A Survey of Estimated Risks of Human Illness and Costs of Microbial Foodborne Disease

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No comprehensive data source estimates human illnesses caused by foodborne contaminants. The U. S. Environmental Protection Agency's (EPA) worst case estimate of risks from chemical contaminants in food (pesticides, animal drug residues, growth hormones, and food additives) range from a high of 6,000 cases of cancer annually to a moderate level of risk (Table 1). In contrast, the basis for estimates of microbial contaminants (bacteria, parasites, viruses, and fungi) are *reported* cases of human foodborne illnesses. These are underestimates of the true incidence. Best estimates of microbial foodborne illness in the literature suggest there are 6.5 to 33 million cases annually of which 9,000 are deaths. Estimates of human illness costs of microbial foodborne disease are $4 to 8 billion annually (Roberts and Foegeding). Public alarm about chemical foodborne disease risks and dismissal of microbial risks are opposite from their threats to human health. More accurate risk assessments and a standard methodology may bring public perceptions into agreement with food safety experts. This paper discusses risk assessment procedures and analyzes the literature estimating human health risks from microbial contaminants in food.

Table 1.
Comparison of Foodborne Disease Risks

Type of risk	Annual estimates of human illness	Source
Microbial contaminants	6.5 million cases/9,000 deaths	CDC/Bennett, Holmberg, Rogers & Solomon
	33 million cases	FDA/Garthright, Archer & Kvenberg
Chemical residues	6,000 or fewer cancer cases	EPA/1987, 1990

Risk Assessment

The National Research Council of the National Academy of Sciences (NAS) has extensively studied risk assessment. Risk assessment is a statistical estimate, based on the best science available, of the likely health effects of natural phenomena and human activities. A 1983 report, *Risk Assessment in the Federal Government: Managing the Process*, set up a general framework for evaluating and controlling risk in federal programs. The framework includes:

> ***Identifying the hazard:*** Determining whether a particular chemical, microorganism, or other substance causes particular human health effects.
> ***Assessing the human health effects at different doses:*** Determining the relationship between the level of contamination and the probability distributions of possible health effects.
> ***Assessing the exposure:*** Determining the extent of human exposure before or after application of regulatory controls.
> ***Characterizing the risk:*** Identifying the economic and social impacts of the human risk by estimating the costs associated with the risk and their impact on special populations, regions, and industries.

While NAS' four-step risk assessment procedure sounds simple, in practice it is very complex. For example, to make linkages between food and human illness, it is necessary to conduct tests to identify the causative pathogen or chemical contaminant. However, tests may not exist or their sensitivity, reliability, speed, or cost may restrict use. In addition, issues of sample size, media used, enrichment procedures all affect test results. This is particularly true since some foodborne pathogens can cause disease at low numbers. The more distant in time it is from consumption to onset of illness and the fewer numbers of people involved, the less likely a food linkage becomes. NAS has published reports developing methodologies for assessing specific risks (NAS 1984; 1988) and for designing regulatory programs for foodborne disease risk (NAS 1969; 1985; 1987a; 1987b; 1990).

The Office of Management and Budget (OMB) supports NAS' statement on the importance of separating risk assessment from risk management. In the risk management process policy considerations are crucial. However, we should base risk assessment only on science. The OMB criticized the conservative, "worst-case" estimates for mixing policy and science: "Conservatism in risk assessment distorts the regulatory priorities of the Federal Government, directing societal resources to reduce what are often trivial carcinogenic risks while failing to address more substantial threats to life and health" (p. 14).[1]

OMB recommends the use of ranges around the best estimate to show scientific uncertainty rather than worst case scenarios. The sources of the scientific uncertainty differ for chemical and microbial foodborne disease estimates. We base estimates of risks from ingesting chemical contaminants in our food on extrapolations from animal test data to reflect the risk of long term health effects, primarily cancer.[2] For long term chemical risks the uncertainty involves the possible inability of finding a good animal model to test the chemical and the expense of animal testing which limits the compounds fully tested. Uncertainty exists in the extrapolation from animal to human health effects, extrapolation from high doses used in tests to low doses expected in human exposure,[3] and estimates of human exposure for the entire population and for specific high-risk groups.

In contrast, we estimate microbial risks based on cases of human illness reported immediately after consuming contaminated food. The uncertainty in microbial risks results from undercounting acute illnesses, especially microorganisms causing disease after a lag of a few days (botulism, campylobacteriosis) or weeks (listeriosis, hepatitis). This lag reduces the ability of the individual or their physician to identify the food connection, as well as missing chronic effects which infrequently result from most microbial foodborne illnesses.

Acute Illnesses from Microbial Foodborne Sources

Exposure. Humans exposure to microbes from animal sources occurs in several ways (Table 2). The most obvious is consuming contaminated food. Most researchers also include contamination of food by the food handler which then causes human illnesses. Often omitted is the contamination of workers from handling contaminated food or breathing contaminated air in food processing plants or slaughterhouses. We could include illnesses in farm families contracted from exposure to animals raised for food in estimates of foodborne diseases, arguing that these human illnesses would not have occurred if these families were in another business.

