Incidence and Cost of Foodborne Diarrheal Disease in the United States

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ABSTRACT

An estimated 68.7 to 275 million cases of diarrheal disease episodes from all causes occur annually in the United States, representing an average of 0.29 to 1.1 cases per person per year. The total number of cases of foodborne origin and subsequent person-to-person transfer was estimated to be at least 24 million and perhaps as many as 81 million or more cases per year. Updating previously published patient cost estimates, including lost wages as well as direct medical costs, the average estimate-based value for food-associated illness is in the billions of dollars per year. Scientifically established chronic sequelae to diarrheal disease further increase the total economic burden but cannot be estimated from available data. Other associated clinical problems that are likely to be related to acute diarrheal episodes would further increase costs.

Hauschild and Bryan (40) estimated the cases of foodborne and waterborne illness in the United States to be 1.4 to 3.4 million per year. The estimate was based on results of follow-up surveys conducted by the Centers for Disease Control (CDC) after various disease outbreaks. The ratio of estimated cases vs. those initially reported for all foodborne diseases was 25:1 (40). Todd (79) estimated the number of foodborne disease cases in the U.S. to be 5 million per year.

The economic costs of foodborne disease have also been determined (29,55). Most recently, Todd (79) estimated the cost of all foodborne disease in the U.S. to be $1 billion to $10 billion annually; this figure included such factors as direct medical costs, lost wages and productivity, and industry loss through embargo, voluntary destruction and recall.

Surveillance of foodborne disease in the U.S. is a passive, voluntary system wherein states may request the assistance of CDC. It is generally accepted that the true incidence of foodborne disease is underreported and that economic loss estimates, which are directly dependent on incidence estimates, may also be underestimated. This report estimates the incidence of total diarrheal disease in the U.S. by extrapolation from total incidence data of the National Center for Health Statistics (NCHS) and foodborne diarrheal disease incidence from the most current data available, with consideration of new pathogens and knowledge of their pathogenic mechanisms.

Previous reports, which dealt with extrapolations from foodborne incidence data only, could not present incidence estimates from the perspective of total diarrheal disease, and chronic conditions associated with or following diarrheal disease were not considered with regard to their impact on morbidity and mortality. Our estimate of total and foodborne diarrheal disease is the strongest argument to date for increased surveillance and research.

SCOPE OF CONCERN—SYMPTOMOLOGY AND SPREAD

Although diarrhea may be of short duration and self-limiting, it may in some instances, depending on the nature of the infecting organism or the age and health of the host, be protracted and lead to other disorders. Infection need not always present itself as diarrhea, e.g., yersiniosis may be present as severe abdominal pain without diarrhea (77). Symptoms of vomiting and fever are highly variable (12,77) with disease of foodborne origin.

Diarrheal disease is transmitted through food and water and person-to-person (fecal-oral) contact and from living animals to man. The latter is a minor consideration in overall incidence. Much diarrheal disease previously thought to be due to person-to-person transmission is actually foodborne. It is often difficult to determine the ultimate source of an outbreak, that is, food may be the source, followed by person-to-person spread. It seems illogical to separate these two transmission modes since it is impossible in many instances to determine what proportion of total disease is caused by each. It seems more appropriate to consider that diarrhea transmitted person-to-person with foodborne infection as the principal source is a disease of foodborne origin; this is particularly true when foodborne transmission is indicated epidemiologically.
INCIDENCE OF SYMPTOMS

