CORRESPONDENCE

An *Escherichia coli* O157:H7 Outbreak?

SIR—Friedman et al. [1] describe an outbreak of hemolytic uremic syndrome and bloody diarrhea, which they ascribe to an inadequately maintained swimming pool. They consider the causative pathogen to be enterohemorrhagic *Escherichia coli* (EHEC) O157:H7 on the basis of isolating the organism from 1 case only and some serological evidence from 17 (59%) of 29 cases. In only 2 cases was *E. coli* O157:H7 sought by use of the Sorbitol Maconkey (SMAC) agar.

We consider that for an outbreak of infection in at least 29 people, a single isolation of the pathogen is inadequate to ascribe the whole outbreak to it. An inadequately maintained swimming pool in a rural area, such as the one Freidman et al. describe, is likely to contain many *E. coli,* including a variety of serotypes of EHEC, which would not be detected by SMAC agar. The prevalence of antibodies to the O157 antigen of *E. coli* in healthy individuals in rural communities in Canada has been found to be as high as 12.5% [2]. Unless serological tests and/or cultures for other EHEC are performed, we consider it inappropriate to ascribe an outbreak to a single EHEC serotype, such as O157:H7. We have shown that if SMAC agar alone had been used to test patients infected in a recent outbreak that was due predominantly to EHEC O111:H−, the outbreak could have been erroneously ascribed to EHEC O157:H7 [3].

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3. Goldwater PN, Bettelheim KA. An outbreak of hemolytic uremic syndrome due to *Escherichia coli* O157:h7: or was it? Emerg Infect Dis **1996**; 2:153–4.

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Reply

SIR—We appreciate Drs. Bettelheim and Goldwater pointing out that *Escherichia coli* O157:H7 is not the only *E. coli* serotype that can cause bloody diarrhea and hemolytic uremic syndrome [1]. As they stated in their 1996 letter to the editor [2], investigators should look for all enterohemorrhagic *E. coli* (EHEC) in these settings. However, our investigation was conducted >3 weeks after the pool party, so we did not attempt to perform stool cultures and had to rely on serology to determine whether the illnesses were related to the single culture-proven secondary case. It is theoretically possible but highly unlikely that the single person cultured early and appropriately had an *E. coli* O157:H7 infection, whereas other pool party attendees had infections caused by other EHEC serotypes. *E. coli* O157 infections (0.85 reported infections/100,000 population/year in 1997–1998) are unusual in Georgia, despite active statewide laboratory-based surveillance.

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Clinical Infectious Diseases 2000 30:984

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Aspergillus: An Increasing Problem in Tertiary Care Hospitals?

SIR—We analyzed the microbiologic laboratory data regarding isolation of *Aspergillus* species from clinical specimens of patients hospitalized at the Detroit Medical Center from 1994 through 1998, and identified a steady increase in the number of *Aspergillus* isolates recovered (figure 1). Duplicate isolates from the same patients were few (<5%). Of the 423 isolates, 284 (66%) were *Aspergillus fumigatus.* Common non-*fumigatus* species were *Aspergillus flavus* and *Aspergillus niger.* The isolates included those that caused disease as well as colonizers and contaminants. Mere presence of *Aspergillus* species in clinical specimens does not imply invasive disease. Invasion must, however, be strongly suspected in certain settings such as neutropenia and bone marrow transplantation [1, 2]. Most source patients were immunocompromised children and adults (i.e., cancer patients, marrow trans-

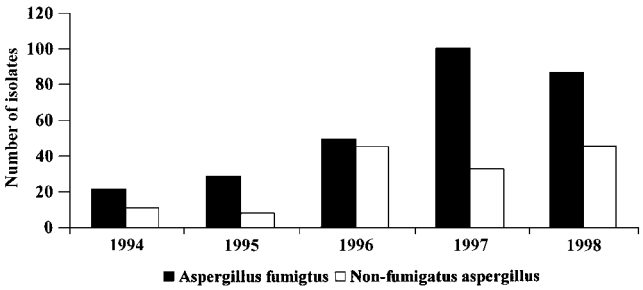

Figure 1. *Aspergillus* species isolates from patients hospitalized at the Detroit Medical Center, 1994–1998. Solid bars, *Aspergillus fumigatus;* open bars, non-*fumigatus* aspergillus.

plant recipients, AIDS patients, and those receiving immunosuppressive agents).

