rituximab or other immunosuppressive treatments should be prescribed cautiously; the possibility for rare complications should be recognized.

Because of massive ablation of humoral immunity, the relationship between rituximab and virus infection has been addressed, including varicella–zoster infection, parvovirus B19 infection, and CMV reactivation (*7*). In immunocompromised patients, rituximab might lead to higher risk for virus infection. This issue has been addressed with HIV/AIDS patients with high-grade B-cell lymphoma for whom rituximab is not generally recommended because B-cell ablation could result in more opportunistic infections. For LYG, increased frequency is associated with both congenital and acquired immunodeficiency, such as X-linked lymphoproliferative syndrome, Wiskott–Aldrich syndrome, and HIV/AIDS in which T-cell surveillance is deficient (*8*). Thus, for a patient with LYG whose immune system might be abnormal (*9*), the risks associated with rituximab therapy should be considered the same as the risks for HIV/AIDS patients, and the risk for viral infection or reaction to rituximab should be recognized, particularly in areas where CMV seropositivity in the population is high (*10*). In addition, especially for adult and elderly patients, the gradual increase of CMV seroprevalence with age should be recognized (*10*). Moreover, the patient reported here had previously received cytotoxic drugs as well as maintenance steroid therapy, both of which contributed to a severely compromised immune system. These factors may have led to her acute CMV pneumonitis after receipt of rituximab.

In conclusion, the potential for acute CMV reactivation should recognized during use of rituximab to treat patients with LYG. During rituximab treatment of LYG, routine monitoring for CMV reactivation and other viral infections is warranted.

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Livestock-associated *Staphylococcus aureus* in Childcare Worker

To the Editor: Carriage of *Staphylococcus aureus* sequence type (ST) 398 has primarily been reported as occurring among persons in contact with livestock, including swine and cattle (*1*,*2*). This association has given rise to the characterization of this strain as livestock associated (*3*). However, ST398 colonization or infection in persons lacking identified livestock-associated risk factors have been reported (*4*,*5*). We report ST398 colonization in a childcare worker in Iowa, USA.

As part of a surveillance study of *S. aureus* carriage in child daycare facilities, samples were collected from employees, children, and environmental surfaces. Nasal samples were taken from participating children, and nasal and pharyngeal samples were taken from participating employees. All samples were cultured, and *S. aureus* isolates were examined by pulsed-field gel electrophoresis, *spa* typing, and antimicrobial drug susceptibility testing and tested for the Panton-Valentine leukocidin gene. One participant was colonized in the

nose and throat with *t*571, a *spa* type previously reported to correspond to ST398 (*1*). The isolates were nontypeable when *Sma*I was used, also a characteristic of ST398 (*6*). They were digested with *Cfr*9I and found to be closely related to an ST398 isolate of *spa* type *t*034 of swine origin but distinct from *S. aureus* isolated from 2 other employees at the facility (Figure). Both ST398 isolates were susceptible to methicillin.

The colonized employee was a 24-year-old woman who had worked at the facility for ≈5 years. She reported a history of melanoma but was not currently taking any chemotherapy drugs and had not been hospitalized in the previous 12 months. She reported having a family member who worked in a hospital and had direct contact with patients, but the employee lived alone and responded negatively to questions about whether she or immediate family members had had contact with animals or worked in a processing plant.

ST398 may be transmitted from livestock to community members and then from person to person. It can potentially be transmitted in food; several studies have documented ST398 in raw meats (*7*,*8*), and we identified this strain in retail meat products in Iowa (T.C. Smith et al., unpub. data). Secondary transmission of ST398 from colonized persons to contacts has also been suggested, but the few publications reporting this suggest that ST398 seems to be less transmissible by this route than are common human strains (*9*).