A "National Ambulatory Medical Care Survey" for the U.S. was conducted by NCHS for 2 years (1977-1978) (65). Questionnaires completed by a cross-section of physicians nationwide yielded the following information. The number of persons per year with the given symptom as the principal reason for the visit: abdominal cramps/pain, 8,783,500; diarrhea, 2,423,500; and vomiting, 1,873,000 (65). The most frequently stated second or third complaints were nausea or diarrhea. Significantly, 362,500 visits/year involved gastrointestinal bleeding as the principal reason. In summary, of the 2,423,500 principal complaints of diarrhea per year for 1977-78, 1,332,000 were diagnosed as diarrheal disease. Another 676,000 were diagnosed as diarrheal disease when abdominal cramps/pain was indicated as the principal complaint; 285,000 females with vomiting as principal symptom were also so diagnosed (data unavailable for males). An additional 454,500 diagnoses of diarrheal disease were made when principal complaints were nausea or stomach cramps, pain or spasm (65). In total, approximately 2,750,000 cases of physician-diagnosed diarrheal disease occurred annually for 1977-78.

ESTIMATING TOTAL DIARRHEAL DISEASE IN THE U.S.

NCHS data indicate conservatively that 2.75 million annual diagnoses of diarrheal disease were made by physicians for 1977-1978 (65). The question then becomes, How many people seek a physician's care for diarrhea or an associated symptom (e.g., abdominal cramps, vomiting, nausea)? The answer depends on many variables such as severity and duration of symptoms, which may relate to pathogen dose, the nature of the pathogen, health status/age of the host and many intangible factors such as educational level, economic status and affordability of health care. For example, it was clearly shown that most children attending day-care centers were sent to the centers regardless of diarrhea (67). Of the foodborne disease-associated symptoms, abdominal pain was the predominant reason that persons sought medical attention. Although severe abdominal pain plus occult blood and fecal leukocytes are strongly associated with invasive pathogens (57), diarrhea does not always result. Persons with watery diarrhea only are unlikely to seek medical care unless it persists. By then, it is usually too late to obtain valid culture results. In fact, totally asymptomatic infections can occur, which can be determined only by serological or stool culture methods. Probably only one person in 25 (at best) or one in 100 (at worst) seeks medical attention. It is likely then that between 68.7 million (2.75 million diagnoses × 25) and 275 million (2.75 million diagnoses × 100) actual cases occur annually in the U.S.

This estimate is consistent with an annual rate of 224 million clinically significant enteric infections estimated in an unpublished report of the Health Policy Task Force, August 1984. (See Acknowledgments)

INCIDENCE OF FOODBORNE DISEASE

Hauschild and Bryan (40) calculated the number of human cases of salmonellosis per year in the U.S., using data obtained from post-outbreak questionnaires, and derived a ratio of 29.5:1 for the estimated ill to the initial human isolations; this ratio was designated Rj. The data on which this ratio was based are presented in the principal reference (40). The average number of human Salmonella isolations/year during the period 1969 to 1978 was 25,000/year. Applying the Rj, Hauschild and Bryan (40) calculated an average number of estimated cases of salmonellosis for any year during the period 1969-1978 to be 740,000 cases/year (29.5 × 25,000). The number of human isolates of Salmonella in 1983 was 38,888 (21); application of the Rj would predict that 1,147,000 human cases of salmonellosis probably occurred in 1983.

Campylobacter jejuni is a recently recognized, important cause of foodborne disease in the U.S. and abroad. A recent case-control study of salmonellosis and campylobacteriosis conducted in Seattle, WA (72) reported that the incidence of campylobacteriosis was 2.5-times greater than that of salmonellosis. Other investigators have concluded that the incidence of campylobacteriosis exceeds the combined total cases of salmonellosis plus shigellosis (12,76). If the figures were extrapolated to the entire country it would mean that approximately 2,867,000 cases of campylobacteriosis occurred in the U.S. in 1983 (2.5 × 1,147,000 estimated salmonella cases). Campylobacter isolations from humans are not reportable to CDC; therefore estimates based on Rj cannot be made.

Shigella isolates from humans reported to CDC in 1983 numbered 14,946 (22). Shigellosis is thought to be transmitted primarily person-to-person; however, foodborne and waterborne outbreaks have been documented. Applying the Salmonella Rj of 29.5 (assuming its applicability) 440,900 cases of shigellosis would have occurred in 1983. If the estimates for diseases caused by Salmonella, Campylobacter and Shigella in 1983 are totalled, 4,455,000 cases of diarrhea resulted from these three pathogens.

