tory of casual contact between its citizens and the travelers and noncitizens who reside there.

Hospitals have functioned as junctions for varied communities in spreading the SARS virus further. Because of SARS' likely place of origin, the initial "community" included Chinese persons who then kindled the chain of transmission to other communities throughout the world. Daily, close contact between SARS patients and hospital personnel led to an unusually large number of infections among medical staff members. Effective prevention measures such as vaccines are not available and may be a factor in the spread of the infection.

Even in the era of globalization and mass air transit, most persons live inside a relatively small circle of community, made up of others of similar ethnicity, religious beliefs, educational level, and social class who live in the same vicinity; this sort of small circle has been described as "mutual coexistence" by anthropologist Kinji Imanishi (1). Basically, the SARS-associated coronavirus began circulating among members of such a community. This theory does not suggest that certain ethnic groups are predisposed to be susceptible to SARS.

Why have few cases of SARS occurred in children? All age groups are susceptible to the SARS virus, which is new to humans. However, adults have more chance to become infected through contacts in their daily lives, whereas children do not. Rapid isolation of the adult patients contributed to reduced frequency of exposure for children in that household, which is in contrast to the usual infectious diseases of childhood (since children do not have immunity against many age-old microbes).

Some contradictions exist for our interpretation of the SARS transmission pattern. Investigations have shown that in Canada, Hong Kong and elsewhere, some casual brief contact caused the infection or that the

link between the source and the case was not at all clear. We may have missed other important routes of transmission, or a totally unknown element may be involved. Without an answer for this discrepancy, we note that the clinical virology for SARS, such as pattern of virus shedding and host immune response, is still developing (2). For example, a total of 19 cases in China were identified as SARS by coronavirus isolation, polymerase chain reaction, or serologic tests. For two case-patients, the results of three tests were positive; 10 case-patients had negative test results; and in 14 case-patients, the virus was not isolated. Interpreting these results is difficult. In the United States, 97% of the probable cases were attributed to a recent history of international travel to SARS-affected areas. Antibodies to SARS-associated coronavirus were demonstrated for 8 of 41 probable case-patients in convalescent-phase serum, bringing the proportion of laboratory-confirmed cases to 20%, even in the probable cases, and 0% among the suspected cases in the United States so far (3). These results are the best available by laboratories with the current limited technical knowledge. We are not persuaded that casual contact with SARS patients in unfamiliar settings results in contracting the disease.

The winter of 2003 will be critical for observing how the virus behaves, whether the winter climate accelerates the transmission, and how we handle that acceleration. Despite current global efforts, thin lines of transmissions may remain in China; the virus may flare up again. Officials in China and sites of the outbreak must interrupt as many chains of transmission as possible before October. Surveillance should also be intensified. Ongoing study to improve laboratory diagnosis and clinical virology is key, so that effective isolation can be practiced; at present, these measures are the only ones known to inter-

rupt the transmission of SARS. The group on which to focus should be the community in close contact with previous outbreak areas.

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Home-prepared Hamburger and Sporadic Hemolytic Uremic Syndrome, Argentina

To the Editor: Argentina has the highest incidence of hemolytic uremic syndrome (HUS) in the world, and 10.4 cases per 100,000 children <5 years of age were reported in 2001. HUS is the leading cause of acute renal failure in children (1); in 20% to 35% chronic renal failure develops, ranging from mild to serious, and HUS is the second leading cause of chronic renal failure (2,3) in Argentina. Recently, evidence of

Shiga toxin–producing *Escherichia coli* (STEC) infection was found in 59% of Argentine HUS cases; O157:H7 was the predominant serotype isolated (4). Although outbreaks of *E. coli* O157:H7 have been linked to eating contaminated ground beef (5), the organism is rarely isolated from the implicated meat, and the sources of infection for sporadic cases have rarely been identified. We report a sporadic HUS case linked to the consumption of home-prepared hamburger contaminated with *E. coli* O157.

A 2-year-old girl was brought to the emergency room of the Hospital Nacional de Pediatría "Prof. Dr. Juan Garrahan" in Buenos Aires on April 26, 2002, with a 1-day history of bloody diarrhea. Results of a physical examination were normal, and a stool culture was requested. The patient was sent home with dietary and general instructions. As watery diarrhea persisted with vomiting and fever, the girl was brought in again 3 days later. At that time, she exhibited moderate dehydration, pallor, drowsiness, and a generalized seizure of 10 to 15 min duration, tachycardia, tender and tense abdominal wall, and a history of oligoanuria for the last 48 h. Blood pressure was 128/67 mm Hg. The child was hospitalized with a presumptive diagnosis of HUS and anuric renal failure.

Initial laboratory findings included the following: hematocrit, 26%; hemoglobin level, 8.8 g/dL; leukocyte count, 34,800/mm^3; segmented neutrophil count, 29,928/mm^3; platelet count, 91,000/mm^3; serum glucose, 160 mg/dL; blood urea nitrogen (BUN), 268 mg/dL; serum creatinine, 6.3 mg/dL; albumin, 1.7 g/dL; uric acid, 14.8 mg/dL; calcium, 6.9 mg/dL; phosphorus, 6.7 mg/dL; magnesium, 2.0 mg/dL; sodium, 113 mEq/L; potassium, 7.6 mEq/L; pH 7.28; bicarbonate, 10 mmol/L; base excess, –14.9 mmol/L. Chest x-ray findings were normal with a cardio-thoracic index of 0.5; results of an abdominal sonogram were normal. A sonogram of the renal system also showed that the kidneys were of normal shape and size and had increased echogenicity. Results of a brain scan showed nonspecific brain atrophy.

