Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
It was not until the first part of the 20th century that gastrointestinal diseases were finally controlled to an acceptable level in most developed countries. Multiple factors led to this level of control: wastewater disposal, personal hygiene improvements, pasteurization of milk, and advances in food preparation and storage. The availability of high-quality tap water in every household was also a crucial step. Showers, baths, and toilets reduced the level of fecal contamination in the household and allowed persons to cleanse themselves easily and frequently. Although it is still assumed that a significant proportion of gastrointestinal illnesses is waterborne and foodborne, we do not have enough data to estimate the proportion of the overall burden of disease by route of exposure. Available data pertain mostly to outbreaks (waterborne, foodborne, or contagious), but these data are only the tip of the iceberg. In a holistic approach, an action relating to one of the exposure routes should result in a measurable reduction of gastrointestinal illnesses associated with the other transmission routes. This reduction of gastrointestinal illnesses is mainly the result of limiting subsequent person-to-person transmission, thereby reducing the risk of contaminating food and surfaces.

At the onset of the 21st century, what do we know of the impact of gastrointestinal illnesses on our societies? Thousands of outbreaks caused by bacterial, viral, and parasitic microorganisms are still associated with the consumption of contaminated food or water. The World Health Organization (WHO) estimated that in 1996 a child died from a water-related disease every 8 seconds, and that each year more than 5 million people die from illnesses linked to inadequate sanitation. WHO also suggests that if sustainable, safe drinking water and sanitation services were provided to all people, each year there would be 200 million fewer episodes of diarrhea and 2.1 million fewer deaths caused by diarrhea.

**MICROORGANISMS AND OTHER CAUSES OF GASTROINTESTINAL ILLNESSES**

Hodges et al offered a table of circumstances explaining the gastrointestinal symptoms observed in the city of Cleveland: 116 of 362 cases were caused by acute infectious diseases, 63 by dietary indiscretion, 59 by coughing or gagging, 45 by medication, 18 by emotional causes, and 61 were of unknown origin. The etiologic agents of acute diarrheal disease are well described in the literature and include parasites (eg, *Cryptosporidium parvum*, *Giardia lamblia*, *Cyclospora histolytica*, and *Entamoeba histolytica*), bacteria (eg, *Salmonella*, *Shigella*, *Campylobacter*, *Vibrio cholerae*, enterovirulent *Escherichia coli*, *Aeromonas*, *Yersinia*, *Bacillus perfringens*, and *Clostridium perfringens*), and viruses (eg, enteroviruses, rotaviruses, parvoviruses, adenoviruses, calcivirus, and astroviruses). Pathogens can be transmitted to humans by water, person-to-person contact, animal-to-human contact, food, and aerosols. Parasites are identified as pathogens of importance in all countries, and numerous waterborne outbreaks of giardiasis and cryptosporidiosis have been reported in the United States and the United Kingdom. Infectious agents are not the only cause of acute diarrheal disease. Milk or soy protein intolerance, food abuses or diet changes, adverse
effects of prescription drugs (especially antibiotics), as well as fungal, algal, or shellfish toxins may all cause diarrhea. A number of chemicals can also induce gastrointestinal symptoms, such as monosodium glutamate, organic mercury, antimony, and copper.5,6

INCIDENCE OF GASTROINTESTINAL ILLNESS IN INDUSTRIALIZED COUNTRIES

In general, deriving a good estimate of the incidence of endemic acute gastrointestinal disease in a community is very difficult. Symptoms develop in only a few of the persons who are infected, and only a proportion of these persons will seek medical attention. The doctor may not report the illness, or if samples are taken they may fail to reveal a pathogen. If a pathogen is identified, the result may or may not be reported to a surveillance system (if such a system exists). Only a small proportion of acute episodes reach this level. Existing surveillance systems are thus likely to significantly underestimate the real burden of gastrointestinal disease.7,8 Few studies have investigated the incidence of gastrointestinal disease at the community level, and most of these studies have been conducted in North America. These studies include the Cleveland study,4,9 the Tecumseh study,10,11 and the Virus Watch Program.12 Reported gastrointestinal illness rates were in the range of 0.5 to 2 episodes per year per person, varying from 5 to 100 episodes per 1000 persons per week, according to seasons and age. The number of episodes of gastrointestinal illnesses reported in these studies is similar to the number reported in some recent epidemiologic studies. A seasonal pattern of gastrointestinal illness is often observed. Adults are usually the least susceptible, and children younger than 5 years are the most susceptible. Peaks can be observed in children in autumn, whereas values as low as 5 episodes per 1000 persons per week are reported in summer.