Other well-known foodborne pathogens, such as Staphylococcus aureus, Clostridium perfringens, Yersinia enterocolitica, Bacillus cereus and Vibrio parahaemolyticus, accounted for 33.9, 13.4, 3.8, 0.9 and 0.2%, respectively, of confirmed foodborne disease cases in 1981 (20). That same year, C. jejuni accounted for only 5.6% of reported cases. Missing altogether were pathogenic Escherichia coli, Vibrio vulnificus, Cryptosporidium and enteroviruses, all of which were involved in diarrheal disease outbreaks and, in some cases, deaths in 1983, e.g., the E. coli strains that caused nationwide sporadic outbreaks of hemorrhagic colitis (24,26,66). Y. enterocolitica, a recognized cause of gastroenteritis and
FOODBORNE DIARRHEAL DISEASE

Estimating foodborne diarrheal disease requires assumptions and extrapolations; it also requires a definition. If a person acquires diarrheal illness through food consumption and subsequently spreads the disease through person-to-person contact, logically all involved persons in the outbreak could be scored as illness due to a food source. A perfect example of the difficulties in determining the role of foodborne vs. person-to-person contact can be found again in day-care centers. A follow-up study of day-care centers in the Houston area was recently conducted by Lemp et al. (54); several of the authors had been involved in the original Houston study by Pickering et al. (67). In the original survey, *Shigella*, rotavirus and *Giardia* were the leading causative agents of diarrhea (67); teachers caring for ill infants and children were felt to be responsible for person-to-person transmission of disease (67). In the follow-up study from October 1980 to May 1981, rates of diarrhea increased somewhat, with the highest incidence rate in day-care centers where diapering by staff was combined with food preparation or service (54). Since only diarrhea occurring outside the centers and reported to the centers by parents was included in this study, Lemp et al. (54) believe that the incidence of 0.68 case per person per year is probably an underestimate. Much of what may have been considered person-to-person transmission, therefore, may be linked directly or indirectly to food; this may be particularly true for the rotavirus diarrhea in the Houston studies (54). In contrast, in the recently reported outbreak of Norwalk agent diarrhea in Florida, where a common food source was clearly identified, little evidence of person-to-person spread was noted (56).

If the foodborne rate (4,455,000 cases/year, based on *Salmonella, Campylobacter* and *Shigella*) is doubled to account for all other known pathogens transmitted by food, a trend with a historical basis, 8,910,000 foodborne diarrhea cases/year result. It would seem equally valid to again double this number, since recent history indicates that a causative agent can be found in only 1/2 of all medically investigated cases; yet evidence in the form of occult blood and fecal leukocytes indicates the presence of enteric pathogens. This second doubling factor may also account for those cases in which overt diarrhea is absent but other symptoms are present (e.g. cramps, fever, nausea). Thus approximately 18,000,000 cases/year is not an unlikely figure. One-third again as many persons may contract illness owing to person-to-person transfer subsequent to primary foodborne disease acquisition; this could raise the number of cases/year directly (or indirectly, depending on one's views) to 24,000,000 illnesses/year attributable to foodborne pathogens. This estimate assumes that the $R_t = 29.5$ (for *Salmonella* and *Shigella*) of Hauschild and Bryant (40) is valid. The authors point out, however, that in several large outbreaks, the ratio of estimated cases of salmonellosis to the...
number of reported human isolations was 100:1. If this is assumed to be a worst-case situation, the 24,000,000 estimate (based on \( R_f = 29.5 \)) may actually be as high as 81.36 million cases/year (based on a worst-case \( R_f \) of 100). In the light of all the facts concerning variables and difficulties associated with estimating true incidence, plus new epidemiologic data on virus transmission (54,56), this estimate does not seem unreasonable and in fact may still be conservative. Greater efforts should be made to determine the incidence of all diarrheal and foodborne diarrheal disease in the U.S. Surveillance data on infectious diseases provide public health officials with a valuable and irreplaceable source of information on which to make priority decisions; efforts must be intensified, not curtailed.