Acute Illnesses Reported to CDC. The Centers for Disease Control (CDC) in Atlanta is notified of the *reported* cases of acute human illnesses caused by microbial contamination of food. These cases start with state and local health departments receiving information from two or more individuals experiencing a similar illness.[4] The state or local health department conducts an investigation to determine if food is the common source. The last step is notification of the CDC.

The data reported to the CDC differ widely from state to state due to different state health departments (Chalker and Blaser, Centers for Disease

Table 2.
Pathways of Human Exposure to Disease from Animal Products

I.	**Direct contact with live animal**
	• Animal bite
	• Contact with the skin, fur, feathers, tail, etc., and microbes found there
II.	**Indirect contact with the live animal**
	• Aerosol contamination of the barn and air system
	• Contamination of the wall, floor, gates, etc.
	• Animal wastes
	• Flies or fleas biting the infected animal and then biting humans and transmitting disease
III.	**Direct contamination by the carcass or other animal products**
	• Some organisms penetrate the skin of personnel handling meat, poultry, or other animal products
	• Entry of organisms through cuts and nicks on the hand of slaughter house or processing plant workers
IV.	**Indirect contamination by animal products**
	• Aerosol contamination when meat or other animal products are cut up or processed
	• Contact with knives, wiping cloths, sinks, etc. where pathogens have been deposited
V.	**Cross contamination of other foods**
	• Carcass contaminating other carcasses in the slaughterhouse
	• Animal products in the processing plant contaminating clean processed foods
	• Cross contamination of clean raw or cooked foods in private or public kitchens
VI.	**Consumption of food**

Control). Typically health officials annually report 400 to 500 foodborne outbreaks to the CDC with an average of about 50 cases per outbreak. This results in a total of 20,000 to 25,000 foodborne disease cases each year (Table 3). Not identified are about 60 percent of the organisms causing outbreaks (Centers for Disease Control; Bean et al.).

CDC's Laboratory Based Data. The CDC's laboratory-based data are an annual series of test results reported to the CDC from state laboratories identifying organisms as causes of human illness. The CDC has set up a laboratory-based surveillance systems for the following diseases (which are often foodborne): salmonellosis, shigellosis, listeriosis, botulism, campylobacteriosis, trichinosis, cholera, typhoid fever, and hepatitis A. The cases reported by laboratories are higher than the cases reported in the foodborne outbreak system because most foodborne cases are sporadic, not outbreak-associated. Annual reports to the CDC include around 44,000 salmonellosis cases, 20,000 shigellosis cases, 10,000 campylobacteriosis cases (Nancy Bean, telephone interview, February 1990). The laboratory-base surveillance data are also likely to be nationally representative and represent a good cross-section of the cases when the patient was ill enough to seek medical attention. However the distribution of illness severities will be unknown.

Table 3.
Confirmed U.S. Foodborne Disease Outbreaks, 1983-87

Etiologic agent	Outbreaks	Cases	Deaths
		number	
BACTERIAL			
Bacillus cereus	16	261	0
Brucella	2	38	1
Campylobacter	28	727	1
Clostridium botulinum	74	140	10
Clostridium perfringens	24	2,743	2
Escherichia coli	7	640	4
Salmonella	342	31,245	39
Shigella	44	9,971	2
Staphylococcus aureus	47	3,181	0
Streptococcus, Group A	7	1,001	0
Streptococcus, other	2	85	3
Vibrio cholera	1	2	0
Vibrio parahemolyticus	3	11	0
Other bacterial	3	259	70
Total	600	50,304	132
CHEMICAL			
Ciguatoxin	87	332	0
Heavy Metals	13	176	0
Monosodium glutamate	2	7	0
Mushrooms	14	49	2
Scombrotoxin	83	306	0
Shellfish	2	3	0
Other chemical	31	371	1
Total	232	1,244	3
PARASITIC			
Giardia	3	41	0
Trichinella spiralis	33	162	1
Total	36	203	1
VIRAL			
Hepatitis A	29	1,067	1
Norwalk virus	10	1,164	0
Other viral	2	558	0
Total	41	2,789	1
CONFIRMED TOTAL[a]	909	54,540	137

Source: Data from Bean et al. or Centers for Disease Control.

a. Over these 5 years, total foodborne outbreaks numbered 2,397 and outbreaks with an unknown etiologic agent numbered 1,488. Confirmed outbreaks were 38% of the total and those with an unknown agent were 62% of total foodborne outbreaks.

The CDC sentinel county studies are surveillance investigations of laboratory-identified cases for a specific purpose. These include identifying the number of foodborne disease cases caused by a new pathogen (for example, *Listeria monocytogenes*, identifying virulence mechanisms, discovering food vehicles, or identifying the distribution of case severities. Recent sentinel county studies have addressed *Campylobacter, Listeria, Salmonella, and Shigella.* These sentinel county studies obtain the best data because the CDC selects a nationwide sample of counties for the mail survey of ill people. These sentinel county studies are not annual series, are somewhat more expensive, and cover only a few microorganisms.

