-1986," *Morbidity and Mortality Weekly Report*, 31,SS-2(1988):1-14.

Table 3—Pathogen food reservoirs/transmission and possible acute symptoms and chronic complications

Pathogen	Food sources	Acute symptoms and chronic complications[1]
Campylobacter jejuni or *coli*	Major: poultry Minor: milk, mushrooms, clams, hamburger, water, cheese, pork shellfish, eggs, cake icing.	Acute: abdominal pain, diarrhea (sometimes bloody), fever, malaise, vomiting. Chronic: appendicitis, arthritis, carditis, cholecystitis, colitis, endocarditis, erythema nodosum, Guillain-Barré syndrome, hemolytic-uremic syndrome, meningitis, pancreatitis, Reiter syndrome, septicemia, urinary tract infection.
Clostridium perfringens	Major: meat, meat stews, meat pies, and beef, turkey and chicken gravies. Minor: beans, seafood.	Acute: diarrhea, nausea Chronic: gas gangrene, necrotizing enteritis.
Escherichia coli O157:H7	Major: beef particularly ground beef. Minor: poultry, apple cider, raw milk, vegetables, cantaloupe, hot dogs, mayonnaise, salad bar items.	Acute: abdominal pain, diarrhea, fever, malaise. Chronic: erythema nodosum, hemolytic uremic syndrome, chronic kidney disease, thrombotic thrombocytopenic purpura, seronegative arthropathy.
Listeria monocytogenes	Major: soft cheese, pâté, ground meat. Minor: poultry, dairy products, hot dogs, potato salad, chicken, seafood, vegetables.	Acute: fever, severe headache, vomiting, sometimes delirium or coma. Chronic: chronic neurological complications, endocarditis, granulomatous lesions in organs, internal or external abscesses, meningitis, sepsis, septicemia.
Salmonella (non-typhoid)	Major: poultry, meat, eggs, milk, and their products. Minor: vegetables, fruits, chocolate, peanuts, shellfish.	Acute: abdominal pain, bloody stools, cold chills, dehydration, diarrhea, exhaustion, fever, headache, and sometimes vomiting. Chronic: abscesses, aortitis, arthritis, cholecystitis, colitis, endocarditis, epididymo-orchitis, meningitis, myocarditis, pericarditis, pneumonia, proderma or pyelonephritis, rheumatoid syndromes, septicemia, reactive arthritis, Reiter syndrome, splenic abscesses, thyroiditis.
Staphylococcus aureus	Major: workers handling foods: meat (especially sliced meat) poultry, fish, canned mushrooms. Minor: dairy products, prepared salad dressing, ham, salami, bakery items, custards, cheese.	Acute: severe nausea, cramps, vomiting, prostration, often with diarrhea. Chronic: none identified to date.

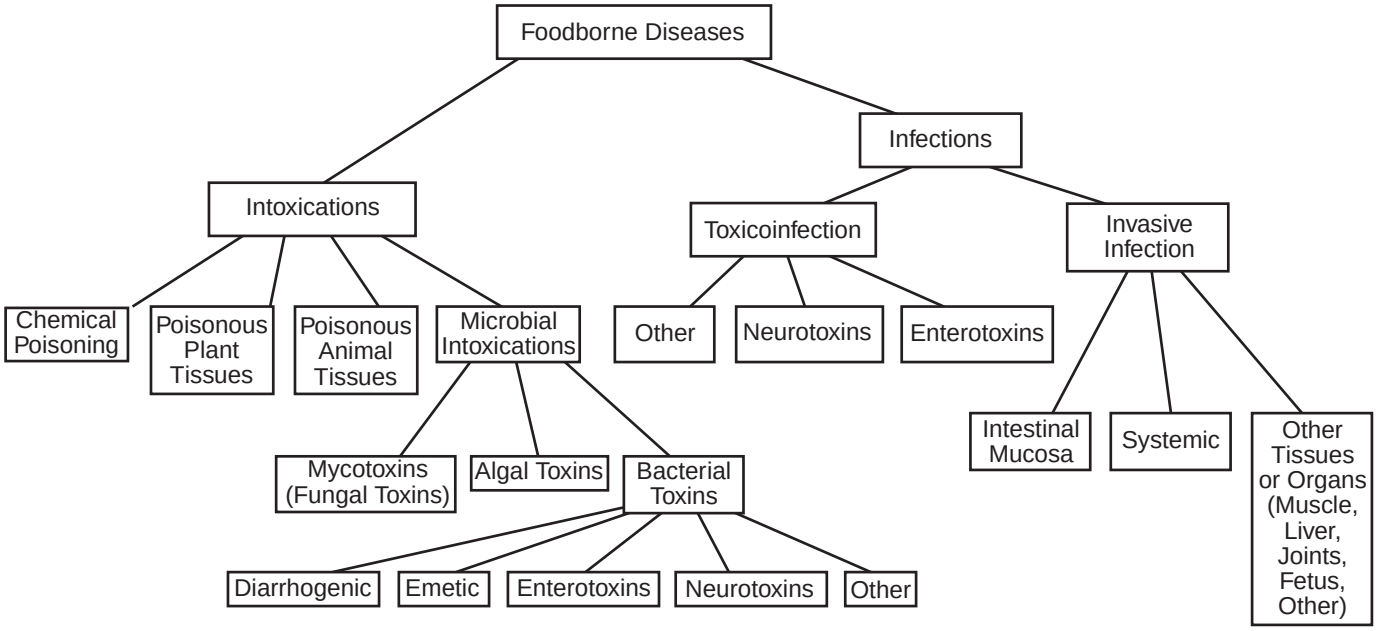
[1]USDA, Economic Research Service, adapted from:

Bean, N. H., P. M. Griffin, J. S. Goulding, and C. B. Ivey. "Foodborne Disease Outbreaks, 5 Year Summary, 1983-1987," *CDC Surveill. Summ. Morb. Mort. Weekly Rep.* (MMWR) 39, SS-1(March 1990):15-59 and *J. Food Prot.* 53(1990):711-728.

Benenson, Abram S. Ed., *Control of Communicable Diseases in Man.* Amer. Public Health Assoc., 15th edition, 1990.

CAST Report, "Foodborne Pathogens: Risks and Consequences," Task Force Report No. 122, Washington, DC: Council for Agricultural Science and Technology, Sept. 1994.

Figure 1
A Classification of foodborne disease causes



Source: CAST report, Figure 2.1, 1994 (adapted from Bryan, 1982).

Archer and Kvenberg (1985) estimate that 2-3 percent of these acute cases develop secondary long-term illnesses or chronic sequelae. Chronic sequelae of foodborne illnesses can occur in any part of the body and include rheumatoid, cardiac, and neurological syndromes (table 3). These chronic illnesses may afflict the patients for the remainder of their lives or cause premature death. For example, reactive arthritis and Guillain-Barré syndrome (a major cause of non-trauma neuromuscular paralysis in the United States) may follow *Campylobacter* infections.

Traditionally, only acute cases of some foodborne diseases have been recorded. Improved collection and documentation of data on foodborne illnesses may increase our understanding of the magnitude of chronic sequelae and show that the longrun effects of chronic illnesses are often greater than the initial acute illnesses.

Regulation

Food safety regulations reduce human illnesses through preventing and controlling the presence and amount of foodborne pathogens and other disease-causing elements (*e.g.*, pesticides) in food. In making food safety policy decisions, the Federal Government relies, in part, on economic information of foodborne illnesses and alternative regulatory programs that reduce foodborne health risks (*i.e.*, increase food safety). A comparison of societal benefits and costs among programs aimed at reducing different pathogens can facilitate setting priorities as to which pathogens should be targeted first. Estimates and comparisons of the benefits and costs of competing programs can also help the Federal Government efficiently allocate tax dollars.

A case in point is the Pathogen Reduction Program proposed in 1994 by the Food Safety and Inspection Service (FSIS) of the USDA. As part of this program, FSIS promulgated a Hazard Analysis Critical Control Point (HACCP) system to improve the current meat and poultry inspection. A portion of the COI estimates from this document plus the estimated annual costs of illness caused by *Toxoplasma gondii*, a parasite, provide the foundation of the estimated benefits of HACCP (*Federal Register* Feb. 3, 1995). Preliminary results indicate that the benefits of implementing HACCP outweigh the costs.

Table 4 outlines societal costs of foodborne illnesses. The three cost categories are costs incurred by individuals/households, industry, and the regulatory and public health sector. Note that traditional COI analyses often include only individual's/household's medical costs and cost of lost productivity. Other costs are usually omitted due to lack of suitable measures.

The costs of food safety regulation include expenditures associated with design and implementation of, and compliance with, such programs. In fiscal year 1994, the Federal Government budgeted $1.2 billion on food safety regulatory activities, such as inspection and laboratory testing (GAO March 1996). The food industry also incurs millions of dollars of expense to comply with food safety rules and regulations. If new regulations are added to the current system, industry compliance costs will be higher.

Societal benefits of food safety regulation arise from improvement of individuals' health status. From an economic perspective, these benefits include, at least, savings in disease prevention and mitigation expenditures, increases in worker productivity, reduction in pain and suffering, and reduction in anxiety about foodborne health risk.

ERS COI estimates represent the maximum benefits that could be obtained if the microbial infections or intoxications were eliminated or reduced. However, eradication of these illnesses is neither technically nor economically feasible at present. The information contained in this report can help Federal agencies identify the most cost-beneficial risk-reduction strategies for bacterial pathogens.

Overview of the Willingness-to-Pay and Cost-of-Illness Methods

Three principles guide economic analysis of regulations aimed at improving health and safety. The first is that benefits from the regulation need to be measured and compared with costs, because regulatory costs are opportunity costs. That is, the resources used could have been applied elsewhere, with potentially greater health benefits. For example, an expenditure of $100 million that is expected to prevent 4 deaths may not be very sensible if that $100 million could have prevented 50 deaths by being spent in another application.

The second principle asserts that health and safety regulations typically do not aim to save the lives of specific people who would otherwise die, but rather aim at reducing the level of risk of illness and death faced by large populations. That view intertwines with the third principle, that the benefits of a regulation do not represent the value of keeping a specific person alive, but rather the value of reducing those risks. To this end, the most theoretically appropriate way to value a risk reduction is to ask what affected individuals are willing to pay for it. However, as we shall see here, estimating the costs of an illness by summing estimated medical costs and costs of lost earnings is useful for studying specific policy questions.

Taken together, the principles recognize that regulators act on behalf of society to reduce societal risk by spending taxpayers' money and by setting and enforcing regulations. In other contexts, people spend their own money to reduce health risks (*e.g.*, through regular physician visits, or through diet control, or when choosing among different brands of durable goods like cars, household appliances, or power equipment). Once it is recognized that regulation delivers an outcome (small risk reductions) that people also purchase in other public and private venues, one can ask whether publicly delivered risk reductions appear to be worth it to the relevant populations, based on what they are willing to expend to achieve risk reductions in other contexts.

Economic theory provides a precise framework that associates the benefits of risk reduction with the amount that people are willing to pay to achieve the reduction, and suggests methods of measuring those benefits (Just *et al*. 1982). As is often the case, some key theoretical constructs cannot be observed; as a result, practical applications of the theory aim at approximations of the theoretically appropriate measures. The practical applications for human illnesses can be grouped into two primary methods of benefit estimation, the cost-of-illness (COI) method and the willingness-to-pay (WTP) method. The WTP method aims, as the name implies, to estimate the value that individuals place on reductions in risk to identify the value to society of publicly provided risk reduction.

Table 4—Societal costs of foodborne illness

Costs to individuals/households[1]
Human illness costs:
- Medical costs—
  - Physician visits
  - Laboratory costs
  - Hospitalization or nursing home
  - Drugs and other medications
  - Ambulance or other travel costs
- Income or productivity loss for—
  - Ill person or person dying
  - Caregiver for ill person
- Other illness costs—
  - Travel costs to visit ill person
  - Home modifications
  - Vocational/physical rehabilitation
  - Child care costs
  - Special educational programs
  - Institutional care
  - Lost leisure time
- Psychological (psychic) costs—
  - Pain and other psychological suffering
  - Risk aversion

Averting behavior costs—
- Extra cleaning/cooking time costs
- Extra cost of refrigerator, freezer, etc.
- Flavor changes from traditional recipes (especially meat, milk, egg dishes)
- Increased food cost when more expensive but safer foods are purchased

Altruism (willingness to pay for others to avoid illness)

Industry costs[2]
Costs of animal production:
- Morbidity and mortality of animals on farms
- Reduced growth rate/feed efficiency and increased time to market
- Costs of disposal of contaminated animals on farm and at slaughterhouse
- Increased trimming or reworking at slaughterhouse and processing plant
- Illness among workers because of handling contaminated animals or products
- Increased meat product spoilage due to pathogen contamination

Control costs for pathogens at all links in the food chain:
- New farm practices (age-segregated housing, sterilized feed, etc.)
- Altered animal transport and marketing patterns (animal identification, feeding/watering)
- New slaughterhouse procedures (hide wash, knife sterilization, carcass sterilizing)
- New processing procedures (pathogen tests, contract purchasing requirements)
- Altered product transport (increased use of time/temperature indicators)
- New wholesale/retail practices (pathogen tests, employee training, procedures)
- Risk assessment modeling by industry for all links in the food chain
- Price incentives for pathogen-reduced product at each link in the food chain

Outbreak costs:
- Herd slaughter/product recall
- Plant closings and cleanup
- Regulatory fines
- Product liability suits from consumers and other firms
- Reduced product demand because of outbreak:
  - Generic animal product - all firms affected
  - Reduction for specific firm at wholesale or retail level
- Increased advertising or consumer assurances following outbreak

See footnotes at end of table.