The clinical findings and the laboratory features of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure were consistent with the diagnosis of HUS. The patient remained anuric for 17 days, required 17 peritoneal dialysis procedures, and six infusions of packed red blood cells. One month after the acute period, she had elevated BUN and serum creatinine levels and massive proteinuria.

The rectal swab sample collected on April 26 was routinely cultured for *E. coli*, *Salmonella*, *Shigella*, *Yersinia*, *Aeromonas*, *Plesiomonas*, *Vibrio,* and *Campylobacter* species. Sorbitol nonfermenting colonies were recovered on sorbitol-MacConkey (SMAC) agar (Difco Laboratories, Detroit, MI) and SMAC supplemented with cefixime (50 ng/mL) and potassium tellurite (25 mg/mL) (CT-SMAC). The bacterial confluent growth zones of both SMAC and CT-SMAC were positive for *stx*2 and *rfb*O157 genes by multiplex polymerase chain reaction (PCR) using the primers described by Pollard et al. (6) and Paton et al. (7), respectively. The *E. coli* O157 isolates were identified by standard biochemical methods and serologic tests by using specific O157 antiserum (INPB-ANLIS "Dr. Carlos G. Malbrán") and sent to the Servicio Fisiopatogenia as National Reference Laboratory (NRL) for further characterization.

As part of the case-control study conducted in the pediatric hospital to identify the risk factors associated with the STEC infection, parents of the 2-year-old girl were interviewed with a standardized questionnaire 8 days after onset of symptoms. Information was collected about her clinical illness, potential exposures in the 7 days before the onset of diarrhea, and demographic issues. Her parents reported that on April 25 the girl had eaten a home-prepared hamburger, made from ground beef purchased from a local market. No other family members reported diarrhea.

Three days after the interview, on May 6, a formal complaint was presented by the mother at the Division of Public Health of Lanús, in the southern area of Buenos Aires, where the family lives. The frozen leftover ground beef from the same package used to make the hamburgers was provided by the child's family and processed at the Laboratorio Central de Salud Pública.

A 65-g portion of the ground beef was incubated overnight at 42°C in 585 mL of modified *E. coli* medium broth containing novobiocin (final concentration, 20 µg/mL). The sample was positive using the *E. coli* O157 Visual Immunoassay (Tecra Internacional Pty. Ltd., French Forest NSW, Australia) (8). Immunomagnetic separation was performed with 1 mL of the culture, according to the instructions of the manufacturer (Dynal, Inc., Oslo, Norway). The concentrate sample was plated onto CT-SMAC and O157:H7 ID medium (bioMérieux, Marcy l'Etoile, France). Up to 20 sorbitol-nonfermenting colonies were selected, confirmed as *E. coli* O157, and sent to NRL.

At NRL, both clinical and ground beef O157 isolates were confirmed as *E. coli* O157:H7, susceptible to all of the antibiotics assayed, as previously described (9). Genotypic characterization showed that the isolates harbored *stx*2, *eae,* and EHEC-*hly*A genes.

To establish their clonal relatedness, the strains were assayed by subtyping methods (9). The identity of the strains was confirmed by the unique pulsed-field gel electrophoresis (PFGE) pattern with the restriction enzymes *Xba*I and *Avr*II, and the same phage type 4. In addition, both strains

were characterized as *stx*2/*stx*2vh-a by PCR-restriction fragment length polymorphism.

To our knowledge, this is the first HUS case in our country in which the source of infection was identified. No investigation was conducted to trace back the source of the ground beef. This study illustrates the importance of the surveillance of STEC infections and the usefulness of molecular subtyping techniques, such as PFGE and phage typing, to determine the relatedness of strains and assess epidemiologic associations.

The public should be made aware that hamburgers, even when prepared at home, can be a source of infection. A primary strategy for preventing infection with *E. coli* O157:H7 is reducing risk behaviors through consumer education (10).

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Q Fever in Thailand

To the Editor: *Coxiella burnetii,* a strict intracellular bacterium, is the etiologic agent of Q fever, a worldwide zoonosis. Humans are infected by inhaling contaminated aerosols from amniotic fluid or placenta or handling contaminated wool (1). The bacterium is highly infectious by the aerosol route. Two forms of the disease are typical: acute and chronic. Acute Q fever is the primary infection and in specific hosts may become chronic (1,2). The major clinical manifestations of acute Q fever are pneumonia and hepatitis. Less common clinical manifestations are aseptic meningitis and/or encephalitis, pancreatitis, lymphadenopathy that mimics lymphoma, erythrema nodosum, bone marrow necrosis, hemolytic anemia, and splenic rupture (2). The main clinical manifestation of the chronic form is culture-negative endocarditis, but infection of vascular grafts or aneurysms, hepatitis, osteomyelitis, and prolonged fever have also been described (1,2). Fluoroquinolones, co-trimoxazole, and doxycycline are active against C. *burnetii* in vitro, and ceftriaxone has been shown to have a bacteriostatic effect and could be effective in the phagolysosome of *C. burnetii*–infected cells (3). However, the treatment of choice for Q fever is doxycycline.

The incidence of this disease is largely unknown, especially in Asia. Q fever has been reported from Japan and China (1). Seroepidemiologic surveys have shown that subclinical infection is common worldwide. Large outbreaks of Q fever have also been reported in many countries in Europe (4). A case series of acute Q fever was diagnosed in a prospective study in patients with acute febrile illness who were admitted to four hospitals in northeastern Thailand: Udornthani Hospital, Udornthani Province; Maharat Nakhon Rtacha-sima Hospital, Nakornrachasema Pro-