CDC's Expert Opinion to Develop "Best Estimates". Epidemiologists, recognizing that reported data are just the tip of the iceberg, have made several attempts to estimate the extent of underreporting. Chalker and Blaser calculated the likelihood of reporting a salmonellosis case at each stage of the surveillance process (Table 4). Infections can go unreported because the infected person does not become ill, a sick person chooses not to see a physician, the physician does not get a specimen, the laboratory does not identify the organism from the specimen, the laboratory fails to report its test results to the health department, or the health department fails to report the laboratory test results to the CDC.[5] The final column in Table 4 shows the range of estimates reported in the literature for underreporting at each step. The second column is Chalker and Blaser's median reporting ratio for each step. We can get the total reporting ratio by successive multiplication of the ratio from each surveillance step. The total reporting ratio of 39 means that for each case of human salmonellosis

Table 4.
Summary of Sequential Artifacts in *Salmonella* Surveillance

Surveillance step	Estimated total cases/reported cases	
	Median	Range
1. Patient infected	1.0	
2. Patient ill	2.2	1.3-17.0
3. Patient consults a doctor	2.2	1.3-12.0
4. Doctor obtains specimen	2.4	1.2-4.3
5. Laboratory identifies the organism	1.4	1.2-2.9
6. Laboratory reports to health dept.	2.0	1.3-2.4
7. Health department reports to CDC	1.2	1.2[a]
Salmonella surveillance reporting ratio[b] = 39. Or 39 estimated total salmonellosis cases for each one reported.		

a. Only one study in the literature.
b. The reporting ratio of 39.0 is obtained by successive multiplication of ratios from each surveillance step.
Source: Adapted from Chalker and Blaser, p. 115.

reported under the CDC's surveillance system, they estimate that 39 cases actually occurred.

Experts sometimes apply a multiplication factor to the CDC data, similar to the Chalker and Blaser work, to compensate for underreporting (Hauschild and Bryan). Recently, CDC researchers estimated morbidity and mortality for all infectious and parasitic diseases (Bennett et al.). They identified seventeen diseases as foodborne, in whole or in part. The process used CDC surveillance and outbreak data, published reports, and expert opinion on the overall incidence and case-fatality ratios for each of the pathogens involved. They estimated the proportion spread by food for illnesses caused by each pathogen, and that estimate was then used to allot cases and deaths to foodborne vehicles (John V. Bennett, Carter Center, Emory University, personal communication, April 1990). The estimated incidence of symptomatic illness from foodborne microorganisms was 6.5 million cases annually in the United States, with about 9,000 deaths.[6]

In general, factors influencing the likelihood of a foodborne disease being identified include: the seriousness of the disease, the unusualness of the disease symptoms, and the more quickly the disease follows the ingestion of the contaminated food.[7] Other factors include the availability of tests, "name recognition" (all physicians know *Salmonella* causes food poisoning), whether many people become ill simultaneously, and the availability of resources to conduct foodborne disease investigations. These resources are often in competition with other routine and emergency programs, such as the increase in environmental concerns, AIDS cases, and reduced budgets.

The types of data available from the CDC for salmonellosis show low estimates of the outbreak data, a lack of information on case severity from laboratory data, as well as a greater detail on severity gained by the sentinel county surveys. The data also show a much higher estimate of cases and full description of severity using expert opinion (Table 5).

FDA Estimates Based on Health Surveys. Garthright et al. estimated 99 million acute cases of Intestinal Infectious Disease occur each year in the United States. This disease is defined as vomiting and/or diarrhea without respiratory symptoms. The illnesses caused more than a day of restricted activity in half of the cases. A physician consultation occurred in only 7.9 million cases. Hospitalization occurred in 250,000 cases. The authors made no attempt to estimate deaths. The disadvantage of this data is lack of identification of specific pathogens responsible for disease and the inability to identify which foods contain the pathogens.

Earlier Archer and Kvenberg suggested that 30 to 35 percent of these Intestinal Infectious Diseases were foodborne, and FDA is using 33 million cases as

Table 5.
Salmonellosis Data from CDC Sources, Annual Cases

	Outbreak[a] data, 1983-87	Lab-based data, 1989[b]	Surveillance Studies[c] 1979 & 1984	Extrapolations based on Outbreak[d] Data	Extrapolations based on Surveillance Studies[c,d]
	#				
Outbreaks	68				
Cases	6,250	44,000	40,000	2,000,000	400,000-4,000,000
Severity					
deaths	8		500	2,000	1,000
hospitalization			18,000	34,000	36,000
physician seen			22,000	101,000	44,000
no medical attention				1,906,000	1,919,000

Sources:
a. Centers for Disease Control 1990
b. Bean, N.H. 1990, Centers for Disease Control, Atlanta, Georgia, personal communication.
c. Cohen & Tauxe 1986
d. Roberts 1989

the best estimate of foodborne disease. If the severity of foodborne disease were the same as Intestinal Infectious Disease from other sources, foodborne disease would cause 83,000 hospitalizations, 2.6 million illnesses, 15 million cases involving no physician consultation but restricted activity for more than a day, and 15 million cases that involved restricted activity for less than 1 full day.

Discussion. Because of the difficulty in identifying foodborne cases, expert opinion remains the best way to estimate the actual number of cases. The range reported in the literature of 6.5 to 33 million cases annually seems plausible.[8] However, Chalker and Blaser show the variance in reporting a case of salmonellosis at each step of the process. For diseases which are less well-known and more difficult to identify, the variance would be even greater.