--Continued

Table 4—Societal costs of foodborne illness--Continued

Regulatory and public health sector costs for foodborne pathogens

Disease surveillance costs to:
- Monitor incidence/severity of human disease by foodborne pathogens
- Monitor pathogen incidence in the food chain
- Develop integrated database from farm to table for foodborne pathogens

Research to:
- Identify new foodborne pathogens for acute and chronic human illnesses
- Establish high-risk products and production and consumption practices
- Identify which consumers are at high-risk for which pathogens
- Develop cheaper and faster pathogen tests
- Risk assessment modeling for all links in the food chain

Outbreak costs:
- Costs of investigating outbreak
- Testing to contain an outbreak (for example, serum testing and administration of immunoglobulin in persons exposed to Hepatitis A)
- Costs of cleanup
- Legal suits to enforce regulations that may have been violated[3]

Other considerations:
- Distributional effects in different regions, industries, etc.
- Equity considerations, such as special concern for children

[1] Willingness-to-pay estimates for reducing risks of foodborne disease is a comprehensive estimate of all these categories (assuming that the individuals have included employer-funded sick leave and medical programs in their estimates). The estimate is comprehensive and covers reduced risks for everyone—those who will become ill as well as those who will not.

[2] Some industry costs may fall with better pathogen control, such as reduced product spoilage, possible increases in product shelf-life, and extended shelf-life permitting shipment to more distant markets or lowering shipment costs to nearby markets.

[3] In adding up costs, care must be taken to assure that product liability costs to firms are not already counted in the estimated pain and suffering cost to individuals. However, the legal and court expenses incurred by all parties are societal costs.

Source: USDA, Economic Research Service, based on Roberts, Tanya, and Ewen Todd. "Approaches to Estimating the Cost of Foodborne Disease," *WHO Consultation on the Economic Implications of Animal Production Food Safety.* Washington, DC, June 8-10, 1995.

The COI approach can be thought of as measuring the costs of an illness to the measured economy, via effects on current and future Gross Domestic Product. In brief, COI measures the sum of medical expenses, forgone earnings of affected individuals, and productivity losses to employers of affected individuals on paid sick leave. The important advantage of a COI measure is that it employs readily available and reliable data. Also, these relevant data are precise enough to allow for sensitivity analyses of the response of the measure to changes in medical costs, productivity losses, and disease severity categories of affected individuals. Because they are so tractable, COI measures have been widely used for several decades.

For some human illnesses, patients die prematurely or are unable ever to return to work. In a COI analysis, the lost productivity for these patients can be represented either by human capital estimates of forgone earnings or by WTP estimates of the value of a statistical life. Therefore, the COI method can partially incorporate the WTP measure for these categories of patients.

In general, COI methods aim at calculating the costs of illness to the measured economy. That is not the same thing as calculating the valuation that people place on reductions in risk, because forgone earnings are not necessarily a good indicator of that valuation. Some authors (Harrington and Portney 1987) argue that COI can serve as a lower bound estimate for willingness to pay.

Taken literally, human capital estimates of forgone earnings suggest that the method places little value on reducing risk of the elderly, because they have low future earnings to forgo. Similarly, the method typically attaches rather low values to risk reduction for children, because future earnings are discounted to present values. Depending on the discount rate

used, the present value of children's future earnings can be quite small. Common observation suggests that assigning low values for reducing risks to children and the elderly is not a good approach, since we can observe people spending substantial amounts on risk avoidance for those groups, especially children.

More generally, the COI approach seems to be crudely "economic" in the sense that it values lost income and the associated consumption expenditures; but in fact the approach does not conform with economic theory because it fails to recognize the value that individuals may place on (and pay for) feeling healthy, avoiding pain, or using their free time. Because the COI approach explicitly ignores these valuable aspects of health, the method is generally thought to understate the true societal benefits from risk reduction.

While there are several methods that attempt to isolate willingness to pay, recent attention in estimating the value of a statistical life has focused on one, hedonic wage estimation.[3] Hedonic wage studies derive the value of risk reduction by statistically estimating the effect of occupational mortality and injury risks on wages. Typically, employers must offer workers higher wages to induce them to take a job with some injury risks, as opposed to a similar job with no such risks. Conversely, workers accept jobs with lower wages, given that those jobs offer minimal risks. The "risk premium" is then the increased wage needed to attract workers to riskier jobs. Economists began to use statistical methods to estimate typical risk premiums in the 1970's, in analyses that could control for other factors that influenced wages, such as education, experience, and location.

Economists are partial to the hedonic wage method, because it uses actual choices made in response to differing risk environments. However, early applications of the method were controversial for reporting widely varying values for risk premiums. With the development of more precise risk measures, better understanding of the affected populations in the studies, and better control variables, analysts have been able to generate a narrower range of values, leading to greater acceptance of hedonic wage models in recent years.

The typical hedonic wage study uses a measure of mortality risk, and measures the effects of a change in mortality risk on wages; a typical study might find that an increase in mortality risk of 1 in 10,000 (one extra death in a year for every 10,000 workers in the relevant population) would be associated with a wage increase of $300. In an industry with 10,000 workers, then, we could expect one additional worker to die each year on average, and as a result total wage payments would be $3 million higher ($300 times 10,000 workers). In that case we would say that the value of a statistical life was estimated to be $3 million, because industry had to pay that amount to induce workers to take on a risk that would likely leave one dead (alternatively, one could in this analysis say that workers would in the aggregate be willing to pay $3 million, through wage reductions, to purchase a reduction in risk).

Note the emphasis above on our second principle, that what we purchase in these cases are small reductions in health risks. Analysts often carelessly refer to the "value of a life" derived from these studies, which is easily confused with the crude idea that economic analyses associate the value of a life with lifetime earnings. But that is not what economic theory describes as the appropriate measure, nor is it what willingness-to-pay studies seek to uncover. They seek to measure the value that individuals place on small reductions in risk, a value likely to be only loosely related to income.

Does method matter? Apparently so. Cost-of-illness studies, as shown in more detail below, estimate separate values of forgone earnings for illnesses and for deaths. COI aggregate estimates are usually dominated by the forgone earnings associated with premature deaths; typical values of forgone earnings vary between methods. The Landefeld and Seskin value of statistical life varies, depending on age, from $11,867 to $1,584,605 in 1993 dollars. However, hedonic wage studies suggest that employed people would be willing to pay between $3 million and $7 million (1990 dollars) to reduce the risks generating each additional death (Viscusi 1993). As a result, the COI method is likely to give extremely conservative benefit estimates for publicly provided risk reduction.

[3]Fisher *et al.* (1989, p. 89) divide the WTP approach into three categories of studies: (1) contingent market studies, (2) consumer market studies, and (3) wage-risk studies.

Our example shows how hedonic wage studies use the wage response to mortality risks to generate estimates for the value of a statistical life. The exercise points out one current weakness of hedonic wage studies; these studies have much better data, and more useable estimates, for mortality risks than they do for morbidity risks (*i.e.*, risk of temporary or chronic illness). Even where morbidity risks are incorporated, it is questionable how closely risk aversion to the job-related illnesses resembles risk aversion to illnesses associated with foodborne pathogens. If we base estimated values of risk reduction on hedonic wage studies only, we may make the error of understating the value of the risk reduction because these studies focus on mortality risks and often implicitly ignore morbidity risks.

Cost-of-Illness (COI) Method

This section outlines the basic framework behind the six COI analyses presented here. Due to differences in available data and differences in chronic sequelae examined for each bacterial pathogen, these six analyses vary in depth, though all basically look at both medical costs and the costs of lost productivity from the illnesses.

Incidence

The first step in any COI analysis is to determine the incidence of a specific illness. Incidence rates are often expressed as the number of new cases of a disease per 100,000 individuals in the U.S. population in a 1-year period.[4] The quantification of foodborne disease incidence is a matter of great controversy because of uncertainties over the true state of the world (CAST 1994; Roberts and Foegeding 1991). The enumeration of a case of a foodborne disease depends on whether: (1) the affected individual recognizes food as the cause of the illness, (2) a physician is consulted, (3) a hospital is sought for treatment, (4) the physician recognizes the illness as foodborne, (5) the laboratory identifies a foodborne pathogen, and (6) the case is reported to the CDC.

Because the nature and reporting of foodborne diseases result in vast under-counting of the actual incidence of illnesses, incidence rates are often estimated by expert opinion.

There are five main data sources for incidence of acute foodborne illnesses: (1) national surveys or databases such as those conducted and published by the National Center for Health Statistics (NCHS), (2) CDC data ranging from reports of foodborne disease to active surveillance studies, (3) risk models based on pathogens' prevalence in foods, and on infectious doses, (4) medical data on individual cases, often published in the literature as a case history, and (5) extrapolations by experts to obtain estimates of the total number of cases and the disease severity distributions (CAST 1994, p. 40).[5] Extrapolations are upward adjustments made to account for those cases that go undiagnosed or unreported. Estimates from these data sources form the basis for some of the COI studies of foodborne diseases. Where data were available to suggest a range of cases and/or deaths, ranges were used.

Costs

Given the incidence of an illness, we computed annual medical costs and costs of lost productivity. Where possible, we considered both acute and chronic illnesses.

In general, medical costs include physician and hospital services, supplies, medications, and special procedures required for a specific foodborne illness. Hospitalization accounts for a large proportion of these costs. Estimates of medical costs come from nationwide databases such as the published Medicare reimbursement rates and per capita expenditures on physician services from the Health Care Financing Administration (HCFA), the American Hospital Association's Hospital Statistics, and the National Center for Health Statistics' National Hospital Discharge Survey (NHDS) and National Mortality Follow-back Survey.

In general, productivity loss measures the decline in production (output) because workers were ill and either missed work, performed poorly at work, were unable ever to return to work, or died prematurely.

[4]To facilitate comparison of the relative occurrence of different pathogens, incidence rates are used rather than prevalence rates. The *incidence* of a disease signifies the number of new cases occurring during a year in the United States. *Prevalence* measures the number of people sick, regardless of when the disease began, at a given point of time or over a period of time which may vary by pathogen. For decisionmaking with respect to preventative programs (such as food safety regulations), incidence rates are the appropriate statistics (Hartunian *et al.* 1980, p. 1249).

[5]Bennett *et al.* (1987) estimated the proportion of infections, for known categories, acquired through food.

Productivity losses for those who died or were unable to return to work were calculated differently from those who missed some work but later resumed work.

For those cases in which work is interrupted temporarily, the productivity loss is the product of time lost from work multiplied by the corresponding wage rate. The daily wage of an individual is frequently used in economic studies as a proxy for the value of output produced in a day's work. When data are not available on time lost from work due to illness, this lost time is estimated by assuming a typical ratio of time spent in the hospital to time lost from work. Time spent by parents, as well as payments to paid caretakers, caring for sick children may also be included as forgone productivity.

In this report, estimated productivity losses for those who die or were unable to return to work were based on Landefeld and Seskin's (LS) (1982) human capital/WTP measure. We used the LS estimates directly in the first four COI analyses (salmonellosis, listeriosis, *E. coli* O157:H7 disease, campylobacteriosis). In the remaining two COI analyses (*Staphylococcus aureus* intoxications and *Clostridium perfringens* intoxications), we extrapolated COI estimates from other analyses that used LS estimates directly.[6]

The LS method combines elements of both the human capital and WTP methods to generate the present value of expected lifetime after-tax income and housekeeping services. The LS method generates the present value of expected lifetime after-tax income and housekeeping services at a 3-percent real rate of return, adjusted for an annual 1-percent increase in labor productivity and a risk aversion factor of 1.6. The risk aversion factor is based on the ratio of life insurance premium payments to life insurance loss payments. In most cases, life insurance premiums represent "household WTP for potential losses associated with the death of an income-earning household member" (Landefeld and Seskin 1982, p. 562). The LS value of a statistical life lost is:

$$VOSL = \left[\sum_{t=0}^{T} \frac{Y_t}{(1 + r)^t}\right]\alpha \qquad (1)$$

where T = remaining lifetime, t = a particular year, Y_t = after-tax income including labor and nonlabor income, r = household's opportunity cost of investing in risk-reducing activities, and α = risk aversion factor. Table 5 provides estimates of the value of statistical life using LS estimates, after averaging across gender, interpolating between the LS's 4-year age groups, and updating to 1993 dollars.