Our data suggest that the incidence of aspergillosis may be on the rise at our medical center. A large retrospective study of aspergillosis conducted at a transplant center has observed a similar trend [3]. There may be several reasons for this increase: an increase in the number of immunocompromised hosts in the hospital setting, a heightened awareness of aspergillosis among clinicians that causes them to perform more fungal diagnostic tests and so detect fungal infections more often in clinical specimens, better fungal isolation techniques in the laboratory, and/or an increase in the number of building construction sites around hospitals. Another contributing factor may be the recent availability of triazoles (e.g., fluconazole) for better treatment of and prophylaxis for candidal infections.

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African Trypanosomiasis

SIR—Sinha et al. [1] have provided an excellent review of African trypanosomiasis in travelers from the United States. Their review purports to represent "all or nearly all" of the US clinical experience with East African trypanosomiasis in the past 30 years. However, their series does not include a case of fulminant East African trypanosomiasis that we had reported in conjunction with the Centers for Disease Control and Prevention (CDC) in 1989 [2].

Our patient, a previously healthy 58-year-old woman, presented with fever 10 days after sustaining a tsetse fly bite while on a photographic safari in Rwanda. This case was complicated by adult respiratory distress syndrome, cardiac arrhythmias, thrombocytopenia, lactic acidosis, and hepatic and renal failure, but ultimately responded to treatment with suramin. In addition, spurious hypoglycemia, apparently related to in vitro metabolism of glucose by high concentrations of trypanosomes in initial blood specimens, was demonstrated. This phenomenon had not been reported previously in trypanosomiasis, although in our report we referenced another case acquired in 1983 in which, retrospectively, spurious hypoglycemia was likely to have been present. This case was reported in 1986 [3] and is included in the review by Sinha et al., but evidence that suggested possible spurious hypoglycemia documented in CDC internal records was not included in the published case report.

East African trypanosomiasis may be a fulminant infection that involves multiple organ systems. Such cases are readily diagnosed by peripheral blood smears and are overwhelmingly likely to be reported to the CDC. Further review of the archives of the Parasitic Diseases Drug Service of the CDC might disclose other cases not listed in this recent review.

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Screening for Latent Tuberculosis in Sexually Transmitted Disease Clinics

SIR—I read with interest the article by Nolan [1] in *Clinical Infectious Diseases*. Dr. Nolan discusses strategies for targeted screening for latent tuberculosis infection in such venues as jails, needle-exchange programs, and health clinics. In 1993, we conducted a study of tuberculin skin testing among attendees of an inner city sexually transmitted diseases (STD) clinic [2]. The prevalence of positive skin test results was 34%. Compliance with return visits for skin test interpretation was doubled by use of a simple incentive package of condoms, candy, and a fact sheet on tuberculosis. I agree with Nolan that identification and treatment of individuals with latent tuberculosis is a potentially important contributor to the prevention of tuberculosis in the United States. STD clinics, which generally serve a younger clientele who may benefit from isoniazid prophylaxis and which may have a high prevalence of infection, should be added to the list of appropriate screening sites.

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Nosocomial Bloodstream Infections: Are Positive Blood Cultures Misleading?

SIR—We read with great interest the article of Edmond et al. [1] on nosocomial bloodstream infections and were impressed by the findings and the originality of some of the analyses. However, although the trend for increase in gram-positive pathogens is already well established [2], the finding of 32% coagulase-negative staphylococci (CNS) in nosocomial bloodstream infections (BSI) far exceeds the findings of previous studies (11% from nationwide data [3] and 18% in critically ill patients [4]). Our concern is that many, if not most, of the CNS isolates included here represent contamination. All of the criteria used in this study could not assure that these isolates represent true bacteremias. The presence of intravascular catheters, the initiation of antimicrobial therapy, and the presence of fever are almost universal characteristics of patients from whom blood cultures are obtained.

The problem of distinguishing between true pathogens and contaminants in blood isolates has not been satisfactorily resolved to date [5], despite its everyday occurrence. The most common criterion used in many studies for this purpose is the exclusion of cases where potential skin contaminants grow only from a single bottle. Although there is the possibility of excluding a few true bacteremias by this method, it ensures that not too many contaminants are included. This criterion can be improved with the use of some method of bacterial fingerprinting for the comparison of 2 isolates from the same patient, but unfortunately there is no practical and inexpensive technique to be widely recommended. The best approach of which we are aware is the individual consideration of all clinical and bacteriologic data (signs, symptoms, underlying conditions, presence of devices, number of blood-culture bottles taken per clinical episode and number positive, source of specimens for cultures [e.g., obtained through lines or by peripheral venipunctures], how did symptoms resolve, etc.) by an experienced expert in infectious diseases. We admit that these are not practical criteria, since they are subjective, cumbersome, and not feasible for a multicenter study such as this one.