A difficulty is identifying the distribution of disease severity for acute cases of foodborne illness. Most foodborne microorganisms can cause a wide range of disease severities from mild, almost unnoticeable, infection to death. The variability of human effects depends upon individual genetics, immune system functioning, and gastric secretions. The variability also depends on the contaminated food consumed (fatty foods can protect *Salmonella* from the stomach acids) and the species and strain of each pathogen. Normally, low levels of bacterial contamination in food does not affect healthy individuals. Because of different food consumption patterns as well as differences in human genetics, persons at high risk for foodborne illness differ from organism to organism. Genetic factors altering individual susceptibility include stomach acidity, iron

level of the blood, potassium levels, and intestinal receptor sites (which may affect susceptibility to some foodborne pathogens without altering other health risks or shortening life expectancy). Other conditions, such as infancy, old age, medication (antibiotics of any sort or immuno-suppressants), surgical operations (like gastrectomy) or other disease conditions can affect the immune system and increase susceptibility to foodborne disease. Archer has estimated that, "AIDS patients are 20 times more likely to contract salmonellosis than normal individuals, and 200-1,000 times more likely to come down with listeriosis" (Food Chem News).

Systematic colleetion of data on long-term complications does not exist.[9] Complications of foodborne illness may include arthritis, kidney damage, blood poisoning, heart disease, pancreatic infections, pneumonia, permanent liver impairment and neurological damage (Archer 1984, 1985; Mossel). The range of possible effects is varied for most organisms. Kvenberg and Archer estimate that chronic illnesses are likely to occur in 2 to 3% of cases of foodborne infection. Identifying the specific chronic illness is difficult. The long term results of the chronic illness may be more serious than the acute illness. Because the chronic symptoms are not unique nor closely linked in time to a foodborne illness incident, these illnesses are difficult to relate to a specific pathogen or food vehicle.

Economic Costs Associated with Foodborne Disease

The final step in the risk assessment process is to assess the economic and social consequences of the risk. Because of the difficulties in identifying foodborne hazards and estimating the number, distribution of severities, and identifying chronic effects of foodborne infections, we will likely underestimate the costs.

Uncertainty also complicates cost estimation. For example, will estimated costs be nationally representative? What classes of cases are particularly vulnerable to undersampling? As more studies are done, researchers should discuss what portion of the disease severity distribution their estimates cover. If we can make adjustments for missing cases, so much the better. Cost estimations will improve by comparing one's work to other estimates in the literature and dissecting whether differences are due to estimates of cases or disease severities or are due to economic methodology.

Conceptual Issues. There are different methods for valuing costs of foodborne illness. The "cost of illness" approach estimates what resources society would save by avoiding the foodborne illness. The core costs in this approach are

medical costs and productivity losses.[10] We convert cost estimates into a present value. Economists agree that the discount rate is in the 2 to 5 percent range for comparing consumers' willingness to exchange future consumption with present consumption (Howe). Landefeld and Seskin have extended the cost of illness approach from a social cost perspective to the individual's perspective of loss by defining income more broadly and including a risk aversion factor:

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

where,
T = remaining lifetime,
t = a particular year,
Yt = after-tax (labor + non-labor) income + household production,
r = individual's opportunity cost of investing in risk-reducing activities, and;
α = risk-aversion factor.

The risk aversion factor reflects the increased premium individuals pay for life insurance over the expected income loss due to death of a household member.[11] The concreteness and simplicity of the cost of illness method is attractive. Harrington and Portney concluded that the cost of illness estimate would be a lower bound estimate of the true social cost of illness.

There is no necessary correspondence between the cost of an illness and the value a person would place on not having the illness. Most prefer the willingness-to-pay approach because it is derived from demand theory. Researchers have made steady progress using the willingness to pay method to evaluate recreational benefits, job related risks, and environmental attributes (Mitchell and Carson). Some studies use survey techniques to ask people what they would be willing to pay for certain contingencies (contingent valuation method). Others use laboratory simulations to see what participants will bid. Finally, others examine markets to tease out hidden values by examining risky attributes of consumer products or the wage/risk tradeoff in labor markets (hedonic method) (Fisher et al.).

Cost Estimates in the Literature. Published estimates of the costs of foodborne disease have mostly used the cost-of-illness method. Epidemiologists and microbiologists were the first to estimate foodborne disease costs. Often omitted however were deaths because few occur and because of the difficulty in valuing a lost life (Cohen et al.; Shandera et al.). Garthright (et al.) did not include deaths in their cost analysis of Intestinal Infectious Diseases. If foodborne

sources cause one-third of the cases, estimated costs for foodborne illness (excluding the deaths) are $7.7 billion annually. The estimates do not identify cases (and accompanying severity) associated with specific pathogens which diminishes their usefulness in designing control programs.

In contrast, estimates by economists (Mauskopf et al.; Roberts 1989) have shown that deaths are likely to be an important part of foodborne illness costs. Roberts estimated medical costs and productivity losses for salmonellosis (1987) and listeriosis (Roberts and Pinner). Deaths were the largest part of costs for the high salmonellosis estimate. Productivity losses caused by deaths dominated listeriosis costs.