General Framework for the Six COI Analyses

The general framework for the first two COI analyses presented here (salmonellosis and campylobacteriosis) used the following classification system: all cases were first divided into four severity categories for acute illness, and costs were then applied to each of the categories. The four severity categories were those who: did not visit a physician, visited a physician, were hospitalized, or died prematurely. Costs were summed over the four categories to calculate total costs. Medical costs and costs of lost productivity were not calculated separately.[7] The COI analyses for salmonellosis and campylobacteriosis did not consider chronic complications.

The general framework for the third and fourth COI analyses presented here (*E. coli* O157:H7 disease and listeriosis) was more inclusive than the COI analyses for salmonellosis and campylobacteriosis, because some chronic complications were considered. In the *E. coli* O157:H7 disease and listeriosis COI analyses, we first calculated medical costs and then calculated lost productivity costs. For each of these cost categories, acute and chronic cases were considered separately. We used the four severity subcategories for acute illness from the previous two analyses (*i.e.*, those who: did not visit a physician, visited a physician, were hospitalized, or died). After costs in each subcategory were estimated, total costs were calculated. For chronic cases, the lifetime course of disease and associated costs were estimated.

The remaining two COI analyses (*Staphylococcus aureus* intoxications and *Clostridium perfringens* intoxications) were more simplistic in that estimates

[6]As previously mentioned, this study updates the annual COI of *Staphylococcus aureus* and *Clostridium perfringens* intoxications from Roberts (1989) to 1993 dollars and uses more recent estimates of the numbers of cases and deaths. Roberts (1989) used the LS estimates in this fashion and we continue the practice.

[7]These COI analyses rely heavily on data from Cohen *et al.* (1978). Their data for the four disease severity categories did not distinguish between medical costs and costs of lost productivity but instead made vague comments such as "could mainly be attributed to medical care." Rather than impose assumptions about what percentage of total costs were attributed to medical costs and costs of lost productivity, we did not separate these two types of costs.

Table 5 — Estimates of the value of a statistical life

Individual age	Annual interpolated value	Individual age	Annual interpolated value	Individual age	Annual interpolated value
0	$1,097,792	28	$1,506,486	58	$401,484
0.5	$1,112,617	29	$1,447,742	59	$365,960
1	$1,127,442	30	$1,448,998	60	$330,436
2	$1,157,093	31	$1,420,254	61	$294,911
2.5	$1,171,918	32	$1,391,510	62	$259,387
3	$1,184,108	33	$1,361,425	63	$237,002
4	$1,208,488	34	$1,331,340	64	$214,617
5	$1,232,869	35	$1,301,255	65	$192,232
6	$1,257,249	36	$1,271,170	66	$169,847
7	$1,281,630	37	$1,241,085	67	$147,462
8	$1,308,213	38	$1,205,340	68	$136,672
9	$1,334,796	39	$1,169,594	69	$125,881
10	$1,361,379	40	$1,133,849	70	$115,090
11	$1,387,962	41	$1,098,104	71	$104,300
12	$1,414,546	42	$1,062,358	72	$93,509
13	$1,438,530	43	$1,018,773	73	$86,870
14	$1,462,514	44	$975,188	74	$80,231
15	$1,486,497	45	$931,602	75	$73,592
16	$1,510,481	46	$888,017	76	$66,953
17	$1,534,465	47	$844,431	77	$60,314
18	$1,544,493	48	$803,427	78	$56,302
19	$1,554,521	49	$762,423	79	$52,290
20	$1,564,549	50	$721,418	80	$48,278
21	$1,574,577	51	$680,414	81	$44,265
22	$1,584,605	52	$639,410	82	$40,253
23	$1,574,730	53	$598,929	83	$34,576
24	$1,564,855	54	$558,449	84	$28,899
25	$1,554,980	55	$517,969	85	$23,221
26	$1,545,105	56	$447,489	86	$17,544
27	$1,535,229	57	$437,009	87	$11,867

Source: Compiled by USDA's Economic Research Service, based on Landefeld and Seskin (LS) estimates. LS are divided by gender and use four-year age groups. Here, these 1977 estimates are updated to 1993 dollars, averaged across gender, and interpolated within age groups.

of the annual number of cases were multiplied by estimates of the average annual costs provided by Roberts (1989) and updated to 1993 prices. Roberts (1989) did not report medical costs and costs of lost productivity separately (or acute and chronic cases separately), and therefore they are not reported separately here.

COI Estimates of Salmonellosis

Salmonella is the main cause of documented foodborne human illnesses in most developed countries (CAST 1994, pp. 16, 32). Although there are over 2,000 *Salmonella* serotypes,[8] only around 200 serotypes are detected in the United States annually (Benenson 1990, p. 382). Most strains of *Salmonella* are from the *S. enteritidis* species and these strains are traditionally classified by their serotype designation and not by their species name (Helmick et al. 1994, p. 104). For example, the serotype *typhimurium* is found among the *S. enteritidis* species, yet is referred to as *S. typhimurium* (Helmick et al. 1994, p. 104). The 10 most common serotypes are responsible for over 70 percent of the U.S. human illnesses (Helmick *et al.* 1994, p. 104). Human illness due to *Salmonella* infections is most commonly caused by *S. typhimurium*, *S. enteritidis*, or *S. heidelberg* serotypes in the United States (Merck 1992).[9]

Typically, *Salmonella*-caused human disease is limited to salmonellosis, an acute gastroenteritis. After eating contaminated food, salmonellosis generally appears in 6 to 74 hours with an average incubation period of 12 to 36 hours (Benenson 1990, p. 383). Salmonellosis may cause only mild abdominal discomfort, with diarrhea lasting less than a day. Most people who become ill in salmonellosis outbreaks believe they have the stomach flu, not salmonellosis (Tauxe, 1987, personal communication with Roberts). Other symptoms may include dehydration, fever, headache, nausea, stomachache, and sometimes vomiting (Benenson 1990). In rare cases, blood may be present in the stools.

Salmonella infections, like many other bacterial and parasitic infections, can cause secondary-disease syndromes, some of which may be chronic illnesses (Archer 1984 and 1985; Mossel 1988) (table 3). Most *Salmonella* serotypes can penetrate the intestinal lining in humans without advancing deep into other tissues (CAST 1994, p. 17). Infrequently, the organism may invade the bloodstream causing bacteremia or septicemia, with potentially deadly results. Some complications of septicemia include endocarditis (infection of the heart), meningitis (infection of the brain or spinal tissues), and pneumonia (Merck 1992). Deaths are uncommon.

Most human salmonellosis comes from eating contaminated food (Helmick *et al.* 1994, p. 107), especially food from animal origin (Tauxe 1991, p. 565). Tauxe and Blake (1992, p. 267) found that 87 percent of all salmonellosis cases were foodborne (100 percent minus 10 percent person-to-person and 3 percent pets). Bennett *et al*. (1987, p. 109) estimated that, due to improvements in sanitation, nearly all (96 percent) salmonellosis infections are foodborne (3 percent waterborne and 1 percent from day care). We assume that 87 to 96 percent of all salmonellosis cases are foodborne.

Salmonella contamination occurs in a wide range of animal and plant products (table 3). Poultry products and eggs are frequently contaminated with *S. enteritidis*, while beef products are commonly contaminated with *S. typhimurium* (Merck 1992).[10] Other food sources of *Salmonella* may include raw milk or other dairy products and pork. *Salmonella* outbreaks also have been traced to contaminated vegetables, fruits, and marijuana (Helmick *et al.* 1994, p. 107). Tauxe reports that *Salmonella*-associated bacteremia is common in AIDS patients, some of whom consume raw milk, raw eggs, or raw beef under the mistaken belief that these products will improve their health (Tauxe 1991, p. 566).

Individuals vary greatly in their susceptibility to salmonellosis, depending partly on the virulence of the

[8]Webster's dictionary (1988) defines serotypes as "a group of related microorganisms distinguished by its composition of antigens."

[9]S. typhi, the cause of typhoid fever in the United States between the late 1800's and 1949, used to be the chief serotype affecting humans in the United States (Tauxe 1991). Typhoid fever has been virtually eliminated in the United States through public health measures such as improved drinking water treatment and sewage disposal (Tauxe 1991). Therefore, this report covers non-typhoid Salmonella cases.

[10]S. enteritidis serotype enteritidis has emerged as a major food safety problem with shell eggs during the past decade because of genetic or other changes that permit the organism to get inside the egg. Infected hens transmit S. enteritidis to the egg as the egg is produced (Tauxe 1991, p. 567). This has resulted in an epidemic of S. enteritidis infections and a corresponding shift in the overall ranking of serotypes. Salmonella outbreak investigations have shown that the most common source of infections are grade A shell eggs (Helmick et al. 1994, p.104).

serotype and partly on the individual's immune system, along with other factors such as the quantity of *Salmonella* ingested. The infectious dose may be as low as one colony forming unit (CFU) for some *Salmonella* serotypes (CAST 1994, p. 13; Archer and Young 1988, p. 380). Usually, ingestion of at least 10^{2-3} organisms are required for infection (Benenson 1990, p. 382).

Those most vulnerable to *Salmonella* infection and secondary complications are infants, the elderly, and the immunocompromised (*e.g.*, those with cancer, sickle cell anemia, and AIDS) (Helmick *et al.* 1994, p. 104). People who take oral antibiotics may have decreased colonization resistance to *S. typhimurium* (CAST 1994, p. 25). Riley *et al.* (1984, p. 878) suggest that those *Salmonella* serotypes that are resistant to antimicrobial agents depend more heavily on the characteristics of the host to cause human illness than do those serotypes sensitive to antimicrobial agents. Arthritis complications provoked by *Salmonella* infection are more likely to be found among those who are genetically predisposed (Helmick *et al.* 1994, p. 107). Ryan *et al.* (1987, p. 3271) found that children under 10 years of age were more likely to contract salmonellosis than people 10 years old or older.[11] They also found that those most severely affected out of a sample of culture-confirmed salmonellosis cases were children 1 to 4 years old (Ryan *et al.* 1987, p. 3271). Salmonellosis patients who are immunocompromised, very old, or very young face a higher risk of death (Benenson 1990, p. 381). Figure 2 shows *Salmonella* isolation rates by age and sex.

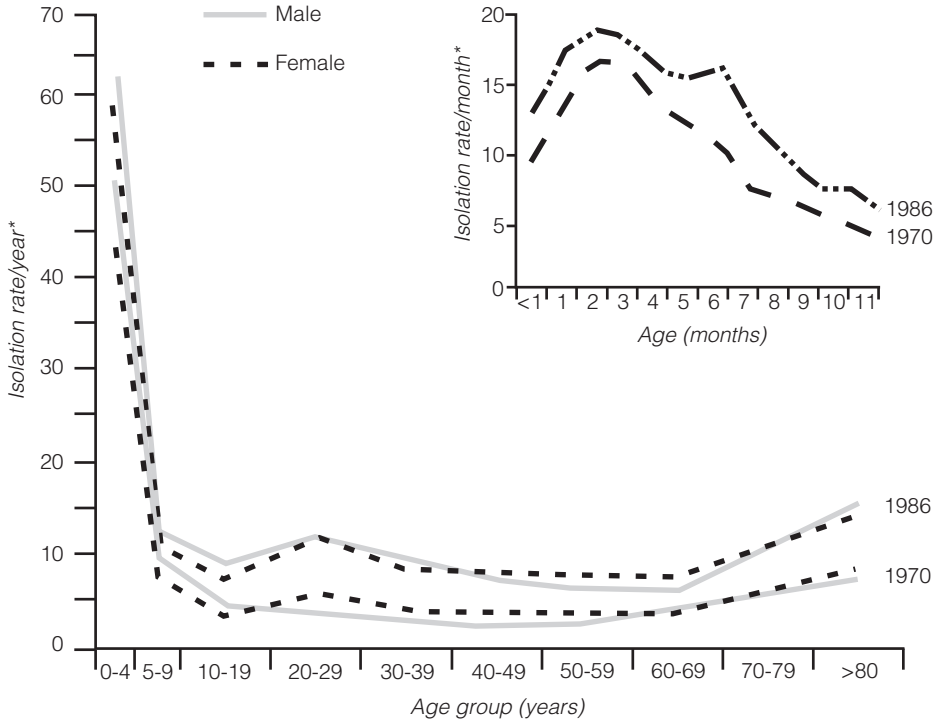
Estimates of Cases

Three basic sources of data can be used to estimate the number of annual salmonellosis cases: surveillance data, outbreak data, and extrapolations from sur-

[11]This result is based on a sample of persons with a salmonellosis-like illness after drinking contaminated milk. The data are from a survey of persons in two Illinois counties (April 14-25, 1985).