Despite vastly improved recovery techniques and the recognition of new enteric pathogens that affect large numbers of persons, the proportion of cases of unknown etiology remains constant, suggesting either that our present methods are inadequate for some causative agents or that as yet unrecognized causative agents exist. Why are funds not available for concerted efforts to determine the true incidence of foodborne diarrheal disease? The answer probably stems from the poor image of diarrheal disease held by many health professionals. Diarrhea is all too often thought of as an unpleasant nuisance, a self-limiting, acute disease with no long-lasting consequences.

### TABLE 1. Rheumatic conditions as sequellae to enteric infections.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Target populationa</th>
<th>Estimated % affected as sequellae to enteric infection</th>
<th>Duration of symptoms</th>
<th>Genetic predisposition</th>
<th>Reviews</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seronegative spondarthropathies</td>
<td>P/A</td>
<td>2-3% Sal, Cj, Shig (47), 2-10% Cj (38); 33% Ye (47) Eh and Gl? (47); asymptomatic Ye (31, 53); Yp (53)</td>
<td>6 mo (47); 22 mo (53); 4 to 5 yr (63)</td>
<td>HLA-B27 (47)</td>
<td>(4, 47, 52)</td>
</tr>
<tr>
<td>Reactive arthritis</td>
<td></td>
<td></td>
<td></td>
<td>HLA-B27 other (53)</td>
<td></td>
</tr>
<tr>
<td>Reiter’s syndrome (complete)</td>
<td>A/P</td>
<td>&lt;0.1% for all three symptoms (6) in total population; same as for reactive arthritis (3, 47). Causes: Shig, Sal, Ye, Yp, Cj, Kp? (3)</td>
<td>variable with symptoms; urethritis &gt; conjunctivitis &gt; arthritis, &gt;1 mo by definition (3) 30-50% Reiter’s cases relapse for mo-yr, 15% for 30 yr (3)</td>
<td>HLA-B27 (47)</td>
<td>(3, 4, 46)</td>
</tr>
<tr>
<td>Guillain-Barré syndrome</td>
<td></td>
<td>Very rare sequellae to Reiter’s (34)</td>
<td></td>
<td>HLA-B27 other (47)</td>
<td></td>
</tr>
<tr>
<td>Ankylosing spondylitis</td>
<td>P/A</td>
<td>0.1%-1.5% (16, 46), Kp colonization role (4) Ye, Sal, Shig? (16, 46, 50)</td>
<td></td>
<td>HLA-B27 (4, 16)</td>
<td></td>
</tr>
<tr>
<td>Cardiac manifestations</td>
<td>A</td>
<td>&lt;Rate of AS, reactive arthritis; second only to coronary artery disease as cause of conduction disturbances (8); sequella to any seronegative arthropathy (8)</td>
<td></td>
<td>HLA-B27 + A2 (16)</td>
<td>47,50</td>
</tr>
<tr>
<td>Heart disease</td>
<td></td>
<td>From onset to death (9) aortic regurgitation, conduction disturbances, complete heart block; frequently requires pacemaking (8); mitral valve damage (70) can be fatal</td>
<td></td>
<td>HLA-B27 (8, 9)</td>
<td>(8, 9)</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>P/A</td>
<td>Can develop from reactive arthritis (53, 63); <em>Clostridium perfringens</em> alpha toxin? (1) rare: Sal, Ye, Cj</td>
<td></td>
<td>Life-long debilitating disease</td>
<td>HLA-DR4/DR5</td>
</tr>
<tr>
<td>Septic arthritis</td>
<td>P/A</td>
<td></td>
<td></td>
<td>Resolves with infection</td>
<td>None</td>
</tr>
</tbody>
</table>