Using the estimated cases and fatalities published by Bennett (et al.), Roberts (1989) extrapolated medical costs and productivity losses to the other bacterial pathogens.[12] I estimated an approximation of average costs for other bacterial pathogens by extrapolating from the salmonellosis and listeriosis cost estimates. I assumed that the average costs per case were a function of the death rate. The reliability of such an extrapolation depends on the extent to which all human illness costs vary directly with the death rate. It also depends on the share of total costs attributable to deaths caused by foodborne illnesses.[13] I estimated total medical costs and productivity losses at $4.8 billion annually for all foodborne bacterial diseases evaluated by Bennett (et al.) (Table 6). Note that the bacterial pathogens with the highest total estimated costs are *Campylobacter, Salmonella,* and *Staphylococcus,* which all have costs of around $1 billion annually. A second cluster of pathogens has estimated costs of around $200 million annually—*Listeria, Streptococcus,* and *Vibrio.*

Only a few estimates of willingness to pay to avoid foodborne disease are in the literature. A few studies used the contingent valuation method, but results have not been replicated. In a survey of Kansas consumers, Kramer and Penner found consumers willing to pay 1 to 3 cents per pound more for beef to avoid residues. Smallwood reported a nationwide survey in which over one half of respondents were willing to pay about 17 cents more per pound for "disease-free" chicken. (Nearly one of three respondents expressed a willingness to spend up to 20 additional minutes in preparation and cleanup time to reduce the risk of foodborne illness). Hammitt found that shoppers in organic food stores perceived that the health risks of pesticide residues on fruits and vegetables were greater than the risks perceived by shoppers at supermarkets.

Mauskopf (et al.) estimated losses for several foodborne disease severities using health status indexes. This technique's advantage is that all sources of well-being associated with a change in health status are measured:

Table 6.
Estimated Medical & Productivity Costs, Foodborne Bacterial Diseases, 1988

Bacterial Pathogen	Percent Foodborne	Estimated Annual Foodborne Cost
	%	$ million
Campylobacter jejuni and *coli*	100	1,470.0
Salmonella, nontyphi	96	1,344.0
Staphylococcus aureus	17	910.8
Miscellaneous enteric	95	256.4
Listeria monocytogenes	85	213.4
Vibrio infections, excl. cholera	90	198.9
Group A *Streptococococcus*	5	175.0
Shigella	30	107.3
Escherichia coli-enteric	25	59.6
Clostridium perfringens	100	51.3
Salmonella typhi	80	14.3
Clostrium botulinum	90	3.6
Yersinia	65	1.4
Bacillus cereus	100	1.0
Vibrio cholera	100	0.1
Brucella	5	0.1
TOTAL		$4.8 billion

Source: data from Roberts 1989.

> Bodily malfunctions are described on scales indicating the degree of, for example, restriction in mobility, social interaction, and physical activity and the pain or other symptoms a consumer may experience. Once a set of health states has been defined, estimates of the relative utility of the different health states can be obtained by using survey instruments that ask people to estimate the relative utilities (or level of well-being) associated with different health states. (pg. 3-1)

If we combined the Mauskopf (et al.) estimates for salmonellosis costs per case with the best estimates of cases in Table 5, the costs of salmonellosis become several times greater than mine (Table 7). The greatest difference between the mine and Mauskopf (et al.) estimates is the evaluation of death. Since the Mauskopf (et al.) estimate is a more comprehensive measure, the estimated values are higher.[14]

Table 7.
Annual Salmonellosis Costs: Roberts vs. Mauskopf et al., 1987

Case severity	Roberts' cost/case	Health status index used by Mauskopf et al.		
		Rosser & Kind	Bush et al.	Vaccine study
	$			
mild	221	222	550	175
moderate	680	860	2,100	615
severe	4,350	6,760	5,300	9,275
deadly	372,000	5,309,638	5,309,638	5,000,000
	billion $			
Total costs[a]				
sentinel county	1.0	6.0	6.6	5.8
outbreak	1.4	11.3	12.0	10.7

a. number of cases in each severity group determined by analysis of outbreak data or sentinel county data in Table 5.

A final concern is calculating losses caused by chronic illnesses with a foodborne source. Case estimates are rather vague, although Kvenberg and Archer estimate such effects occur in 2 to 3 percent of all foodborne infections. The range of estimates for annual cases of foodborne disease is 6.5 to 33 million annually as discussed earlier, which says that 130,000 to 990,000 cases of chronic disease start each year. If these persist throughout a person's remaining lifetime, 25 years is a reasonable assumption since it is the average remaining life expectancy at the time of infection for people who end up dying from salmonellosis (Vital Statistics-life tables and mortality tables). This translates to 3 to 25 million persons actively suffering from chronic conditions caused by food in any given year.

Thompson surveyed rheumatoid arthritis sufferers and asked what they would be willing to pay for an arthritis cure. The answer was an average of 22% of household income. Rheumatoid arthritis is likely to be more severe than other chronic diseases. Costs of chronic diseases may be as high as costs of acute illness from foodborne disease (Roberts and Frenkel).