Figure 2

Salmonellosis rates, by age and sex of patient and year, United States, 1970 and 1986



* Per 100,000 population

Source: Hargrett-Bean N., A. T. Pavia, and R. V. Tauxe. "*Salmonella* Isolates from Humans in the United States, 1984-86" *Morbidity and Mortality Weekly Report,* 37, SS-2 (1988): 25-31.

veillance and outbreak data.[12] Since 1943, CDC's National Notifiable Diseases Surveillance System has recommended that States report salmonellosis cases (Helmick *et al.* 1994, p. 104). This means that, in participating States, physicians report salmonellosis cases to local health departments, local health departments report these cases to State health departments, and State health departments report total annual cases to CDC (Tauxe 1991, p. 564). Local health departments also investigate to determine if the cases are foodborne. This reporting results in a better estimate of the incidence of salmonellosis than for most other bacterial diseases. A second surveillance reporting system, introduced in 1963 where State public health laboratories report *Salmonella* isolates to CDC (Helmick *et al.* 1994, p. 104), has an even higher rate of salmonellosis positive tests.

Both surveillance data and outbreak are clearly underestimates. As previously mentioned, infection may not be suspected, cultured, diagnosed, or reported for a variety of reasons. Therefore, estimates of the "true" incidence of salmonellosis are generally extrapolated from these two types of data.

Chalker and Blaser (1988, p. 120) investigated various methods to calculate the annual number of *Salmonella* infections not reported. They estimated that each year, only 1-5 percent of all *Salmonella* infections are reported to CDC. Tauxe (1991, p. 564) found that in the late 1980's, the National *Salmonella* Surveillance System reported 40,000 to 45,000 *Salmonella* isolates each year. Using the low estimate of isolates (40,000) and Chalker and Blaser's multipliers,[13] the CDC's best estimate of human *Salmonella* infections annually in the United States is 800,000 to 4 million cases (Helmick *et al.* 1994, p. 104) (table 6).

This report uses a range of 800,000 to 4 million annual cases of salmonellosis to update Roberts' (1988) COI estimates for salmonellosis. As with each of the six foodborne illnesses discussed here, salmonellosis cases are divided into four severity categories, those who: do not seek medical attention, visit a physician, are hospitalized, and die prematurely. Both a low and a high cost estimate were calculated for each severity category. Table 6 presents the estimated U.S. salmonellosis cases by severity category. Figure 3 presents the distribution of estimated annual U.S. cases of salmonellosis and disease outcomes.

Table 6—Estimated U.S. salmonellosis cases, 1993

Severity of illness	Estimated cases	
	Low	High
	Number	
No physician visit[1]	746,880	3,734,400
Physician visit[2]	40,320	201,600
Hospitalized[3]	12,000	60,000
Deaths[4]	800	4,000
Total[5]	800,000	4,000,000

[1] Cases in this category were calculated as a residual.
[2] Assuming 5.04% of all cases visit a physician (Ryan 1987).
[3] This category is for those who were hospitalized and survived. Assuming 1.5% of all cases are hospitalized (Ryan *et al.* 1987).
[4] Deaths are calculated using a case fatality rate of 1/1,000. Those who die are assumed to be hospitalized prior to their deaths. Therefore, the total number of salmonellosis patients hospitalized each year is 12,800 in the low estimate and 64,000 in the high estimate.
[5] The low estimate of 800,000 cases was calculated by multiplying CDC's estimate of 40,000 *Salmonella* isolates (Tauxe 1991) by Chalker and Blaser's (1988) low estimate of the number (20) of unreported cases to each reported case. The high estimate of 4 million cases was calculated by multiplying CDC's estimate of 40,000 *Salmonella* isolates (Tauxe 1991) by Chalker and Blaser's (1988) high estimate of the number (100) of unreported cases to each reported case.

A 1984-85 survey found a 1.3-percent death rate for reported cases of salmonellosis (CDC memo from the Foodborne and Diarrheal Disease Branch, Oct. 31, 1994).[14] After adjusting for unreported cases, it is assumed that there is a 0.1 percent death rate for all salmonellosis cases. Applying this ratio to the estimated range of 800,000 to 4 million salmonellosis cases results in a range of 800 to 4,000 deaths occurring annually. This range of annual salmonellosis deaths was used in this report.

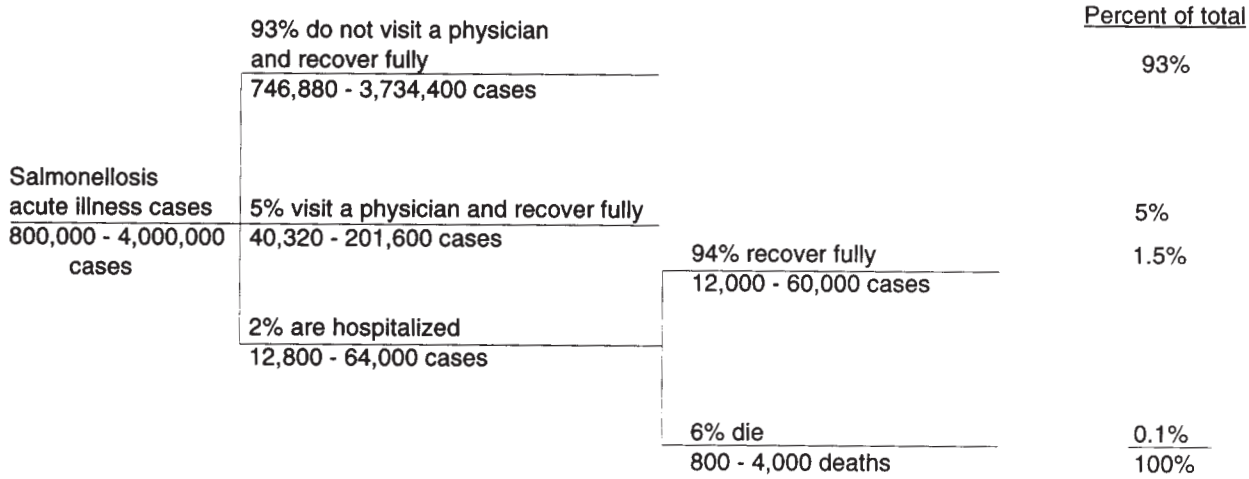
[12]In the United States, most *Salmonella* infections are believed to be sporadic cases as opposed to being associated with outbreaks (Feldman and Blaser 1980, p. 436).

[13]That is, the actual number of infections is 20 to 100 times larger than the 40,000 reported cases of salmonellosis.

[14]Death certificate data (Vital Statistics) reported to CDC show an average of 79 deaths per year due to salmonellosis between 1979 and 1984 (Public Health Service, U.S. Dept. of Health and Human Services, 1979-84). Death certificate information is very inaccurate for two reasons. First, death certificates are filled out within hours of death and are not updated with laboratory tests showing the causative organism. Second, autopsy data are not subsequently entered on the death certificate. However, these death data were useful here in providing an age distribution of deaths attributed to salmonellosis.

Figure 3

Distribution of estimated annual U.S. salmonellosis cases and disease outcomes[1]



[1]Percentages are rounded.
Prepared by Economic Research Service, USDA.

The largest U.S. outbreak of salmonellosis occurred in Chicago in 1985. It was linked to two antimicrobial-resistant strains of *S. typhimurium* in pasteurized milk from one dairy plant (Ryan *et al.* 1987).[15] Two surveys of randomly selected households in two Chicago area counties provided data on the extent of this outbreak. For each of these surveys, Ryan *et al.* (1987) estimated the average number of salmonellosis cases based on the amount of illness in the survey samples and the distribution of implicated milk. The average of these two incidence estimates is roughly 185,000 cases. A third survey found that at least 2,777 of the culture-confirmed cases were hospitalized (Ryan *et al.* 1987, pp. 3270 and 3272).[16] These figures were used to conservatively calculate that approximately 1.5 percent (2,777/185,000) were hospitalized and survived.

Applying this rate to the estimated 800,000 to 4 million annual cases of salmonellosis provides an estimate of the range of cases that were hospitalized for salmonellosis (12,000 to 60,000). To this range, the 800 to 4,000 estimated annual deaths were added because those who died were likely to incur medical costs associated with hospitalization prior to death. Therefore, including deaths, the estimated number of annual hospitalizations for salmonellosis ranges from 12,800 to 64,000.

Data from the 1985 Chicago milk outbreak were also used to obtain estimates of the annual number of U.S. salmonellosis patients who visited a physician. A sample of 600 cases was randomly drawn from the culture-confirmed cases. Of this sample, 63 percent of the respondents reported that they visited a physician either in the emergency room or in a private office (Ryan 1987, personal communication with Roberts). It was assumed that 63 percent of the roughly 16,000 culture confirmed cases saw a physician and that none of the remaining 200,000 cases (184,000) saw a physician.[17] The rate of doctor visits

[15]Ryan *et al.* (1987) suggest that the original source of the resistant strains was the use of antimicrobials on dairy cattle. A cross-connection between pasteurized and raw milk lines may have been responsible for the outbreak (D'Aoust 1989, p. 360).

[16]Questionnaires were given to 15,459 (93 percent) of the 16,659 culture-confirmed cases. Of the 12,624 respondents, 2,777 (or 22 percent) were hospitalized (Ryan *et al.* 1987, pp. 3270 and 3272).

[17]Note that these estimates are slightly different than those used for the calculations on hospitalizations. These estimates are from preliminary data and are used to calculate a conservative estimate of the percentage of salmonellosis cases who visited a physician. The assumptions used here were suggested by Ryan (1987), CDC, to offset the higher-than-average rate of people seeking medical attention because of the publicity and the possibility of class action suits against the dairy in this unusually well-publicized case.

then is 5.04 percent for the whole Chicago outbreak ((63%)(16,000)/200,000).[18] Multiplying 5.04 percent times the estimated range of 800,000 to 4 million annual salmonellosis cases results in an estimated 40,320 to 201,600 doctor visits for salmonellosis annually.

The estimated number of salmonellosis cases where no medical care was sought was computed as a residual (total cases minus all hospitalizations, including deaths and physician visits). Estimated cases for this category range between 746,880 and 3,734,400 cases annually.

Costs of Salmonellosis from All Sources

Cohen *et al.* (1978) surveyed 234 people who had culture-confirmed cases of salmonellosis during the 1976 outbreak caused by eating *S. heidelberg*-contaminated cheddar cheese in Colorado. For the current analysis, cost estimates for the different severity categories were taken from Cohen *et al.* and updated to 1993 using average weekly earnings or consumer price indexes (CPI), where appropriate. These costs are the only estimates available in the literature. Although Cohen *et al.* state that 26 percent of the total cost of this outbreak is for lost income or productivity, 68 percent is for medical costs, and 5 percent is for miscellaneous costs, they do not separate medical costs from lost productivity for each disease severity category.[19] Therefore, table 7 presents the costs summary for annual cases of salmonellosis, broken down by disease severity category but not by type of cost.

- No physician visit. For this category, Cohen *et al.* (1978) estimated that the costs per case were $125 in 1976 dollars.[20] Per case costs increased from $125 to $371 after adding 39 percent to account for fringe benefits (U.S. Dept. Comm., Bureau of the Census 1993, table 677) and updating to 1993 dollars. Costs were updated to 1993 dollars with Bureau of Labor Statistics' (BLS) average weekly earnings for all production or nonsupervisory workers in private nonagricultural industries in 1993 (GPO: *Economic Indicators*, July 1994). Estimated costs for the 746,880-3,734,400 cases each year who did not seek physician care for salmonellosis totaled $276.8 million to $1,384.1 million.

- Physician visit only. Cohen *et al.* (1978) estimated that salmonellosis patients who saw a physician but were not hospitalized incurred an average cost of $222 per case (1976 dollars) and stated that these costs "could mainly be attributed to medical care."[21] We updated Cohen *et al.'s* (1978) estimate of $222 per case to $794 using BLS's CPI for the physician services component (U.S. Dept. Comm., Bureau of the Census 1993, tables 151 and 163). This amount is all inclusive (*e.g.*, costs of doctors' fees, laboratory charges, and medication).[22] For the 40,320-201,600 cases in the category, we estimated total costs to run $32-$160 million annually.