Since the 1950s, with the development of methods to detect and identify viruses, many outbreaks of gastrointestinal illness that would have been simply classified as nonbacterial in origin have been attributed to enteric viruses such as hepatitis A and E, astroviruses, caliciviruses, and many others. Enteric viruses are excreted through the feces in the environment by infected persons with or without clinical illness. The enteric viruses include more than 100 types: enteroviruses (eg, poliovirus, coxsackievirus, echovirus, and hepatitis A), reovirus, rotavirus, adenoviruses, coronavirus, calicivirus, astrovirus, coronavirus, and Norwalk-like agents.13 Health effect studies have provided data on the prevalence of antibodies to several enteric viruses in US populations.14 Data indicate that the hepatitis A virus is an infection acquired progressively in life, and that in the United States, relatively few children have antibodies to this virus. In contrast, these infections generally are acquired early in life by people in other countries because of low hygiene levels.

INCIDENCE OF ENDEMIC GASTROINTESTINAL DISEASE IN OTHER COUNTRIES

Getting a clear understanding of the incidence of diarrhea in developing countries is even more difficult than it is in industrialized countries. Using the results of available studies to produce an overall estimate of diarrheal disease in developing countries is difficult for a number of reasons. Levels of diarrheal disease may markedly differ between relatively close communities because of different socioeconomic factors, such as the availability of a clean water supply and hygiene behavior. Estimates of diarrheal disease incidence from a variety of prospective epidemiologic studies indicate a range of 1 to 25 episodes per person per year. It seems clear that the incidence is higher in poorer communities and in rural environments than in urban environments. The age distribution of persons with diarrheal disease in developing countries seems to be similar in all regions in which this information reported. Disease incidence is relatively low in the first few months of life, and then peaks at about 24 months before declining toward adulthood. Travelers to developing countries are at risk.15,16 Data from various countries indicate a similar incidence of 1 to 15 episodes per person per year for travelers, suggesting a high degree susceptibility and a high attack rate. This difference is even more notable given the fact that travelers usually live in more hygienic surroundings than do local persons. Thus, the evidence presented supports the hypothesis that local people build up a substantial immunity to the enteropathogens circulating in their communities. However, a consequence of achieving this level of immunity there is that young children in developing countries have a substantially higher incidence of illness than do children in developed nations. This high incidence of gastrointestinal disease in children is one of the reasons behind the high childhood mortality rates in developing countries.

The proportion of diarrheal disease caused by the various routes of exposure varies substantially between communities because of varying behavioral and socioeconomic factors. Despite the importance of sanitation and hygiene, a significant proportion of diarrheal disease could be related to water quality. Rivers are perhaps the source of the poorest quality water. In southeastern China17 and Uzbekistan,18 diarrhea related to the source of drinking water was estimated to comprise about 85% of the illnesses in people who drank river
water. In the Philippines, Moe et al. reported that improved drinking water would have little or no effect in areas with poor environmental sanitation. In areas with good community sanitation, reducing fecal coliform counts by 2 orders of magnitude would result in a 40% reduction in the incidence of diarrhea; eliminating excreta from around houses would result in a 30% reduction; and providing private excreta disposal would result in a 42% reduction.

**ELIMINATING GASTROINTESTINAL DISEASES**

Because most of the etiologic agents responsible for gastrointestinal illness are present in fecal material and biologic fluids, such as saliva, part of the effort to achieve a significant reduction in these diseases must be directed toward minimizing contact with these fomites. In an unsanitary environment, contaminated fomites are everywhere. Furthermore, when the family or the community includes a large number of children, rates of circulation and transmission of infectious diseases are extremely high. Often these children have not received training in personal hygiene. The level of hygiene attained by a community will control gastrointestinal diseases to an equivalent level. Actions should be directed toward keeping surfaces (eg, floors, walls, tables, and fixtures) as clean as possible. Handwashing and other personal hygiene activities will prevent fecal material from being transferred to surfaces where pathogens can survive for various periods. Transmission of pathogens from one person to the other has been demonstrated to occur after a simple handshake; significant contamination can be transmitted to as many as 6 people. A single contaminated doorknob can act as the reservoir to contaminate an entire household. In a very hygienic community, very few persons will harbor pathogens at any given time. Even if fecal contamination of surfaces occurs, the fecal material does not contain pathogens most of the time. However, it can serve as an index of fecal contamination and poor personal hygiene in all environments.