Discussion

The available data for assessing foodborne risks are different for chemical contaminants versus microbes. The two involve different scientific disciplines and the risk time frame is not the same. One relies on animal models, the other on human illness data. Finally, the statutory regulatory requirements differ. The risk assessments for chemicals are *over*estimates, and the most commonly known data for microbes (reported cases) are *under*estimates.

The best estimates for microbial risks range from 6.5 to 33 million cases annually of which an estimated 9,000 are deaths. Microbial foodborne disease cost estimates in the literature range from $4-8 billion annually, although these are rough approximations (Roberts 1989; Todd 1989). One of the estimates ignores the costs of deaths (Garthright, Archer, and Kvenberg). All the estimates ignore costs of chronic complications of foodborne disease. Use of willingness-to-pay measures will increase cost estimates, as illustrated by Mauskopf et al.'s work.

Drawing further upon the environmental literature, we can apply new methods of describing and measuring various attributes of a good to food safety (Table 8). The parallels between food safety and environmental benefits for the two older concepts, cost of illness and averting behavior, are straightforward (Harrington et al.). The newer ideas are "use value," "option value," and "existence value." One can think of a use value estimate for producing pork which is truly disease-free and eaten raw in new European-type recipes (Mitchell and Carson). Or an option value for certain ethnic foods, perhaps cerviche (raw fish marinated in lime juice), which one might be willing to try if it is disease-free (Bishop). Or an existence value for aesthetics, such as being a vegetarian but preferring that others who do eat meat will not become ill. These concepts expand our understanding of food safety and, like the history in environmental economics, can increase the values estimated for food safety.

Table 8.
Food Safety Applications of Environmental Benefit Estimation Techniques

Category	Environmental Benefit Estimate	Food Safety Application
Cost of illness	Expenditure on medical care & lost productivity caused by air pollution	Expenditure on medical care and lost productivity caused by foodborne disease
Averting behavior	Asthmatics restricting their activity on bad air quality days	Giardiasis outbreak and use of bottled water, boiled water, or water from another watershed
Use Value	Travel cost; hedonic; contingent valuation	Willingness to pay for disease-free pork which can be eaten raw in European-type recipes
Option value	Willingness to pay for fishing hole which we may use in the future	Ethnic food not eaten now, but may wish it to meet certain standards because may eat someday
Existence value	Aesthetics, like to know the air is clean & safe to breathe in unpopulated areas	Aesthetics, what a vegetarian might be willing to pay for a meat inspection program

Notes

The views expressed in this paper are not official policy of USDA. I presented some of the analysis in this paper in a paper co-authored with Peggy Foegeding. Review comments by members of the CAST Task Force, Risks Associated with Foodborne Pathogens; Jo Mauskopf, Research Triangle Institute; Rob Tauxe, Bacterial Diseases Division, Centers for Disease Control; John Bennett, Carter Center, Emory University; and anonymous reviewers were greatly appreciated and helped improve the accuracy and thoughtfulness of this paper.

1. A numerical example of the confounding effect of conservatism: if a 5 step risk assessment procedure is used and estimates at each step are doubled to be conservative, or to reflect some underlying uncertainty, the resulting estimate is not simply conservative by a factor of 2 but overestimates risks by 32 ($32 = 2^5$).
2. However, risks to farm workers exposed environmentally to pesticides do include acute illnesses and teratogenic risks (fetal risks) as well as long term risks.
3. Ames and Gold have expressed particular concern over extrapolating from high doses to low doses because of the apparent ability of the human body to detoxify low doses of natural carcinogens.
4. For botulism and chemical poisoning only one case is necessary to call it an outbreak.
5. Estimates of the degree of underreporting varied greatly for the first two steps—whether the infected person actually becomes ill and whether the ill person actually consults a physician. The variability may be a function of the severity of illness which can fluctuate due to variability in the number of *Salmonella* organisms ingested, the *Salmonella* serotype, or host factors.
6. Food is omitted as the vehicle for some of the illnesses which have known foodborne causes: listeriosis, toxoplasmosis, giardiasis, cysticercosis, *Escherichia coli* O157:H7, and Norwalk virus. Furthermore, only 5 percent of cases of brucellosis are identified as being foodborne. These omissions reflect our rapidly changing knowledge of foodborne pathogens and the difficulty in defining what is meant by foodborne cases. Todd (1989) compared the estimates of Bennett (et al.) with estimates for the U.S. based on Canadian surveillance. This approach assumes that the underlying foodborne disease incidence is the same in both countries, but that the different reporting systems cause different estimates. Various methods of extrapolating from outbreak data were compared, but Todd ended up using the same multiplication factor for all diseases (with the exception of botulism). The Bennett (et al.) estimates however have multiplication factors

tailored to each disease, suggesting that the Bennett (et al.) estimates of U.S. cases of foodborne illness may be more accurate.