We hope that this letter will stimulate discussion regarding the issue raised, since it continues to be an important challenge to formulate reliable and objective criteria to distinguish between true and false bacteremias.

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Is an Isolated Initial Phase of a Tick-Borne Encephalitis a Common Event?

SIR—Tick-borne encephalitis (TBE) is one of the most important human infections of the CNS in several European countries. It has been endemic primarily in southern Scandinavia and central Europe, including a large part of Slovenia. The etiological agent of TBE is an RNA virus belonging to the *Flavivirus* genus of the family *Flaviviridae*. The hard tick *Ixodes ricinus* is the principal vector of the central European virus subtype. It is well known that in at least two-thirds of patients who develop CNS involvement, the disease has a characteristic biphasic course. After the initial phase of illness that corresponds to viremia, followed by an asymptomatic interval, the second phase presents signs and symptoms of meningitis, meningoencephalitis, or even meningoencephalomyelitis [1].

Seroepidemiological studies regarding TBE virus infection in endemic areas of various European countries demonstrated conclusively that asymptomatic infections are common [2–4]. In some of the reports it was also suggested that there is an abortive form of TBE virus infection, which is manifested only by a febrile headache without meningeal involvement (i.e., the initial phase of the illness without subsequent CNS involvement) [1, 3, 5, 6]. However, it seems that this suggestion relies more on data for eastern TBE than on data for central European (western) TBE [7], which are dubious with respect to the frequency of abortive forms of TBE. Thus, in a comprehensive review article on TBE, published recently in *Clinical Infectious Diseases* [7], the authors state that the initial phase of the illness is followed by "an afebrile and relatively asymptomatic period, lasting 2–10 days. In about one-third of patients infected, the second phase of disease develops." A logical conclusion from such a statement, as suggested by Kunz [8], would be that two-thirds of clinically symptomatic TBE virus infections manifest themselves only by the initial phase of the illness. However, later in the article, in a discussion of clinical manifestations, the authors report that in the western European literature this initial phase of TBE is usually not recognized, possibly (according to Ackerman et al. [2]) because of the small proportion of such cases.

We challenged these quite diverse statements with the findings of 2 prospective studies we have performed during the last few years. In one study we searched for the presence of thrombocytopenia in addition to a well-known leukopenia during the initial phase of TBE [9], and in the second study (still ongoing), we examined the etiology of febrile illnesses that occur within 6 weeks after a tick bite [10]. Inclusion criteria for both studies were the presence of a febrile illness after a tick bite, as well as clinical and laboratory assessment (including demonstration of IgM and IgG antibodies against TBE virus using diagnostic kits [Immunozyme, FSME, Immuno AG, Vienna]) at initial examination, 14 days, and 6 weeks. Using this approach, we were able to study patients referred to us by primary physicians who were in the initial phase of TBE and check for the eventual appearance of the second, encephalitic phase of the disease.

From 1994 through 1997, 165 patients aged >15 years were referred to the Department of Infectious Diseases at the University Medical Centre, Ljubljana, Slovenia. These patients had a febrile illness associated with a tick bite during the 6 weeks before the onset of the disease and either normal CSF examination results or complete absence of clinical signs of meningitis. During a follow-up of 6 weeks, infection with TBE virus was demonstrated in 44 (26.7%) of 165 patients by the appearance of specific IgM and IgG antibodies against TBE virus. All of these patients lived in the central part of Slovenia in a region where TBE is known to be endemic, and none of them had been vaccinated against TBE. However, during the follow-up period, all 44 of the patients developed the second, meningoencephalitic phase of the disease; that is, all had clinically biphasic course of the illness with CSF abnormalities (pleocytosis) during the second phase.

Therefore, none of our 44 patients developed only the isolated initial phase of the disease without later developing meningitis or meningoencephalitis. Our findings strongly support the idea that the abortive form of central European TBE is rare, if it exists at all. However, our findings do not negate the fact that a majority of patients are not recognized as having an infection with TBE virus during the initial phase of their disease.