- Hospitalized. Cohen *et al.* (1978) also estimated that salmonellosis patients who were hospitalized (and survived) incurred an average cost of $1,750 per case (1976 dollars) and stated that these costs "could mainly be attributed to medical care or hospitalization." We updated the $1,750 cost per case to 1993 dollars ($9,087) using BLS's CPI for hospital rooms (U.S. Dept. Comm., Bureau of the Census 1993, table 163). This estimate includes the costs of emergency plus regular room charges, hospital doctors' fees, medication, and operations. Estimated medical costs for the 12,000-60,000 hospitalized cases who survived total $109.0-$545.2 million annually.

- Deaths. Roberts (1988 and 1989) extended Cohen *et al.'s* estimates to include the value of lives lost annually to salmonellosis. This report updates Robert (1988 and 1989) COI estimates for salmonellosis to 1993 dollars.

[18]Note that this percentage is strikingly similar to that found in two studies of salmonellosis surveillance cases in 1979-80 and 1984-85 where 2,200 of the 40,000 cases involved a visit to a doctor (or 5.5 percent) (Cohen and Tauxe 1986).

[19]Over all disease severity levels, Cohen *et al.* (1978) estimated that those employed lost, on average, 12 days from work. Moreover, family members missed an average of 3 days from work to care for the sick relative (Cohen *et al.* 1978).

[20]Although Cohen *et al.* (1978) stated that these "costs were primarily accountable to loss of salary or output," they did not specify what proportion is for lost productivity and what proportion is for medical costs. Therefore, costs were not broken down between these two cost sub-categories.

[21]Note that whereas physician expenses and laboratory tests were the key costs cited by Cohen *et al.* (1978), productivity loss is also included in their estimate.

[22]Adults over 50 years of age and infants incurred higher than average costs due to more frequent or longer hospitalization (staying a median of 10 days versus 6 days for other age groups) (Cohen *et al.* 1978).

Table 7—Cost summary for U.S. salmonellosis cases, 1993[1]

Severity of illness	Cost per case: Cohen *et al.*	Cost per case: This analysis	Estimated cases and total costs: Low: Cases	Low: Costs	High: Cases	High: Costs
	1976$	*1993$*	*Number*	*Mil. Dollars*	*Number*	*Mil. Dollars*
No physician visit[2]	125	371	746,880	276.8	3,734,400	1,384.1
Physician visit[3]	222	794	40,320	32.0	201,600	160.0
Hospitalized[4]	1,750	9,087	12,000	109.0	60,000	545.2
Deaths[5]	N/A	385,355	800	308.3	4,000	1,541.4
Total[6]	N/A	N/A	800,000	726.1	4,000,000	3,630.8
If 87-96% are foodborne, foodborne costs are $0.6-3.5 billion annually.[7]						

[1]Some numbers have been rounded for this table.

[2]Cases in this category were calculated as a residual. We use Cohen *et al.'s* estimate that the costs per case are $125 (1976 dollars), after we increase this value by 39% to account for fringe benefits and update to 1993 dollars using average weekly earnings for non-agricultural workers from the U.S. Dept. of Comm., Bureau of Labor Statistics (BLS).

[3]Assuming 5.04% of all cases visit a physician (Ryan, personal communication, 1987). Cost per case is from Cohen *et al.'s* (1978) estimate of $222 (1976 dollars), updated to 1993 dollars using BLS's CPI for physician services (U.S. Dept. of Comm., Bur. of the Census).

[4]This category is for those who were hospitalized and survived. Assuming 1.5% of all cases are hospitalized (Ryan *et al.* 1987). Cost per case is from Cohen *et al.'s* (1978) estimate of $1,750 (1976 dollars), updated to 1993 dollars using BLS's CPI for hospital rooms (U.S. Bur. of the Census).

[5]Deaths are calculated using a case fatality rate of 1/1,000. Those who die are assumed to be hospitalized prior to their deaths and incur the same costs as those who are hospitalized and survive. Therefore, the total number of salmonellosis patients hospitalized each year is 12,800 for the low estimate and 64,000 for the high estimate. Costs for those who die are the sum of the cost per hospitalized case ($9,087) and Landefeld and Seskin's (1982) average value of a statistical life for the age distribution ($376,268 after averaging across gender and updating to 1993 values using the average weekly earnings.)

[6]The low estimate of 800,000 cases was calculated by multiplying CDC's estimate of 40,000 *Salmonella* isolates (Tauxe 1991) by Chalker and Blaser's (1988) low estimate of the number (20) of unreported cases to each reported case. The high estimate of 4 million cases was calculated by multiplying CDC's estimate of 40,000 *Salmonella* isolates (Tauxe 1991) by Chalker and Blaser's (1988) high estimate of the number (100) of unreported cases to each reported case.

[7]The 87% foodborne estimate is from Tauxe and Blake (1992) and the 96% foodborne estimate is from Bennett *et al.* (1987).

We assumed that each of the 800-4,000 salmonellosis cases who die prematurely because of their illness incurred the same amount of medical costs as a salmonellosis patient who was hospitalized and survived ($9,087). We estimated medical costs for those who die from salmonellosis to range between $7.3 million and $36.3 million annually.[23]

Assuming the reported age distribution is representative of all estimated salmonellosis deaths, it can be used with Landefeld and Seskin's (1982) value of a statistical life (VOSL) numbers to estimate the benefits of reducing premature death from salmonellosis. Updated to 1993 values using the Consumer Price Index for all goods (U.S. Dept. Comm, Bur. Lab. Stats. 1994), the average value for the stream of productivity lost for each premature salmonellosis death is $376,268.[24] Multiplied by the 800-4,000 estimated salmonellosis deaths, the productivity loss estimates range from $301.0-$1,505.1 million annually. Combining this estimated productivity loss with the estimated $7.3-$36.3 million in medical costs sums to an annual total of $308.3-$1,541.4 million for those who die from salmonellosis.

- Total. In summary, the annual human illness costs of salmonellosis are substantial. Our estimate of the total costs of non-typhoid salmonellosis from

[23]Total medical costs for all hospitalized cases (those who survive plus those who die) are estimated at $116.3 million to $581.6 million annually.

[24]Therefore, the total cost of a death is $385,355 (table 7), the sum of medical costs ($9,087) from Cohen *et al.* (1987) and productivity losses ($376,268) from Landefeld and Seskin.

all sources ranges from $726.1 million to $3,630.8 million annually. The difference between the low and the high costs estimates is completely due to the difference in the two estimates of the incidence of salmonellosis.

Costs of Foodborne Salmonellosis

Adjusting for foodborne causes (87-96 percent), an estimated 696,000 to 3,840,000 salmonellosis cases stem from food sources each year (see page 70 of text). Of these cases, 649,786 to 3,585,024 do not visit a physician for their illness and 35,078 to 193,536 visit a physician. When adjusted for foodborne causes, the range of all hospitalized cases becomes 11,136 to 61,440 which includes a range of 696 to 3,840 deaths annually.[25]

The total costs of non-typhoid foodborne salmonellosis were estimated in the same manner as above. Total costs range from $0.6 billion to $3.5 billion annually.

Remarks

Salmonellosis currently ranks as the most costly bacterial foodborne disease estimated to date, partly due to the large number of cases and partly due to its virulence among specific population subgroups: the elderly, infants, and, increasingly, the immunocompromised.

The per person costs for the three non-death severity categories rely exclusively on Cohen *et al.* (1978). Of particular concern is the cost estimate for those who did not seek medical care, because it is based on such a small sample. These cases are a large contributor to total costs because the vast majority of salmonellosis cases are of mild severity. Because Cohen *et al.* (1978) did not explicitly state the medical costs and productivity loss by severity level, estimates could be improved with this information. To the extent the outbreak investigated in Cohen *et al*. (1978) may differ from a "typical" salmonellosis outbreak, several sources of bias are possible; the disease severity may be higher or lower than average depending on the *Salmonella* serotype, the number of *Salmonella* ingested, and the age and sex composition of the group of people affected.

These COI estimates do not include the costs of chronic medical conditions, which may be significant. The likelihood of such occurrences and associated costs are unknown. Archer (1984, 1985) estimated that 2 percent of salmonellosis patients will end up with reactive arthritis, an inflammation of the joints that lasts from a few days to 6 months. A fraction of these cases develop rheumatoid arthritis, a life-long inflammation of the joints.[26] With better data on incidence and associated costs of chronic illnesses caused by salmonellosis, total costs of these chronic illnesses could be computed and added to estimated costs associated with acute salmonellosis.

Other costs to individuals are ignored. For example, a Canadian economist, Leo Curtin (1984) estimated that the loss of leisure time was greater than the loss of work time due to salmonellosis. Estimates using his methodology of valuing leisure time at the prevailing wage rate would more than double the cost of salmonellosis estimates presented here.

Since reporting of *Salmonella* began in 1943, the reported incidence in the United States has increased considerably (Tauxe 1991, p. 563). For the past 30 years, this increase has been progressive and significantly greater than the population increase (Chalker and Blaser 1988, p. 113). During the 1970's, roughly 30-40 outbreaks and 20,000-25,000 isolates of salmonellosis were reported to CDC versus 60-80 outbreaks and 40,000-45,000 isolates in the late 1980's (Helmick *et al.* 1994, p. 104).[27] Massive outbreaks of salmonellosis such as the Chicago milk outbreak have increased the annual average number of infections reported to CDC.

The proportion of *Salmonella* infections due to *S. enteritidis* has increased to the extent that this serotype is now the most common cause of salmonellosis in some regions of the country (Tauxe 1991, p. 566). As previously mentioned, *S. enteritidis* infections have been increasingly attributed to consumption of lightly cooked or raw shell eggs.

[25]Some of these deaths are AIDS patients.

[26]As an aside, one survey found that rheumatoid arthritis sufferers were willing to pay 22 percent of their household income to be rid of arthritis (Thompson 1986, p. 394).

[27]In contrast, *Shigella* infections have remained constant over the past 30 years, even though they are reported by identical mechanisms as *Salmonella* infections (Chalker and Blaser 1988).

Some evidence (*e.g.*, 1985 outbreak traced to milk) exists to support the theory that widespread use of antimicrobials (*i.e.*, tetracycline) for both human illnesses and animal husbandry has led to an increase in resistance and infection with specific strains of *Salmonella* (MacDonald *et al.* 1987). Tauxe *et al.* (1991, p. 566) state that "treatment with antimicrobial agents can actually promote *Salmonella* infections in both humans and animals, particularly if the infecting strain is resistant to the agents being used."

AIDS patients are among the sub-groups most at risk of salmonellosis. Jackson *et al.* (1991, p. 32) discuss AIDS patients with salmonellosis and cite Celum *et al.* (1987) as saying that AIDS patients are 19.2 times more likely to contract salmonellosis than people who do not have AIDS. Recurrent bacteremia caused by *Salmonella* was included as an indicator of AIDS in 1987 (Tauxe 1991, p. 566).

Ryan *et al.* (1987, p. 3274) caution that the trend in food production toward a relatively small number of large producers make catastrophic consequences from *Salmonella* contamination possible. A case in point is the 1994 Schwan *Salmonella* outbreak, associated with consumption of ice cream, which led to around 100,000 cases in 25 States, including 102 hospitalizations (Food Chemical News Feb. 13, 1995, p. 53). The Food and Drug Administration (FDA) has implicated milk tanker trucks that previously carried raw egg products as the source of this outbreak.

In addition, large and dispersed outbreaks have been shown to cross State and national boundaries. For example, in 1984, a large international airline served *Salmonella*-contaminated food on 29 flights from London to the United States over a 3-day period, affecting an estimated 2,737 passengers (Tauxe *et al.* 1987, p. 150).[28] Incidents such as this have the potential to spread infections among people of many countries, especially where the infection can be spread person-to-person. A *S. Chester* outbreak in 1990 in the United States was linked to cantaloupe from Central America and Mexico (Ries *et al.* 1990). These concerns suggest a need for a global strategy for handling food safety issues such as *Salmonella* infections in humans.

[28]Passengers to the United States were not the only ones affected. The airline potentially exposed 23,576 passengers of 125 overseas flights from London to non-European destinations.

COI Estimates of Campylobacteriosis

There are nine named or proposed *Campylobacter* species that are pathogenic or are believed to be potentially pathogenic to humans (Tauxe et al. 1988, p. 1).[29] Of these, *Campylobacter jejuni* and *C. coli* (two closely related species) organisms are the species most frequently associated with campylobacteriosis in humans.[30] In the United States, most *C. jejuni* infections are associated with consumption of poultry and most *C. coli* infections are associated with consumption of pork. Worldwide, *Campylobacter* is estimated to cause 5 to 14 percent of all human diarrheal illnesses (Benenson 1990, p. 69).

Campylobacteriosis symptoms can range from diarrhea and lethargy that lasts a day to severe diarrhea and abdominal pain (and occasionally fever) that lasts for several weeks (Park *et al.* 1991, p. 995). Diarrhea and abdominal pain are the most common symptoms and the vast majority of cases are mild. Skirrow and Blaser (1992, p. 3) report that abdominal pain from campylobacteriosis can be so strong that it has been misdiagnosed as originating from appendicitis and has led to unnecessary appendectomy. The incubation period is 1 to 10 days, with most cases occurring 3 to 5 days after exposure (Benenson 1990, p. 70).