7. The onset time (time from consumption to illness symptoms) may range from 1/2 hour to several weeks; paralytic shellfish poisoning is an example of the former and *Hepatitis A virus* is an example of the latter.
8. Modelling is a new method for estimating the number of human cases of foodborne illness from microorganisms. The crucial components of the model are the level of exposure and the interaction of particular pathogen and host (defined by the dose-response curve)—steps two and three in NAS' risk assessment procedure. Probability models have been used to estimate the risk of infection after varying exposures to pathogens (Haas). Depending on the organism's ability to cause infection or disease, there would be a different model developed for each pathogen or each strain. The host population tested would also influence the model and a different model would be developed for each population with varying sensitivities to the pathogen. The exposure would depend on the initial concentration of the pathogen in the food, processes which would decrease the numbers (such as cooking) or environmental conditions which would increase (bacterial replication) or decrease (microbial death) the numbers, as well as the amount of food consumed. Modelling may be an important method for estimating the total number of cases of foodborne illnesses and the distribution of illness severities in the future, but cannot be used today for estimates.
9. Chronic complications are often a function of infection not illness, since chronic complications can occur even if a person's immune system successfully fights off the illness. In these cases, the activation of the immune system is what is important in causing the chronic condition.
10. In the literature on the human capital method, sometimes medical costs are called direct costs and productivity losses are called indirect costs.
11. Rice, the pioneer in codifying the human capital method for calculating cost of illness, continues to omit the risk aversion factor (Rice, MacKenzie and Associates).
12. Todd (1989) also estimated costs which are not discussed here—see note 6 for comparison of the Bennett (et al.) and Todd methods for estimating foodborne disease cases.
13. Given the wide range of illness severities caused by each bacterial pathogen, comparing the average severity among pathogens is difficult for an economist to evaluate. If either protracted acute illness or a chronic condition occurred as a consequence of a foodborne infection, the cost estimates presented here would be underestimates. Botulism could be an example of protracted acute illness in cases where survivors need hospitalization for

several months (Todd 1988). Salmonellosis cases causing arthritis are an example of a possible chronic condition (Archer 1984; 1985).

14. One concern with the rankings is whether medical insurance or paid sick-leave are included, since respondents often were not given instructions. Some researchers have anchored the rankings to explicit states, such as perfect health and death, but others have not. The former can be assigned utility weights, but the latter cannot (Mauskopf et al., D-8). Other concerns are the small sample sizes, variability of responses, and limited replication of results.

References

Ames, B. N. and L. S. Gold. 1990. "Too Many Rodent Carcinogens: Mitogenesis Increases Mutagenesis." *Science.* 249:970-971.

Archer, D. L. 1984. "Diarrheal Episodes and Diarrheal Disease: Acute Disease with Chronic Implications." *J. Food Protection.* 47(4):321-328.

Archer, D. L. 1985. "Enteric Microorganisms in Rheumatoid Diseases: Causative Agents and Possible Mechanisms." *J. Food Protection.* 48:538-545.

Archer, D. L., and J. E. Kvenberg. 1985. "Incidence and Cost of Foodborne Diarrheal Disease in the United States." *J. Food Protection.* 48(10):887-894.

Bean, N. H., P. M. Griffin, J. S. Goulding, and C. B. Ivey. 1990. "Foodborne Disease Outbreaks, 5-Year Summary, 1983-1987." *J. Food Protection.* 53(8):711-728.

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

Bishop, R. C. 1982. "Option Value: An Exposition and Extension." *Land Economics.* 58(1):1-15.

Centers for Disease Control. 1990. "Foodborne Disease Outbreaks, 5-year Summary, 1983-1987." In, *CDC Surveillance Summaries,* March 1990. *MMWR* 39(SS-1):15-59.

Chalker, R. B., and M. J. Blaser. 1988. "A Review of Human Salmonellosis: III. Magnitude of Salmonella Infection in the United States." *Rev. Infectious Diseases.* 10(1):111-124

Cohen, M. L., and R. V. Tauxe. 1986. "Drug-Resistant *Salmonella* in the United States: An Epidemiological Perspective." *Science.* 234:964-969.

Cohen, M. L., R. E. Fontaine, R. A. Pollard, S. D. Von Allmen, T. M. Vern, and E. J. Gargarosa. 1984. "An Assessment of Patient-Related Economic Costs in an Outbreak of Salmonellosis." *New England J. Medicine.* 299:459-460.

Environmental Protection Agency. 1987. *Unfinished Business: A Comparative Assessment of Environmental Problems.*

Environmental Protection Agency. 1990. *The Report of the Human Health Subcommittee, Appendix B.* EPA SAB-EC-90-021B.

Fisher, A., L. G. Chestnut, and D. M. Violette. 1989. "The Value of Reducing Risks of Death: A Note on New Evidence." *J. Policy Anal. Mgt.* 8(1):88-100.

Food Chemical News. 1990. "Foodborne Illness Among Elderly, AIDS Patients Seen Likely to Grow." *Food Chemical News.* 31(52):25-26.

Garthright, W. E., D. L. Archer, and J. E. Kvenberg. 1988. "Estimates of Incidence and Costs of Intestinal Infectious Diseases in the United States." *Public Health Report.* 103(2):107-116.

Haas, C. N. 1983. "Estimation of Risk Due to Low Doses of Microorganisms: A Comparison of Alternative Methodologies." *Am. J. Epidemiology.* 118(4):573-582.

