(SARS-CoV) (1). The first case of SARS, diagnosed as communicable atypical pneumonia, occurred in Guangdong Province, China, in November 2002. Thousands of patients with SARS have been reported in over 30 countries and districts since February 2003.

SARS is clinically characterized by fever, dry cough, myalgia, dyspnea, lymphopenia, and abnormal chest radiograph results (1–3). According to the World Health Organization (WHO) (4), the criteria to define a suspected case of SARS include fever (>38°C), respiratory symptoms, and possible exposure during 10 days before the onset of symptoms; a probable case is defined as a suspected case with chest radiographic findings of pneumonia and other positive evidence.

Although most reported patients with SARS met the WHO criteria, we found two SARS case-patients who did not exhibit typical clinical features. Case 1 was in a 28-year-old physician. He had close contact with three SARS patients on February 1, 2003. After 10 days, he had mild myalgia and malaise with a fever of 37.3°C. He had no cough and no other symptoms. Leukocyte and lymphocyte counts were normal. The chest radiograph showed no abnormalities. He did not receive any treatment except rest at home. His symptoms disappeared after 2 days. He completely recovered and returned to work 4 days after onset of symptoms. After 12 weeks, his serum was positive for immunoglobulin (Ig) G against SARS-CoV in an indirect enzyme-linked immunosorbent assay (ELISA) with inactivated intact SARS-CoV as the coated antigen. (ELISA) with inactivated intact SARS-CoV as the coated antigen. After 12 weeks, his serum was positive for IgG against SARS-CoV, detected with an ELISA. Those results strongly indicate that both patients had been infected with SARS-CoV, although their signs and symptoms did not meet the criteria for the SARS case definition. Mild SARS-CoV infection may not easily be defined clinically, and such patients may potentially spread the disease if they are not isolated.

Gang Li*, Zhixin Zhao,*, Lubiao Chen,* and Yihua Zhou*
*Sun Yat Sen University, Guangzhou, Guangdong Province, China

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Address for correspondence: G. Li, Department of Infectious Diseases, Third Affiliated Hospital of Sun Yat Sen University, NO. 600, Tianhe Lu, Guangzhou, 510630, Guangdong Province, P.R. China; fax: 86-20-87536401; email: ligangzh@pub.guangzhou.gd.cn
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 transmission 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 interrupt the transmission of SARS. The group on which to focus should be the community in close contact with previous outbreak areas.

Isao Arita,* Kazunobu Kojima,† and Miyuki Nakane*

*Agency for Cooperation in International Health, Kumamoto, Japan; and †Sapporo Medical University School of Medicine, Sapporo, Japan

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Address for correspondence: Isao Arita, Chairman, Agency for Cooperation in International Health (ACIH), 4-11-1 Higashimachi, Kumamoto-city, 862-0901, Japan; fax: 81-96-367-9001; email: arita@acih.com

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