Although most cases of campylobacteriosis are self-limiting, up to 20 percent have a prolonged illness (longer than 1 week) or a relapse (Blaser *et al.* 1979), and 2 to 10 percent may be followed by chronic sequelae (CAST 1994, p. 11). Complications that may follow *Campylobacter* infections include meningitis, cholecystitis (inflammation of the gall bladder), urinary tract infection, appendicitis, septicemia, and Reiter syndrome (urethritis, arthritis, and conjunctivitis) (Mossel 1988) (table 3). Mishu and Blaser (1993) estimate that 20-40 percent of all Guillain-Barré Syndrome (GBS) cases are caused by *Campylobacter* infections. GBS is the major cause of non-trauma-related paralysis in the United States. Although paralysis from GBS is generally reversible over time, some patients die prematurely because of the illness while others are bedridden for life.

[29]In 1913, *Campylobacter* was discovered as a pathogen and was called *Vibrio fetus* because it was associated with abortion and infertility in cattle and sheep (Stern and Kazmi 1989, p. 72). The link with human illness occurred in the 1950's when *Campylobacter* was isolated from human blood (Tauxe *et al.* 1988, p. 1).

[30]For the remainder of this report, *C. jejuni* will be used to refer to both *C. jejuni* and *C. coli*.

Poultry is the predominant source of sporadic cases of *Campylobacter* (Tauxe 1992, p. 12). In the United States, an epidemiological study by Harris *et al.* (1986a, p. 410) found that in approximately half of all *Campylobacter jejuni/coli* enteritis cases, ingestion of contaminated chicken was the primary source of infection. The intestine of birds and warm-blooded animals is a natural habitat for *Campylobacter* (Park *et al.* 1991, p. 101S) and studies have concluded that chicken slaughter and processing leads to heavy surface contamination (Park *et al.* 1991; Skirrow and Blaser 1992, p. 6; Sjögren and Kaiser 1988, p. 3). Up to 80 percent of poultry at retail are contaminated with *Campylobacter* (Skirrow and Blaser 1992, p. 4) and contamination appears to peak during the summer (July through October)(Tauxe *et al.* 1988; Harris *et al.* 1986b, p. 404).[31] This seasonal pattern of contamination in raw poultry is reflected in the reported number of *Campylobacter* isolates, by month and year, in the United States between 1982 and 1989 (fig. 4). To a lesser extent, turkey, raw milk, cake icing, raw clams, raw hamburger, water, and contact with pets have been epidemiologically linked with human diseases in the United States (Blaser *et al.* 1983a, p. 163; Stern 1992, p. 50; Tauxe *et al.* 1988; CAST 1994, p. 11).

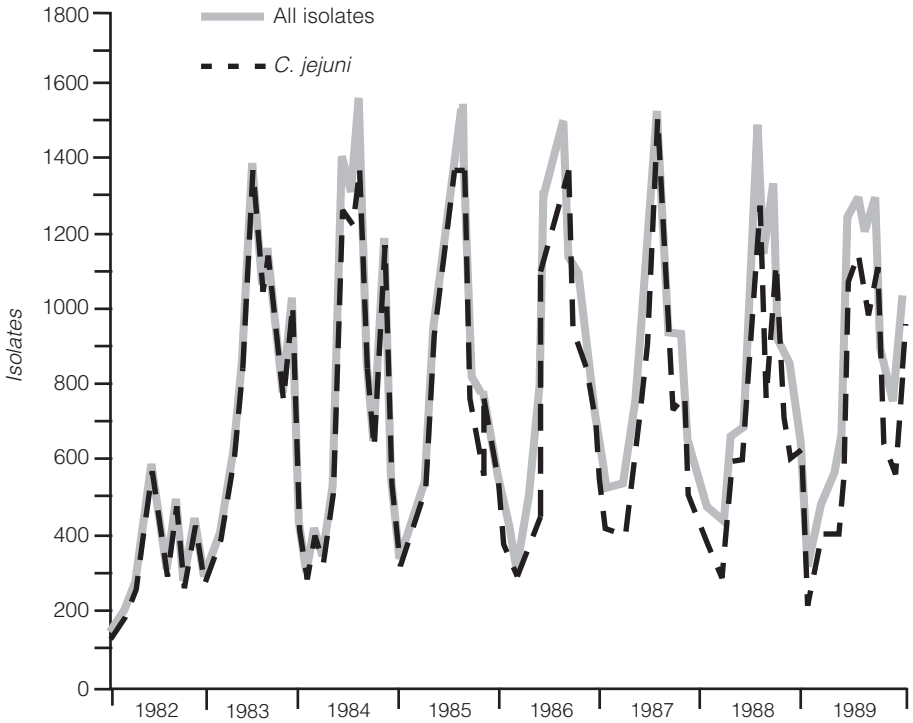
Campylobacteriosis outbreaks are relatively uncommon, perhaps because *Campylobacter jejuni* does not multiply in food; most outbreaks can be traced to drinking untreated stream or river water or to drinking raw milk (Helmick *et al.* 1994, p. 110).[32] *Campylobacter* in unchlorinated water is a major cause of travelers' diarrhea (Benenson 1990, p. 69).

[31]Note that reported isolates, which are primarily from sporadic cases, peak in the summer (July-Aug.) whereas the distribution of outbreaks is bimodal with peaks in May and October (Tauxe 1992, p. 13). This difference may be largely due to the difference in reservoirs, that is poultry for sporadic cases and raw milk and contaminated water for outbreak cases (Tauxe 1992, p. 12).

[32]Bean and Griffin (1990, p. 806) provide estimates of the number of foodborne disease outbreaks, by pathogen. Between 1973 and 1987, there were 53 *Campylobacter*, 190 *Clostridium perfringens*, 367 *Staphylococcus aureus*, and 790 *Salmonella* outbreaks. They did not mention *Listeria* outbreaks and estimated *E. coli* O157:H7 outbreaks are not relevant for this time period, because *E. coli* O157:H7 was not identified as a cause of human illness until 1982.

Figure 4

Reported number of U.S. *Campylobacter* isolates, by month and year, 1982-89



Source: Tauxe, R. V., "Epidemiology of *Campylobacter jejuni* Infection in the United States and Other Industrialized Nations." Chapter 2 in Nachamkin, Irving, Martin J. Blaser, and Lucy S. Tompkins, eds. *Campylobacter jejuni: Current Status and Future Trends.* Washington, DC: American Association of Microbiology, 1992, p. 10.

There is little evidence to suggest that significant person-to-person transmission of *Campylobacter* takes place (Tauxe 1992, p. 11). However, there have been a few documented cases of pregnant women with bacteremia that have been associated with severe fetal infections (Blaser *et al.* 1983a, p. 165).

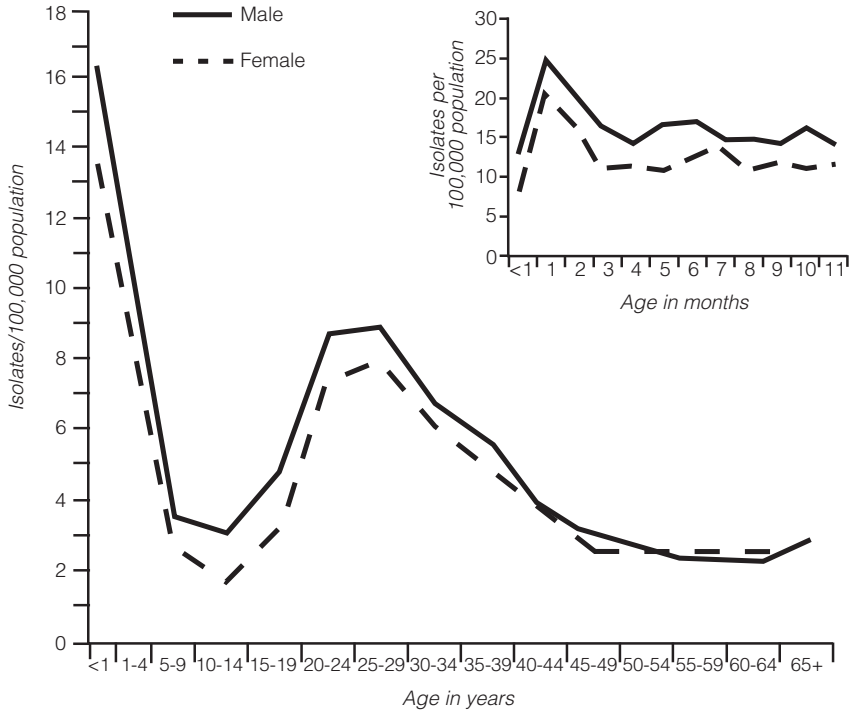
There is uncertainty as to the percentage of *Campylobacter* cases that are foodborne. In a Seattle-King County Department of Public Health surveillance study (1984, p. 153), roughly 55 percent of all *Campylobacter* cases were attributed to food origins: drinking raw milk (5.2 percent) and eating poultry (48.2 percent).[33] Deming *et al.* (1987) summarized in Tauxe [1992, p. 15] found that 70 percent of campylobacteriosis cases in students at a Georgia college were attributed to eating chicken (the remaining 30 percent were attributed to contact with cats). We assumed that 55-70 percent of all U.S. campylobacteriosis cases are foodborne.

The human infective dose is relatively small. Robinson (1981) describes his personal experience where 500 *Campylobacter* cells caused disease. Yet, there is great variation in individual susceptibility and illness severity. Benenson claims that in developing countries, most individuals develop immunity to *Campylobacter* in their first year of life (1990, p. 70). This may also be true in developed countries. In developed countries such as the United States, infants have the highest reported incidence of campylobacteriosis, with young adults in the second highest risk category (Tauxe *et al.* 1988, p. 11). In developing countries, children under 2 years of age are the most likely to have *Campylobacter* infections (Blaser *et al.* 1983a) and illnesses (Benenson 1990, p. 69). Up to age 45, males have higher isolation rates for *Campylobacter* than do females, but this difference has not been adequately explained (Tauxe 1992, p. 10). Figure 5 shows the reported *Campylobacter* isolates by age and sex between 1982 and 1986.

[33]Of the remaining *Campylobacter jejuni* infections, 6.3 percent were from pets, 9 percent from foreign travel (which could also be food related), and 7.6 percent from surface water.

Figure 5

Reported *Campylobacter* isolates by age and sex of patient, United States 1982-86



Source: Tauxe, R. V., "Epidemiology of *Campylobacter jejuni* Infection in the United States and Other Industrialized Nations." Chapter 2 in Nachamkin, Irving, Martin J. Blaser, and Lucy S. Tompkins, eds. *Campylobacter jejuni: Current Status and Future Trends.* Washington, DC: American Association of Microbiology, 1992, p. 11.

Estimates of Cases

The discovery of the extent of infections with *Campylobacter* was made possible in the late 1970's by the development of an isolation technique that requires incubator conditions and a specific medium (Tauxe 1992, p. 9; Helmick *et al.* 1994, p. 109). Helmick *et al.* (1994, p. 109) report that in laboratories that test for foodborne pathogens including *Campylobacter*, *Campylobacter* is the most commonly isolated bacterial pathogen from persons with diarrhea in the United States, and *C. jejuni* is the most commonly isolated *Campylobacter* species.[34] In 5-year CDC surveillance, 91 percent of the isolates reported the species and 99 percent of these specified *C. jejuni* as the reported species (Tauxe *et al.* 1988, p. 9).

Although there has been a national surveillance for campylobacteriosis since 1982, participation is voluntary, and there is tremendous variation in internal reporting requirements between States (Tauxe 1992, p. 9). Unlike *Salmonella*, isolates of *Campylobacter* are not routinely referred for confirmation or serotyping except when an unusual isolate is found or in outbreak situations (Tauxe 1992, p. 9). But even during outbreaks, not all physicians routinely order diagnostic laboratory testing for patients sick with diarrheal illnesses. With better surveillance, *Campylobacter* infections would likely outnumber *Salmonella* infections (Tauxe 1992, p. 9).[35]

The limitations of *Campylobacter* surveillance are especially critical because the vast majority of cases are sporadic and not the result of outbreaks (Tauxe 1992, p. 11). Individuals sick with campylobacteriosis may be more likely to seek medical care (and have diagnostic testing) during outbreaks than when cases are sporadic, especially when there is widespread publicity and the possibility of legal action.