Currently, antibacterial substances to prevent growth of bacteria on surfaces are being marketed. The use of such substances is purely cosmetic, because pathogens other than bacteria will be present. Viruses and parasites are unaffected by antibacterial chemicals and will still be present. Basic cleaning practices (ie, use of soap and water) remain the best approach to reducing the risk of contracting an illness. This approach applies to the personal level (handwashing), to the household level (cleaning the environment and preventing fecal contamination by animals), and to the community level (sanitation and preventing fecal contamination by animals).

**CONCLUSION**

Providing definitive estimates of the burden of diarrheal disease is not possible, because the burden varies substantially on the basis of a community's water source and hygiene practices, along with socioeconomic and behavioral factors. Access to a greater quantity of water will foster more hygienic behavior by permitting washing and reducing the amount of time spent collecting water. Although improved water quality may reduce waterborne disease, actions must be taken at the individual level. The causes of gastrointestinal disease are well described, and pathogenic microorganisms are the common cause. These pathogens originate from fecal material of infected persons. Thus, the burden of gastrointestinal disease can only be reduced through a better control of the fecal contamination of the environment at the individual, household, and community level.

**References**

1. Mara D, Cairncross S. Guidelines for the safe use of wastewater and excreta in agriculture and aquaculture. Geneva, Switzerland: World Health Organization; 1989.
2. Ford TE, Colwell RR. A global decline in microbiological safety of water: a call for action. Washington (DC): American Academy of Microbiology; 1996.
3. Anon. Water and sanitation: WHO fact sheet no. 112. Geneva; 1996.
4. Hodges RG, Mccorkle LP, Badger GF, Curtiss C, Dingle JH, Jordan WS. A study of illness in a group of Cleveland families. XI. The occurrence of gastrointestinal symptoms. Am J Hyg 1956;64:349-56.
5. Ellner PD. Infectious diarrhoeal diseases. Microbiol Ser 1981:12:1-175.
6. Branski D. Specific etiologies of chronic diarrhoea in infancy. Nestle Nutrition Workshop Series 1984;6:107-45.
7. Payment P, Richardson L, Siemiatycki J, Dewar R, Edwartes M, Franco E. A randomized trial to evaluate the risk of gastrointestinal disease due to the consumption of drinking water meeting currently accepted microbiological standards. Am J Public Health 1991;81:703-8.
8. Payment P, Siemiatycki J, Richardson L, Renaud G, Franco E, Prévost M. A prospective epidemiological study of gastrointestinal health effects due to the consumption of drinking water. Int J Environ Health Res 1997;7:5-31.
9. Dingle JH, Badger GF, Feller AE, Hodges RG, Jordan WS, Rammelkamp C. A study of illness in a group of Cleveland families. I. Plan of study and certain general observations. Am J Hyg 1953;58:16-30.
10. Monto AS, Koopman JS. The Tecumseh Study: XI. Occurrence of acute enteric illness in the community. Am J Epidemiol 1980;112:323-33.
11. Monto AS, Koopman JS, Longini IM, Isaacson RE. The Tecumseh study: XII. Enteric agents in the community, 1976-1981. J Infect Dis 1983;148:284-91.
12. Fox JP, Elveback LR, Wassermann FE, Kelter A, Brandt CD, Kogon A. The virus watch program. Am J Epidemiol 1986;83:389-412.
13. Payment P. Viruses: prevalence of disease, levels and source. In: Craun G, editor. Safety of water disinfection: balancing chemical
14. Payment P. Antibody levels to selected enteric viruses in a normal randomly selected Canadian population. Immunol Infect Dis 1991;1:317-22.
15. Ahlm C, Lundberg S, Fessé K, Wiström J. Health problems and self-medication among Swedish travellers. Scand J Infect Dis 1994;26:711-7.
16. Cobelens FGJ, Leentvaar-Kuijpers A, Kleijnen J, Countinho RA. Incidence and risk factors of diarrhoea in Dutch travellers: consequences for priorities in pre-travel health advice. Trop Med Int Health 1998;3:896-903.
17. Chen K, Lin C, Qiao Q, Zen N, Zhen G, Gongli C, et al. The epidemiology of diarrhoeal diseases in southeastern China. J Diarrhoeal Dis Res 1991;9:94-9.
18. Semenza JC, Roberts L, Henderson A Bogan J, Rubin CH. Water distribution system and diarrhoeal disease transmission: a case study in Uzbekistan. Am J Trop Med Hyg 1998;59:941-6.
19. Moe CL, Sobsey MD, Samsa GP, Mesolo V. Bacterial indicators of risk of diarrhoeal disease from drinking-water in the Philippines. Bull World Health Organ 1991;69:305-17.
20. VanDerslice J, Briscoe J Enviromental interventions in developing countries and their implications. Am J Epidemiol 1995;141:135-44.