*Abbreviations: P = pediatric, A = adult, Sal = Salmonella spp., Shig = Shigella spp., Cj = Campylobacter jejuni, Ye = Yersinia enterocolitica, Yp = Yersinia pseudotuberculosis, Kp = Klebsiella pneumoniae, Eh-Entamoeba histolytica, Gl = Giardia lamblia, AS = Ankylosing spondylitis, HLA = Human Leukocyte Antigen (see review in 4).*
Tables 1, 2 and 3 present some rheumatoid, nutritional and miscellaneous conditions, respectively, that are associated with or may occur after enteric infections. The amount of medical care required to treat these conditions varies greatly. In one outbreak of yersiniosis, no arthritis may be reported, whereas in another, a 4% incidence may be reported (75,77); this difference appears to be strain-dependent. In countries such as Belgium, where yersiniosis is endemic, particularly that caused by the O:3 serotype, the Y. enterocolitica serotype O:3 is thought to be the major cause of chronic joint disease (52). Human isolations of the O:3 serotype appear to be on the rise in parts of the U.S. (13). Current census and incidence figures (16) suggest that 250,000 to 3.5 million people in the U.S. have ankylosing spondylitis, another rheumatoid condition associated with enteric pathogens. These types of chronic diseases and their associated cost burdens accumulate in the population. Although the amount of arthropathy-associated heart disease in the U.S. has not been studied, it is significant in other countries (Table 1).

The nutritional and malabsorption conditions presented in Table 2 affect children more often than adults. The important factor is that any loss of nutritional status may greatly increase morbidity from other causes and generally erodes the overall health of the population. Particularly alarming is the apparent epidemic diarrheal disease (23,32,54,67), often with several pathogens in a single patient, that is affecting numerous day-care centers nationwide. Whether this is in part responsible for the increased incidence of cryptosporidiosis (25) in these facilities remains to be determined.

The recently increased number of sporadic outbreaks of hemorrhagic colitis (often associated with ground beef) and the sequelae, hemolytic-uremic syndrome, has caused health officials to speculate that the E. coli serotypes may be the source of many of the diarrheal diseases of unknown etiology (24). The appearance of other adherent, enteropathogenic E. coli with no known associated toxins but multiple antibiotic resistance is further cause for concern (27,51).

THE ECONOMIC IMPACT OF DIARRHEAL DISEASE IN THE U.S.

The report by Cohen et al. (29) concerning patient-related costs in a salmonellosis outbreak is frequently used as the basis for more recently published cost estimates of diarrheal disease. Those estimates were based on 1976 figures and allowed $10 for a doctor’s office visit and