In 1980, a Collaborative Diarrheal Disease Study Group (conducted by CDC) studied the relative frequency with which *Campylobacter*, *Shigella*, and *Salmonella* were isolated from stool cultures at eight hospitals over a 15-month period (Blaser *et al.* 1983b). They found that, for all age groups, *Campylobacter* species were isolated 4.6 times more frequently than *Shigella* species and twice as often as *Salmonella* species (Blaser *et al.* 1983b, p. 360).[36] This supports the hypothesis that if both *Campylobacter* and *Salmonella* infections had equal surveillance efforts, *Campylobacter* isolates would be more common (Tauxe 1992, p. 12). If *Campylobacter* were analyzed in the same fashion as *Shigella* or *Salmonella*, then the estimated isolation rate would be 36-40 per 100,000 (Tauxe 1992, p. 12).[37]

Rosenberg *et al.* (1977, p. 459) used national surveillance data to estimate that as few as 33 percent of physician visits for patients sick with shigellosis resulted in a stool culture. This low proportion that has diagnostic testing further documents the extent of potential underreporting of foodborne illnesses. Following Tauxe (1992, p. 12), we conservatively assumed that 67 percent of patients who visit physicians with complaints of diarrheal illnesses have stool cultures ordered. This raises the estimated isolation rate for *Campylobacter* from 36-40 per 100,000 to 54-60 per 100,000 (Tauxe 1992, p. 12).

Sacks *et al.* (1986) report results from an investigation of a campylobacteriosis outbreak associated with a contaminated community water supply in Florida. They found that of 865 cases, roughly 5.4 percent (or 47) visited a physician for acute gastroenteritis (p. 425). Tauxe (1992, p. 12) uses this percentage combined with his estimated isolation rate for *Campylobacter* (54-60 per 100,000) to estimate the rate of *C. jejuni* infection of roughly 1 percent of the U.S. population annually (not including asymptomatic cases). Given a 1993 residential U.S. population of 257,908,000 (U.S. Dept. Comm., Bureau of the Census 1993), there were over 2.5 million estimated cases of campylobacteriosis in 1993. Helmick *et al.* (1994, p. 109) state that the true number of annual cases of *Campylobacter* infections each year in the

[34] *C. jejuni* is the main reported isolate in stool cultures but this may be because the stool culture media is more appropriate for *C. jejuni* than for other C*ampylobacter* species (Tauxe *et al.* 1988). In general, isolation methods and methods used to identify species other than *C. jejuni* are expensive and cumbersome, which means less is known about the extent and severity of human illness caused by these species.

[35] Underreporting occurs for a host of other reasons previously described (*e.g.*, many ill people do not seek medical care). Another reporting problem for *Campylobacter* is that it has only recently been assigned a code in the International Classification of Disease (ICD) system, which means that many otherwise useful medical databases (*e.g.*, the National Hospital Discharge Survey) cannot provide information on incidence.

[36] Note that Tauxe (1992, p. 12) says that *Campylobacter* was isolated 4.5 times more often than *Shigella*.

[37] The reported isolation rate of *Shigella* species is 8/100,000 and 20/100,000 for *Salmonella* species (Tauxe 1992, p. 12).

United States is likely 2 million to 10 million cases. We (conservatively) assume 2.5 million cases of campylobacteriosis each year in the United States in our update of Lin *et al.*'s (1993) COI estimate for campylobacteriosis (table 2).

Table 8 presents the estimated U.S. campylobacteriosis cases, broken down by the four disease severity categories used throughout this report. Figure 6 presents the distribution of estimated annual cases of campylobacteriosis and disease outcomes.

While the distribution of disease severity is similar to that of salmonellosis, campylobacteriosis is generally less deadly than salmonellosis, leading to an estimated 200 to 730 deaths annually versus an estimated 800 to 4,000 deaths for salmonellosis. Tauxe (1992) reports that Smith and Blaser (1985) used surveillance data to estimate a death rate of 24 per 10,000 culture-confirmed cases of *Campylobacter* infections. Tauxe (1992, p. 14) applied this death rate to the estimated annual number of culture-confirmed *Campylobacter* infections to calculate an estimated 200 deaths from campylobacteriosis each year. Tauxe also used data from a series of *Campylobacter* outbreaks to estimate an upper bound on the number of premature deaths due

Table 8—Estimated U.S. campylobacteriosis cases, 1993

Severity of illness	Estimated cases	
	Low	High
	Number	
No physician visit[1]	2,352,300	2,351,770
Physician visit[2]	135,000	135,000
Hospitalized[3]	12,500	12,500
Deaths[4]	200	730
Total[5]	2,500,000	2,500,000

[1] Cases in this category were calculated as a residual.

[2] Assuming 5.4% of all cases visit a physician (Sacks *et al.* 1986).

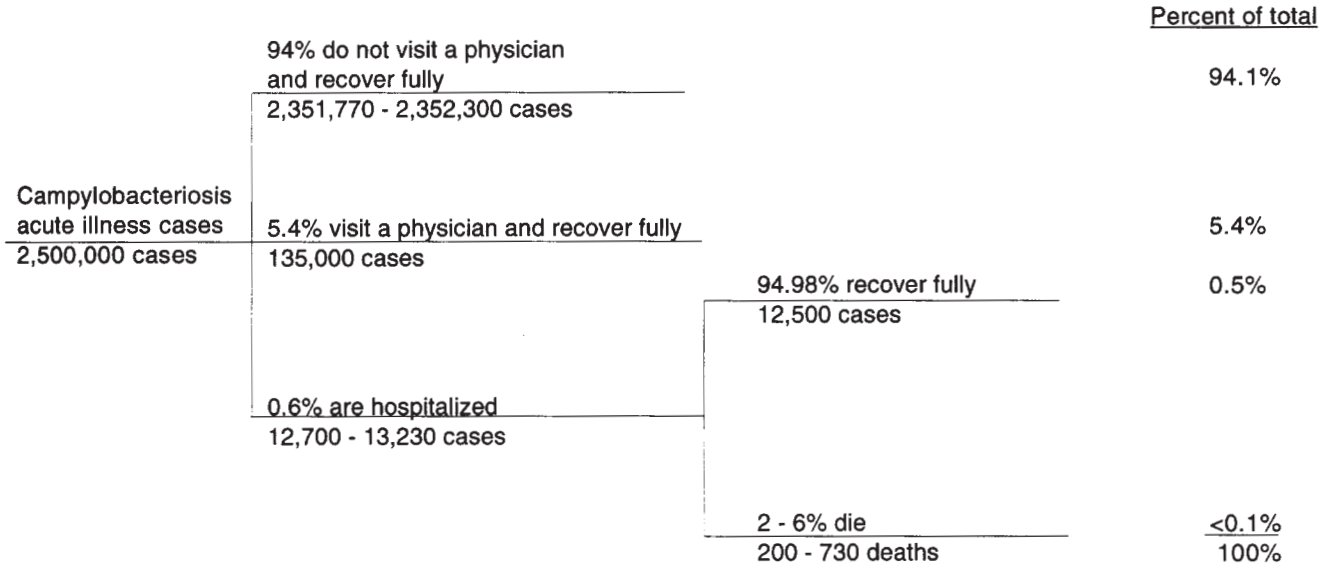
[3] This category is for those who were hospitalized and survived. Assuming 0.5% of all cases are hospitalized (Sacks *et al.* 1986).

[4] The low estimate of 200 deaths was calculated in Tauxe (1992) using Smith and Blaser's (1985) case fatality rate of 24/10,000 culture-confirmed cases. The high estimate of 730 deaths was calculated in Tauxe (1992) using his case fatality rate of 3/10,000 outbreak-associated illnesses. Those who die are assumed to be hospitalized prior to their deaths. Therefore, the total number of salmonellosis patients that are hospitalized each year is 12,700 in the low estimate and 13,230 in the high estimate.

[5] The total number of campylobacteriosis cases is 1% (Tauxe 1992) of the U.S. 1993 population (U.S. Bur. of the Census) rounded down to 2.5 million cases.

Figure 6

Distribution of estimated annual U.S. campylobacteriosis cases and disease outcomes[1]



[1] Percentages are rounded.
Prepared by Economic Research Service, USDA.

to *Campylobacter* in the United States of 730 per year (ibid.). The current study considers a range of 200 to 730 deaths from *Campylobacter* infections annually.

Sacks *et al*. (1986, p. 425) in their study on the *Campylobacter*-contaminated community water supply found that 0.5 percent of cases were hospitalized (4 out of 865 cases). Applying this rate to the annual number of 2.5 million *Campylobacter* infections, we obtain an estimated 12,500 hospitalizations each year for campylobacteriosis. Assuming that the 200-730 who die from the illness are first hospitalized, an estimated 12,700 to 13,230 cases of campylobacteriosis cases are hospitalized each year.

We used Sacks *et al.'s* (1986, p. 425) finding that roughly 5.4 percent visited a physician for campylobacteriosis during the contaminated-water outbreak to estimate the annual number of people in the United States who visit a physician for *Campylobacter* infections. When this rate is applied to the 2.5 million estimated annual infections, this yields an estimate of 135,000 physician visits for *Campylobacter* infections each year.

The estimated number of campylobacteriosis cases where no medical care was sought was computed as a residual (total cases minus all hospitalizations, including deaths, and physician visits). The number of cases in this category are estimated to range between 2,352,300 and 2,351,770 cases annually.

Costs of Campylobacteriosis from All Sources

Nolan and Harris (1984, p. 166) comment that the illness severity of campylobacteriosis mirrors that of salmonellosis. In our analyses of these two illnesses, the breakdown of the severity categories is comparable; the percentage that visit a physician is around 5 percent, the percentage that are hospitalized is around 1 percent, and the percentage that die is ≤ 0.1 percent.

Given the similarity in the four severity groups for campylobacteriosis and salmonellosis and given that there is no parallel cost study like Cohen *et al*. (1978) for campylobacteriosis, we assumed that per-patient costs of illness for each of the four severity groups are identical to the costs of salmonellosis. We used Cohen *et al.* (1978) estimates for all four categories of campylobacteriosis and LS VOSL estimates for the productivity loss from those who die prematurely. As previously mentioned, Cohen *et al.* (1978) did not separate medical costs from productivity losses. Therefore, one cannot divide per-patient or total costs into medical costs and costs of lost productivity. Table 9 presents the cost summary for annual cases of campylobacteriosis, broken down by disease severity category.

- No physician visited. As with salmonellosis, estimated per-patient costs for campylobacteriosis patients who did not visit a physician are roughly $371.[38] For the estimated 2,351,770 to 2,352,300 campylobacteriosis cases in this category, estimated annual costs total $871.7-$871.9 million.

- Physician visit only. As with salmonellosis, estimated per-patient costs for campylobacteriosis patients who visited a physician for their illness are $794.[39] This includes office visits, laboratory charges, and some productivity loss. For the estimated 135,000 cases in the category, estimated costs total $107.2 million annually.

- Hospitalized. As with salmonellosis, estimated per-patient costs for campylobacteriosis patients who were hospitalized for their illness are $9,087.[40] This includes the costs of emergency plus regular room charges, hospital doctors' fees, medication, and operations. For the estimated 12,500 cases who were hospitalized and survived, estimated costs total $113.6 million annually.

- Deaths. As for the estimated 200 to 730 annual deaths from campylobacteriosis, per patient costs are the sum of Cohen *et al.'s* (1978) per patient costs of $9,087 (updated to 1993 as above) plus

[38]Per-case costs increase from $125 (Cohen *et al.* 1978) to roughly $370.65 after adding 39 percent to account for fringe benefits (U.S. Dept. of Comm., Bureau of the Census 1995, table 677) and updating to 1993 dollars. Because Cohen *et al.* (1978) state that "these costs were primarily accountable to loss of salary or output," we updated costs to 1993 dollars with BLS's average weekly earnings for all production or nonsupervisory workers in private nonagricultural industries in 1993 (*Economic Indicators*, July 1994).

[39]Because Cohen *et al.* (1978) state that the costs per case for this category are "mainly . . . attributed to medical care," we update their estimate of $222 per case to $794 using BLS's CPI for the physician services component (U.S. Dept. of Comm., Bureau of the Census 1993, tables 151 and 163).

[40]Because Cohen *et al.* (1978) explain that their estimate of $1,750 per case for this category is mainly comprised of medical costs, we updated this amount to $9,087 using BLS's CPI for the hospital room component (U.S. Dept. of Comm., Bureau of the Census 1993, table 163).

LS's VOSL. We assumed that the age distribution of deaths due to campylobacteriosis mirrors that of salmonellosis. Applying the age distribution of deaths to Landefeld and Seskin's VOSL estimates by age, we obtained an average VOSL of $376,268. When LS VOSL costs are combined with per patient costs ($9,087) from Cohen *et al.* (1978), the per patient costs for those who died are $385,355 (table 9). For the 200-730 campylobacteriosis deaths annually, lost productivity adds up to $77.1-$281.3 million.