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Antiviral Therapy for Neurological Manifestations of Enterovirus 71 Infection

SIR—An analysis of 97 enterovirus 71 (EV 71) culture-positive cases from Taiwan revealed CNS involvement in 35% of cases [1]. MRI showed involvement of the midbrain, pons, and medulla oblongata, which seem to be the target foci for EV 71 replication. It was demonstrated that early detection a CNS lesion by MRI and CSF examination was likely to provide useful information regarding the brain stem encephalitis [1] and thus contribute to judicious clinical management.

In all probability, the clinical course of illness in Taiwanese patients may have been modified by an early therapeutic intervention with IFN. IFN-α therapy within 24 h of admission halted progression of disease in 2 patients. A 16-month-old child with bulbar paralysis due to type 1 wild poliovirus was treated with 1 million doses of im IFN-α, and a 34-year-old man with bulbar paralysis due to type 2 vaccine poliovirus was treated with 3 million doses of im IFN-α, both for 16 days [2]. Both showed improvement within a day or two. IFN therapy, after an indication of brain lesions by MRI, could ensure prompt recovery with no residual sequelae and might lower the existing mortality associated with EV 71 infections.

In addition to the usefulness of MRI to diagnose EV 71 infection, proton magnetic resonance spectroscopy (MRS) is also of value to monitor the functional activity of neurons by scrutinizing the metabolic changes among individual neurons. In a 53-year-old woman with Kreutzfeldt-Jakob disease, neuronal loss was demonstrated by reduced *N*-acetylaspartatex, reduced creatinine-phosphocreatinine ratio and increased sorbitol levels [3].

Earlier detection of viral involvement of EV 71 target nuclei as determined by MRI and MRS, if followed by immediate IFN-α therapy, should reverse the viral damage to neurons. Furthermore, such early detection could help in deciding whether to prescribe IFN-α along with the promising antienteroviral drug pleconaril [4]. This combined therapeutic recipe would be associated with reduced mortality and negligible residual morbidity.

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Clinically Relevant *Aeromonas* Species

SIR—In the October 1999 issue of *Clinical Infectious Diseases,* an update of the current nomenclature, taxonomy, and classification of aerobic and facultative bacteria is presented [1]. While checking the species of *Aeromonas* in the updated list, we were surprised to find *Aeromonas enteropelogenes* included as a valid species, although it was synonymized a long time ago with *Aeromonas trota* on the basis of 16S rRNA sequences [2]. This synonymization has been further confirmed in recent studies with other molecular methods [3–5] and with phenotypical tests [6].

It is encouraging, however, that *Aeromonas sobria,* a misnomer classically used by clinicians to refer to *Aeromonas veronii* biovar *sobria,* was not included in the list. In fact, *Aeromonas sobria sensu stricto* is considered a valid species of environmental origin that is not commonly reported in clinical cases [7].

Another important consideration are the criteria employed for inclusion of species on the list. The genera *Aeromonas* now encompasses 14 species. In the recent overview/review of the genus carried out by Janda and Abbott [7], only 5 species, including 1 with 2 biovars, were considered pathogenic. Three of these are referred to as major pathogens *(Aeromonas hydrophila, Aeromonas caviae,* and *A. veronii* biovar *sobria),* since they account for >85% of clinical isolates of this genus [8], whereas the other 3 *(A. veronii* biovar *veronii, Aeromonas jandaei,* and *Aeromonas schubertii)* are considered minor pathogens [7]. The list published in *Clinical Infectious Diseases* includes these 5 species, as well as 5 others *(Aeromonas*

allosaccharophila, Aeromonas bestiarum, Aeromonas media, Aeromonas salmonicida, and *A. trota),* in addition to the invalid species mentioned above. If the detection of these species independently of their pathological significance in clinical samples is the basis for inclusion, it would be appropriate to include these last 5 species, but then *Aeromonas encheleia* and *Aeromonas eucrenophila,* isolated from an ankle fracture and a wound, respectively, would be missing [7]. *Aeromonas salmonicida* was classified by Janda and Abbott [7] under the group of environmental species. However, similar to the experience of other investigators, we encountered this species with a low incidence in feces of patients with diarrhea [9]. We agree, therefore, with the authors of the list published in *Clinical Infectious Diseases* [1] that these species should be included. In view of these comments, it is important that the list as published be amended in order to be properly updated.