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TABLE 2. Nutrition- and malabsorption-related sequelae to enteric infections.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Target population</th>
<th>Estimated % affected as sequelae to enteric infection</th>
<th>Duration of symptoms</th>
<th>Reviews or refs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure to thrive with enteropathy</td>
<td>P^a</td>
<td>2-3% of pediatric acute gastroenteritis patients (37)</td>
<td>Reversible with proper diagnosis; life-long growth retardation possible. (10,37,45,68)</td>
<td></td>
</tr>
<tr>
<td>Failure to thrive without enteropathy</td>
<td>P</td>
<td>6% with acute gastroenteritis (37)</td>
<td>Reversible with proper long-term nutritional support; 3 wk (mean) (37)</td>
<td></td>
</tr>
<tr>
<td>Malabsorption (mild to severe)</td>
<td>P/A</td>
<td>By definition, always occurs with G.I. tract damage</td>
<td>1 wk to 1 yr depending on agent and other complications (15,27)</td>
<td></td>
</tr>
<tr>
<td>Disaccharide intolerance</td>
<td>P</td>
<td>40% with giardiasis (82); 64% with CNSD (69); 10% with acute gastroenteritis (37); highly variable</td>
<td>During acute episode to 1-5 mo, mean 3 mo (37); 6 mo (71)</td>
<td></td>
</tr>
<tr>
<td>Monosaccharide intolerance</td>
<td>P</td>
<td>4-9% acute gastroenteritis admissions (43,61) 100% with protracted diarrhea</td>
<td>2-3 d as sequela to acute gastroenteritis; 21 d (mean) up to 70 d if develops during protracted diarrhea (61)</td>
<td></td>
</tr>
<tr>
<td>Hyperatraemia</td>
<td>P</td>
<td>0.9% with acute gastroenteritis (becoming far less of a problem)</td>
<td>Readily reversible with proper treatment, fatal if untreated (62)</td>
<td></td>
</tr>
<tr>
<td>Achlorhydria</td>
<td>P&gt;A</td>
<td>High incidence in chronic diarrhea, moderate to nil in acute (59)</td>
<td>May lead to contaminated small bowel syndrome (36)</td>
<td></td>
</tr>
<tr>
<td>Cow’s milk intolerance</td>
<td>P</td>
<td>High incidence in children with occult blood or enteropathy (28)</td>
<td>May be long-term syndrome (80); may cause postenteritis diarrhea (38,80)</td>
<td></td>
</tr>
<tr>
<td>(non-allergenic) Postenteritis diarrhea</td>
<td>P</td>
<td>Dependent on presence of milk proteins in diet (80)</td>
<td>Depends on avoidance of cow’s milk protein (80) (39,80)</td>
<td></td>
</tr>
<tr>
<td>Intractable diarrhea syndrome</td>
<td>P</td>
<td>Treatable; starts with enteropathy; protracted; of unknown etiology (71)</td>
<td>8 d to 6 mo (71) (71)</td>
<td></td>
</tr>
<tr>
<td>Steatorrhea</td>
<td>P&gt;A</td>
<td>Probably very high incidence (58)</td>
<td>About 5 d (45); may lead to failure to thrive (45,58)</td>
<td></td>
</tr>
<tr>
<td>Food allergy</td>
<td>P&gt;A</td>
<td>Unknown; probably fairly high with any enteropathic condition</td>
<td>Unknown; reverses in time in some, not in others (33,42)</td>
<td></td>
</tr>
</tbody>
</table>

^Abbreviations: P = pediatrics, A = adult, CNSD = chronic nonspecific diarrheal syndrome.