- Total. For all patients with campylobacteriosis, costs are estimated at $1,169.8-$1,373.8 million annually. The difference between the low- and the high-cost estimates is completely due to the range of 200-730 deaths.

Very few studies have estimated the costs of campylobacteriosis (Sockett and Stanwell-Smith 1986; Roberts 1989; Todd 1989[b]), partly because only recently has *Campylobacter* been attributed to causing substantial foodborne illnesses. None of these studies are detailed cost analyses for campylobacteriosis in the United States. While it is not an unreasonable assumption to apply the costs per salmonellosis case to those of campylobacteriosis, better estimates are needed.

Sockett and Stanwell-Smith (1986) provide the most detailed cost study of campylobacteriosis to date. They estimated costs incurred by health-care services per case of campylobacteriosis in the United Kingdom (UK) (1985, in £). Because the structure of health care in the UK differs considerably from that in the United States, these costs are not directly

Table 9—Cost summary for U.S. campylobacteriosis cases, 1993

Severity of illness	Cost per case		Estimated cases and total costs			
			Low		High	
	Cohen *et al.*	This analysis	Cases	Costs	Cases	Cost
	1976$	*1993$*	*Number*	*Mil. Dollars*	*Number*	*Mil. Dollars*
No physician visit[1]	125	371	2,352,300	871.9	2,351,770	871.7
Physician visit[2]	222	794	135,000	107.2	135,000	107.2
Hospitalized[3]	1,750	9,087	12,500	113.6	12,500	113.6
Deaths[4]	N/A	385,355	200	77.1	730	281.3
Total[5]	N/A	N/A	2,500,000	1,169.8	2,500,000	1,373.8
If 55-70% are foodborne, foodborne costs are $0.6-1.0 billion annually.[6]						

N/A = Not applicable.
Note: Some numbers have been rounded for this table.

[1] Cases in this category were calculated as a residual. We use Cohen *et al.'s* (1978) estimate that the costs per case are $125 (1976 dollars), after we increase this value by 39% to account for fringe benefits and update to 1993 dollars using average weekly earnings for nonagricultural workers from the U.S. Bureau of Labor Statistics (BLS).

[2] Assuming 5.4% of all cases visit a physician (Sacks *et al.* 1986). Cost per case is from Cohen *et al.'s* (1978) estimate of $222 (1976 dollars), updated to 1993 dollars using BLS's CPI for physician services (U.S. Dept. of Comm., Bur. of the Census).

[3] This category is for those who were hospitalized and survived. Assuming 0.5% of all cases are hospitalized (Sacks *et al.* 1986). Cost per case is from Cohen *et al.'s* (1978) estimate of $1,750 (1976 dollars), updated to 1993 dollars using BLS's CPI for hospital rooms (U.S. Dept. of Comm., Bur. of the Census).

[4] The low estimate of 200 deaths was calculated in Tauxe (1992) using Smith and Blaser's (1985) case fatality rate of 24/10,000 culture-confirmed cases. The high estimate of 730 deaths was calculated in Tauxe (1992) using his case fatality rate of 3/10,000 outbreak-associated illnesses. Those who die are assumed to be hospitalized prior to their deaths. Therefore, the total number of salmonellosis patients that are hospitalized each year is 12,700 for the low estimate and 13,230 for the high estimate. Costs for those who die are the sum of the cost per hospitalized case ($9,087) and Landefeld and Seskin's (1982) average value of a statistical life for the age distribution ($376,268 after averaging across gender and updating to 1993 values using the average weekly earnings.)

[5] The total number of campylobacteriosis cases is 1% (Tauxe 1992) of the U.S. 1993 population (U.S. Dept. of Comm., Bur. of the Census) rounded down to 2.5 million cases.

[6] The 55% foodborne estimate is from the Seattle-King County study (1984) and the 70% foodborne estimate is from Deming *et al.* (1987).

applicable to our analysis.[41] However, if we assumed that costs are similar in the United States and UK, the average cost estimates from the current analysis are roughly $468-$550 per case whereas the Sockett and Stanwell-Smith (1986) estimates would suggest a range of $339-$370 per case (in 1993 U.S. dollars).[42] Their estimates were lower because they assume a lower death rate.

Costs of Foodborne Campylobacteriosis

We assumed that 55-70 percent of all estimated human illness cases of *Campylobacter* in the United States are foodborne (1,375,000 to 1,750,000 cases)(see page 70 of text). Estimates of those who do not visit a physician range from 1,293,765 to 1,646,239 cases annually. A low of 74,250 and a high of 94,500 visit a physician. The number of hospitalized cases (including those who died) ranges from 6,985 to 9,261. Foodborne deaths caused by *Campylobacter* range from 110 to 511 annually.

Given our assumption that 55-70 percent of all U.S. campylobacteriosis cases are attributed to food, estimated costs of foodborne campylobacteriosis range from $0.6-$1.0 billion annually. Due to the limitations of the Cohen *et al.* (1978) data for salmonellosis and the lack of current and detailed cost information for campylobacteriosis, medical costs and the costs of lost productivity cannot be separated.

Remarks

As previously mentioned, poultry is the most common cause of sporadic cases of campylobacteriosis in the United States. This is largely because poultry naturally harbors *Campylobacter* in the crop and gut. Mass mechanized processing can cause heavy cross-contamination (Skirrow and Blaser 1992, p. 6). Cleaning practices at the slaughterhouse and packing plant may influence the *Campylobacter* contamination rate; Harris *et al.* report a progressive increase in contamination of poultry carcasses slaughtered from Monday through Wednesday after a weekend cleanup (1986, p. 403). Park *et al.* (1991, p. 102S) recommend that *Campylobacter* contamination of raw chicken be reduced by improving processing procedures at slaughter to minimize fecal contamination and by reducing available water on the carcass. Washing carcasses in a strong brine solution may be helpful in reducing *Campylobacter* contamination (Park *et al.* 1991, p. 102S). *Campylobacter* tends to be found more frequently on moist meat rather than on dry meat because *Campylobacter* is sensitive to drying (Park *et al.* 1991, p. 102S). Perhaps, air-chilled chicken would have fewer *Campylobacter* than ice-bath-chilled chicken.

According to CDC, approximately 90 percent of *C. jejuni* outbreaks would not occur with universal pasteurization of milk and improved drinking water treatment (Tauxe 1992, p. 12). Raw milk often harbors a wide range of pathogens such as *C. jejuni*, *Salmonella* serotypes, and *Listeria*. For this reason it is illegal to sell raw milk in most locations in the United States (CAST 1994, p. 32).

Additional gains in reducing the annual number of cases of campylobacteriosis could be made through improved food-handling practices at both the retail and household levels. Tauxe (1992, p. 16) states that "ingestion of a small drop of raw chicken juice could easily be the infective dose." That being the case, leaky packages of chicken purchased at supermarkets may be causing illnesses. As previously mentioned, *Campylobacter* needs moisture to survive; reducing free water in poultry packages would seem to have the double benefit of encouraging the die-off of existing *Campylobacter* and of reducing potential kitchen contamination levels.

At the household level, *Campylobacter* infections could be reduced by greater education of consumers on kitchen hygiene for handling and cooking poultry. Hopkins and Scott (1983) reported that "handling raw chicken appeared to be a strong risk factor" for getting campylobacteriosis. Deming *et al.* (1987, p. 532) hypothesize that errors in food handling increase the risk of illness from eating cooked chicken. Tauxe *et al.* (1992) state that the 1984 Seattle study found the risk of campylobacteriosis "was inversely associated with the frequency of using soap to clean the kitchen cutting board."

[41]Regardless of differences in health care, it is interesting that a British study by Kendall and Tanner (1982, p. 155) found that the annual incidence of *Campylobacter* infections in a general practice population in Great Britain was 1.1 percent as compared with Tauxe's (1992) incidence rate of 1 percent in the United States.

[42]The estimated $468-$550 average costs were calculated by dividing the estimated total annual costs of $1,169.8-$1,373.8 million by the estimated 2.5 million U.S. cases (table 9). The estimated $339-$370 average costs were calculated from the Sockett and Stanwell-Smith (1986) estimates by using the exchange rate of £1 to U.S. $1.53 (Riggs Bank, Washington, DC, Nov. 15, 1994) and by updating to 1993 dollars (using BLS CPI for all items).

Consumption patterns affect the risks of foodborne illnesses. Poultry consumption increased 35 percent during the 10-year period between 1982 and 1992, as consumers substituted chicken for other meats (Lin *et al.* 1993, p. 38). This increase is partly because of concerns over dietary fat content, and partly because chicken has become cheaper than other meats. A recent consumer survey showed that 95 percent of the chicken bought for home consumption consisted of fresh products, such as fresh chicken parts (Lin *et al.* 1993, p. 38). The prevalence of *Campylobacter jejuni* and *Salmonella* in raw poultry means that this new consumption trend may lead to increasing numbers of foodborne illness cases.

COI Estimates of *Escherichia Coli* O157:H7 Disease

E. coli O157:H7 was first isolated by CDC in 1975 (FDA Consumer 1994, p. 9) but was not identified as a cause of human illnesses until 1982 when two outbreaks of gastrointestinal illness in Michigan and Oregon were investigated and linked to consumption of contaminated hamburgers (Riley *et al.* 1983)(fig. 7). There has been worldwide detection of *E. coli* O157:H7 and its associated illnesses. Bovine isolates or human cases of *E. coli* O157:H7 have been documented in over 16 countries and on 6 continents (USDA:APHIS:VS 1994, p. 2). Benenson (1990, p. 137) states that infections from enterohemorrhagic strains of *E. coli* (mainly O157:H7) are recognized as important health problems in Europe, southern South America, and North America. The following analysis updates the 1992 COI estimates found in Roberts and Marks (1995) to 1993 dollars.

E. coli O157:H7 and its link to an associated life-threatening illness called hemolytic uremic syndrome (HUS) became well known to the public as a result of the 1993 *E. coli* O157:H7 disease outbreak caused by contaminated hamburger in Washington, California, Idaho, and Nevada. The American Gastroenterological Association's (AGA) Consensus Conference Statement on *E. coli* O157:H7 Infections (1995, p. 1923) indicated that this outbreak led to over 700 illness cases (primarily children) and of these cases, 195 were hospitalized (28 percent), 4 died (0.57 percent), and 55 developed HUS (7.86 percent). In recent years, an increasing number of *E. coli* O157:H7 outbreaks and sporadic cases have been documented (AGA 1995, p. 1923).

E. coli O157:H7 is a hardy organism. Although bile acids can help kill some microorganisms such as *Clostridium botulinum*, *Escherichia is* resistant to these acids (CAST 1994, p. 25). *E. coli* O157:H7 can also survive some acid environments in food such as that found in apple cider (FDA Consumer 1994, p. 8). *E. coli* O157:H7 can grow and multiply slowly at temperatures as low as 44°F and can even survive freezing (FDA Consumer 1994, p. 9). *E. coli* O157:H7 can also survive in water for extended periods (USDA:APHIS:VS 1994, p. 1). However, *E. coli* O157:H7 is easily killed by heat used in pasteurization and cooking (USDA:APHIS:VS 1994, p. 1). The 1993 outbreak of *E. coli* O157:H7 infections indicated that the infectious dose is less than 1,000 organisms (AGA 1995, p. 1925). The CAST report estimates that the infectious illness dose is in the range of 10 to 1,000 colony-forming units (1994, p. 12).

E. coli O157:H7 is a toxicoinfective microorganism because it causes human illnesses through the toxins that it produces (CAST 1994, p. 19). *E. coli* O157:H7 toxins cause human illnesses by adhering to receptors in the kidney, intestine, and central nervous system where it prevents protein synthesis and kills cells (CAST 1994, p. 19). The bloody diarrhea and abdominal cramping typically found in symptomatic cases of *E. coli* O157:H7 disease are caused by the toxins that *E. coli* O157:H7 produces and by the partial destruction of the colon's mucosal lining (USDA:APHIS:VS 1994, p. 1). In the United States, *E. coli* O157:H7 is a major cause of bloody diarrhea (AGA 1995, p. 1924).

E. coli O157:H7 causes a wide range of illness severities in humans from mild cases of acute diarrhea to premature death. Acute illness from *E. coli* O157:H7 disease is manifested by abdominal cramps, vomiting, diarrhea (often bloody), and sometimes fever. Ostroff *et al.* (1989, p. 355) found that 95 percent of the 93 reported sporadic cases of *E. coli* O157:H7 disease in Washington State in 1987 had bloody diarrhea. Griffin and Tauxe (1991, p. 64), in reviewing the literature, speculated that bloody diarrhea is the most commonly reported symptom, because persons with bloody diarrhea are more likely to seek medical care and because physicians are more likely to culture stools if patients report bloody stools.

The incubation period for *E. coli* O157:H7 in humans is typically 3 to 5 days (AGA 1995, p. 1925).