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Klebsiella pneumoniae Necrotizing Fasciitis Associated with Diabetes and Liver Cirrhosis

SIR—We read with interest the two articles in *Clinical Infectious Diseases* on the association of necrotizing fasciitis and *Klebsiella pneumoniae* liver abscess by Hu et al. [1] and Dylewski and Dylewski [2]. Although *K. pneumoniae* is a common co-pathogen in patients with polymicrobial necrotizing fasciitis, the bacterium is rarely the sole cause of this serous soft tissue infection [3]. As was pointed out by Hu et al. [1], all 4 patients who had liver abscess and fasciitis also had diabetes. Therefore, an awareness of the spectrum of underlying diseases in association with monomicrobial klebsiella fasciitis is important. We describe a case of *K. pneumoniae* necrotizing fasciitis and spontaneous bacterial peritonitis in a patient with diabetes and cirrhosis.

A 52-year-old man with diabetes and hepatitis B–related liver cirrhosis (Pudge-Child grade C) was admitted for evaluation of fever, and abdominal pain and distension. Physical examination revealed stigmata of chronic liver disease, ascites, and mild diffuse abdominal tenderness. There was no evidence of infection elsewhere. Abdominal centesis on the day of admission was unsuccessful. Ultrasonography of the hepatobiliary system revealed no evidence of liver abscess or biliary infection. Blood culture was obtained and he was given empirical treatment with cefoperazone/sulbactam for presumed spontaneous bacterial peritonitis. Two days later, blood culture yielded *K. pneumoniae* that was susceptible to cefoperazone/sulbactam, amoxicillin/clavulanate, and ofloxacin. Screening for production of extended-spectrum β-lactamase was negative. Culture of ascitic fluid obtained on day 6 was negative, but there was an increased polymorphonuclear leukocyte (PML) count of 129×10^6/L, a very low protein level of 5 g/L, and a low pH of 7. The patient's condition improved over the next few days and the antibiotic was continued for a total of 10 days.

Four days later, the patient was readmitted for evaluation of a 2-day history of left leg pain, erythema, and swelling. His temperature was 35.6°C; pulse rate, 140; and blood pressure, 67/33 mm Hg. There were multiple patches of purpuric lesions over the right groin, left calf, and mid-thigh. Skin necrosis was present in one of the mid-thigh lesions. Emergency surgery revealed necrotizing fasciitis, and extensive debridement was performed. Parenteral antibiotics including amoxicillin/clavulanate and ofloxacin were administered. Cultures of blood and tissue specimens from the calf both yielded a pure growth of *K. pneumoniae*. The antibiogram and biotype of this isolate were identical to that of the isolate 10 days earlier. The patient's condition deteriorated further despite intensive therapy, and he died 5 days later.

Both diabetes and liver cirrhosis predisposed our patient to serious bacterial infection. Patients with diabetes have elevated rates and severity of *K. pneumoniae* infections, including pneumonia, bacteremia, hepatic abscess, and meningitis [4–6]. In-

fections are also frequent in cirrhotic patients because of their defective defense mechanisms. The most important is spontaneous ascitic fluid infection, which has a prevalence of 10%–27% among cirrhotic patients who are admitted to hospitals with ascites. This type of infection is usually caused by an enteric bacterium, such as *Escherichia coli* or *Klebsiella* species. In one-third to one-half of the cirrhotic patients with spontaneous ascitic fluid peritonitis, bacteremia was also present [7]. Diagnosis of spontaneous ascitic fluid peritonitis is made on the basis of clinical features, an increased ascitic fluid PML count ($> 250 \times 10^6$ cells/L), and/or positive ascitic fluid culture.

Spontaneous ascitic fluid peritonitis cannot be definitely confirmed in our patient because a sample of the ascitic fluid was not available before administration of antibiotics. However, spontaneous ascitic fluid peritonitis is likely because of the abdominal signs, absence of an evident intra-abdominal surgically treatable source of infection, abnormal parameters in the ascitic fluid (day 6), and absence of another source for the *K. pneumoniae* bacteremia. Our case is unique in that a rare complication occurred shortly after an apparently adequate course (10 days) of treatment. During the second hospital admission, there was no evidence of recurrent spontaneous ascitic fluid infection or liver abscess. The bilateral necrotizing fasciitis in the lower limbs, therefore, could be spontaneous or more likely a result of metastatic seeding during the earlier ascitic fluid infection–related bacteremia.