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TABLE 3. Miscellaneous conditions associated with or sequelae to enteric infections.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Target population</th>
<th>Estimated persons affected with enteric infection (%)</th>
<th>Duration of symptoms</th>
<th>Reviews or refs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemolytic-uremic syndrome</td>
<td>P/A</td>
<td>4% with shigellosis (49); 15% with hemorrhagic colitis (66). Varies with organism. Causes: Ec 0157:H7, Cj, Shig, EV, possibly any enteric pathogen (see 30)</td>
<td>4-17 d hospitalization (66); can be fatal. On the rise in U.S. (26); expected to increase (24)</td>
<td>(26,30,66)</td>
</tr>
<tr>
<td>Hypogammaglobulinemia</td>
<td>P</td>
<td>18% with chronic-severe diarrhea (35)</td>
<td>May last 2-4 yr post-onset; contributes to high morbidity and fatal infection</td>
<td>(35)</td>
</tr>
<tr>
<td>Septicemia</td>
<td>P/A</td>
<td>Highly variable; any invasive agent; underlying health disorder increases risk</td>
<td>Up to 2 wk antibiotic therapy; can be fatal 38-67% (2)</td>
<td>(2,74)</td>
</tr>
<tr>
<td>Seizures - febrile</td>
<td>P</td>
<td>In acute cases 12-45% (5), 23.4% (5)</td>
<td>Short episodes preceding diarrhea</td>
<td>(5)</td>
</tr>
<tr>
<td>afebrile</td>
<td>P</td>
<td>Rare</td>
<td>Short episode pre-diarrhea in Cj; may recur</td>
<td>(600)</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>A</td>
<td>Relatively rare</td>
<td>Heart valve damage; permanent murmur</td>
<td>(2)</td>
</tr>
<tr>
<td>Colitis</td>
<td>P/A</td>
<td>Highly variable; almost any invasive/toxic organism; Cj, Ah pseudomembranous, Cl.d (7)</td>
<td>Transient to permanent; may not be antibiotic-related; 8-10 wk</td>
<td>(44,57,64)</td>
</tr>
<tr>
<td>Pseudoappendicitis</td>
<td>P/A</td>
<td>0-10%? (18)</td>
<td>Hospitalization up to 2 wk; unnecessary surgical procedure can lead to septicemia</td>
<td>(11,18,19)</td>
</tr>
<tr>
<td>Chronic nonspecific diarrhea</td>
<td>P</td>
<td>20-25% with acute gastroenteritis (37) could be Ec-related (69)</td>
<td>4-20 mos (69)</td>
<td>(69)</td>
</tr>
<tr>
<td>Chronic diarrhea - specific</td>
<td>P/A</td>
<td>16-17% with acute gastroenteritis (37); Ec 0111:K58:H-(27); Ec 0111:K58:H2(57)</td>
<td>2 wk (27) to 25 ±15 d (51); can lead to urinary tract infection; antibiotic-resistant; recurs frequently</td>
<td>(27,51)</td>
</tr>
<tr>
<td>(misnomer)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: P = pediatric, A = Adult, Ec = Escherichia coli, Cj = Campylobacter jejuni, Ah = Aeromonas hydrophila, EV = enteric viruses, Cl.d = Clostridium difficile.

$139/day for hospital care. At the end of 1984, the same figures would be $30 to $35 and $400 to $410, respectively (American Hospital Association Annual Survey for 1983 and Office of Research, Health Care Financing Administration, DHHS), and income and productivity losses would be at least 40 to 50% higher. The estimated total cost to the patient of $675/person would be 2.5 to 3 times greater, or $1687 to $2025/person. Thus the total cost of salmonellosis in the U.S., based on current cost estimates and 1983 incidence figures, would be $1.9 to $2.3 billion. Cohen et al. (29) had factored in the cost of persons not hospitalized and did not seek a doctor’s care as $125; this figure, which has also undergone inflation, was factored into the overall cost/person estimate of $675. The salmonellosis outbreak used for the calculations of Cohen et al. (29) was an average outbreak with variable hospitalization rates and lengths covering the entire age spectrum.

Even when children are the victims, a parent must often miss work to care for the child. The loss of productivity is not directly patient-related and is difficult to quantify. Other circumstances relative to foodborne disease costs cannot be accounted for. For example, if the general nutritional and health status of children erodes because of diarrheal disease, the increase in morbidity caused by other diseases and the resultant cost would be impossible to quantitate. The cost of disability in rheumatoid diseases, although impossible to estimate, is significant. Another set of variables includes diseases which may be associated with foodborne microorganisms. For example, Y. enterocolitica may trigger thyroid disease (81), and E. coli and Klebsiella pneumoniae may play a role in myasthenia gravis (78). More such associations may be found and eventually proven. These factors will undoubtedly impact greatly on total cost estimates.

Diarrheal disease from foodborne sources is generally preventable. Any money spent on research, surveillance and public education would be only a small fraction of the cost otherwise borne by the economy when disease occurs. This was recently demonstrated dramatically in a report (73) on a foodborne typhoid fever outbreak which showed that the costs incurred by five patients offset the cost of detection and prevention. In most instances detailed by Todd (79) in 1983, food supplier
losses in the form of recalls, lost business, legal fees, legal settlements and wages exceeded the medical costs and lost earnings of the victims; thus the indirect cost to the economy may equal or even double the patient-related costs. Estimating the true cost of diarrheal illness and all associated economic costs is a complex task that will be dealt with at a later date.

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REFERENCES