Hammitt, J. K. 1986. *Estimating Consumer Willingness to Pay to Reduce Foodborne Risk.* Rand Corporation; Santa Monica, CA. (Prepared for the U.S. Environmental Protection Agency.)

Harrington, W., A. J. Krupnick, and W. O. Spofford, Jr. 1985. "The Benefits of Preventing an Outbreak of Giardiasis Due to Drinking Water Contamination." ELI/EPA Seminar, Washington, D.C. 18 July.

Harrington, W., and P. Portney. 1987. "Valuing the Benefits of Health and Safety Regulation." *J. Urban Econ.* 22:101-112.

Hauschild, A. H. W., and F. L. Bryan. 1980. "Estimate of Cases of Food- and Waterborne Illness in Canada and the United States." *J. Food Protection.* 43(6):435-440.

Howe, C. W. 1990. "The Social Discount Rate." *J. Environ. Econ. & Mgt.* 18(2):S-1-2.

Kramer, C. S., and K. P. Penner. 1986. "Food Safety: Consumers Report Their Concerns." *National Food Review.* NFR-33:21-24.

Kvenberg, J. E., and D. L. Archer. 1987. "Economic Impact of Colonization Control on Foodborne Disease." *Food Tech.* 41(7):77-98 (not consecutive).

Landefeld, J. S., and E. P. Seskin. 1982. "The Economic Value of Life: Linking Theory to Practice." *Am. J. Public Health.* 72(6):555-566.

Mauskopf, J. A., R. W. Leukroth, M. T. French, K. D. Fisher, A. S. Ross, C. R. Hollingsworth, and D. M. Maquire. 1988. "Estimating the Value of Consumer Loss from Foods Violating the FD&C Act." Prepared for the Food & Drug Administration.

Mitchell, R. C. and R. T. Carson. 1989. *Using Surveys to Value Public Goods: The Contingent Valuation Method.* Washington, D.C.: Resources for the Future.

Mossel, D. A. A. 1988. "Impact of Foodborne Pathogens on Today's World, and Prospects for Management." *Animal and Human Health* 1(1):13-23.

National Academy of Sciences. 1969. *An Evaluation of the Salmonella Problem.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1990. *Cattle Inspection.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1988. *Complex Mixtures - Methods for in Vivo Toxicity Testing.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1985. *Meat and Poultry Inspection: the Scientific Basis of the Nation's Program.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1987a. *Poultry Inspection:the Basis for a Risk-Assessment Approach.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1987b. *Regulating Pesticides in Food: The Delaney Paradox.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1983. *Risk Assessment in the Federal Government: Managing the Process.* Washington, D.C.: National Academy Press.

National Academy of Sciences. 1984. *Toxicity Testing - Strategies to Determine Needs and Priorities.* Washington, D.C.: National Academy Press.

Office of Management and Budget. 1990. *Regulatory Program of the United States Government: April 1, 1990 - March 31, 1991.* Washington, D.C.: Executive Office of the President.

Rice, D. P., E. J. MacKenzie, and Associates. 1989. *Cost of Injury in the United States: A Report to Congress.* San Francisco, CA: Institute for Health and Aging, University of California and Injury Prevention Center, The Johns Hopkins University.

Roberts, T. 1987. "Salmonellosis Control: Estimated Economic Costs." *Poultry Science.* 67:936-943.

Roberts, T. 1989. "Human Illness Costs of Foodborne Bacteria." *Am. J. Agri. Econ.* 71(2):468-474.

Roberts, T., and P. Foegeding. 1990. "Risk Assessment for Estimating the Economic Costs of Foodborne Disease Caused by Microorganisms." Paper presented at *The Economics of Food Safety* workshop, Alexandria, Virginia, June 4-6.

Roberts, T. and J. K. Frenkel. 1990. "Estimating Income Losses and Other Preventable Costs Caused by Congenital Toxoplasmosis in People in the United States." *J. of Am. Vet. Med. Assoc.* 196:249-256.

Roberts, T., and R. Pinner. 1990. "Economic Impact of Disease Caused by *Listeria monocytogenes.*" In, *Foodborne Listeriosis.* A. J. Miller, J. L. Smith, and G. A. Somkuti, ed. Amsterdam, The Netherlands: Elsevier Science, 137-149.

Shandera, W. X., J. P. Taylor, T. G. Betz, and P. A. Blake. 1985. "An Analysis of Economic Costs Associated with an Outbreak of Typhoid Fever." *Am. J. Public Health.* 75:71-73.

Smallwood, D. 1989. "Consumer Demand for Safer Foods." *National Food Review.* USDA/ERS. 12(3):9-11.

Thompson, M.S. 1986. "Willingness To Pay and Accept Risks to Cure Chronic Disease." *Am. J. Public Health.* 78(4):392-396.

Todd, E. C. D. 1988. "Botulism in Native Peoples—An Economic Study." *J. Food Protection.* 51:581-87.

Todd, E. C. D. 1989. "Preliminary Estimates of Costs of Foodborne Disease in the United States." *J. Food Protection.* 52(8):595-601.

Vital Statistics of the United States—Mortality Tables and Life Expectancy Tables. 1984. U.S. Dept. of Health and Human Services, Public Health Service. Hyattsville, MD: National Center for Health Statistics.