Monomicrobial *K. pneumoniae* necrotizing soft tissue infection is rare. In addition to the 4 reported cases in diabetics with liver abscess, 2 cases have been described in association with bacteremia and advanced cirrhosis [1,2,8]. Data from the small number of reported cases thus suggest that monomicrobial *K. pneumoniae* is associated with diabetes and cirrhosis. When *K. pneumoniae* bacteremia occurs in these patients, clinicians should be vigilant about metastatic soft tissue infections.

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Handwashing—The Semmelweis Lesson Misunderstood?

SIR—Larson's excellent review about hand hygiene [1] highlights the persisting reluctance in the United States to use alcohol-based hand disinfection for preventing the spread of hospital-acquired microorganisms. This letter is intended to provide some further clarifications about Semmelweis' discovery to introduce hand disinfection and not handwashing into clinical practice.

More than 150 years ago, Ignaz Semmelweis (1818–1865) demonstrated that puerperal fever was a contagious disease caused by infectious organisms, which were spread from patient to patient via the hands of health care workers (HCWs). This discovery led to the introduction of hand dips with chlorinated lime at Vienna General Hospital. Since then, many scientists have cited Semmelweis' observations, but, amazingly, grossly misleading impressions still arise about Semmelweis and his original idea of antiseptic hand disinfection, often wrongly cited as "handwashing" in the English-language literature [2–4]. In fact, Semmelweis never promoted handwashing with soap and water; he was opposed to it, since he wrote: "The cadaveric particles clinging to the hands are not entirely removed by the ordinary method of washing the hands with soap.… For that reason, the hands of the examiner must be cleansed with chlorine, not only after handling cadavers, but likewise after examining patients" [5].

Nowadays, sink-based handwashing with water and antiseptic soaps could reduce the risk of cross-transmission. However, many studies have shown that compliance of HCWs with recommended handwashing practice remains unacceptably low, in the range of 10% to 50% [6]. Noncompliance is even higher in intensive care units (ICUs), during procedures that carry a high risk for contamination, and during periods of increased workload [7]. As recently suggested by Pittet et al., full compliance with handwashing guidelines seems unrealistic. The average hand wash duration in routine patient care ranges between 8 and 20 seconds, which is too short for most handwashing agents to be fully effective [7, 8]. Moreover, HCWs need 40–80 seconds for washing hands at the sink and returning to their workplace. In ICUs, where at least 20 opportunities for handwashing occur per

hour of care, the amount of time spent with handwashing at the sink quickly becomes prohibitive [7, 9].

In contrast, waterless, alcohol-based hand rubs, readily available at the bedside in small bottles or dispensers, allow much faster hand hygiene during patient care, achieve higher and more rapid microbial killing, and may even exert a prolonged antimicrobial effect when chlorhexidine or similar substances are added [1, 8–10]. Although alcohol-based hand disinfection has been used in many countries around the world and has been shown to be superior to handwashing in many important microbiological and technical aspects [11], including decreased skin irritation when emollients [1, 10] are added, the use of alcohol-based hand rubs remains very limited in the US health care system. This is remarkable given that Semmelweis' original experience was actually related to hand disinfection and not handwashing! We conclude that there is an urgent need to reconsider the true message of Semmelweis and evaluate ways to implement and disseminate hand disinfection in the United States.

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Association of Primary *Pneumocystis carinii* Infection and Sudden Infant Death Syndrome

SIR—Histological evidence indicates that primary *Pneumocystis carinii* infection is associated with sudden infant death syndrome (SIDS) [1]. Fifty-five (31%) of 177 infants who died in the community from no apparent cause had positive lung specimens, whereas only 10 (2.9%) of 342 infants who died of multiple causes in hospitals had positive lung specimens (cases with AIDS and malignancies were excluded from the study). We recommend that the high prevalence of *P. carinii* infection in infants with SIDS warrants further investigation [1].

In future studies, it will be useful to compare the iron status of case patients with that of control subjects. As with other microbial pathogens, *P. carinii* infection is associated with iron-loaded patients [2] and is suppressed by iron chelator therapy [3]. In a study of 66 infants who died of SIDS and 28 who died of other causes, the hepatic median iron concentration was, at autopsy, 296 μg/g wet weight and 105 μg/g wet weight, respectively [4]. Moreover, increasing the iron concentration of infants by feeding them iron supplements and/or iron-fortified milk formula have been reported as a risk factor for developing SIDS [5].

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