We cannot be sure whether either of these routes played a role in acquisition of ST398 by this employee. Although no other tested persons in this childcare facility were found to carry ST398, only 24 (40%) of the 60 employees and 8 (4.8%) of the 168 children participated, suggesting the possibility of a reservoir in the facility among those who were not tested. Of the 24 employees who participated, 2 reported occupational contact with any animals, 2 reported contact with swine, and 3 reported contact with cattle. However, no participant reported having animals other than cats or dogs on their property. It is possible that ≥1 sampled employee may have been a transient ST398 carrier but negative at the time of our sampling.

Reports of ST398 in persons who had no direct contact with livestock in the United States are rare (*10*). To provide a better understanding of the epidemiology of this novel strain, further examination of the emergence of this isolate in community settings and on farms is needed.

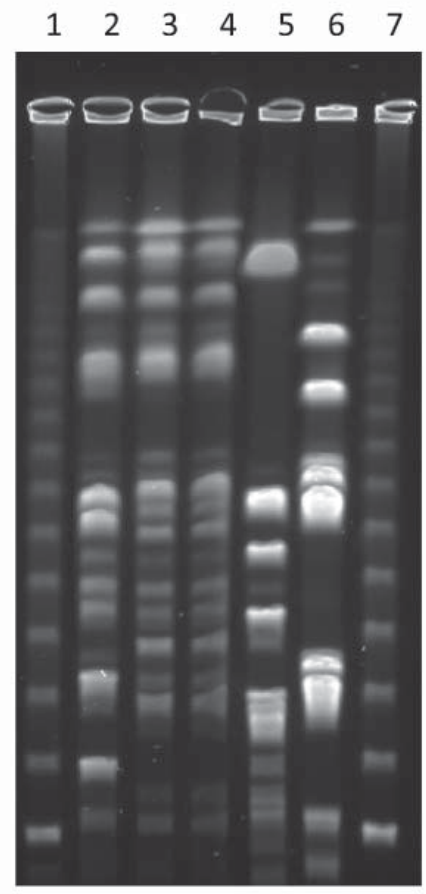

Figure. Pulsed-field gel electrophoresis of *Staphylococcus aureus*. Isolates were digested with *Cfr*9I. Lanes 1 and 7, molecular mass ladder; lane 2, *t*034 sequence type (ST) 398 isolate from pig; lane 3, *t*571 ST398 nasal isolate from colonized childcare employee; lane 4, *t*571 ST398 throat isolate from colonized childcare employee; lanes 5 and 6, non-ST398 isolates (*t*2228 and *t*084, respectively) from 2 other childcare employees.

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Sequence Analysis of Feline Coronaviruses and the Circulating Virulent/Avirulent Theory

To the Editor: Feline coronaviruses (FCoVs) occur as 2 pathotypes, feline infectious peritonitis virus (FIPV) and feline enteric coronavirus (FECV). FECV is common in cats, causing mild transient enteritis in kittens, but is asymptomatic in adult cats. In contrast, FIPV occurs sporadically but is lethal. It replicates in monocytes and macrophages and rapidly disseminates throughout the body causing systemic immunopathologic disease (*1–4*).

The relationship between FECV and FIPV has become a matter of debate. Genetic and animal experimental evidence indicates that FIPV arises by mutation from FECV in the intestinal tract of a persistently infected cat; the virus thereby acquires the monocyte or macrophage tropism that enables it to spread systemically and cause FIP (*5–7*,*8*). According to another view, the 2 pathotypes circulate independently in the field. This circulating virulent/avirulent FCoV theory recently was advocated by Brown et al. (*9*). Their conclusion was based on sequence analyses of parts of the viral genome including the matrix (M) gene, phylogenetic analysis of which revealed reciprocal monophyly of the sequences obtained from FIP cases versus those of asymptomatic FECV-infected animals. In addition, the authors suggested 5 aa residues in the M protein to represent potential diagnostic markers for distinguishing virulent FIPV from avirulent FECV (*9*).

To try to verify the findings of Brown et al. (*9*), we determined and analyzed M genes from 43 FCoV genomes, 20 of which came from cats in single-cat households, and 23 from cattery animals. The latter group consisted of 10 asymptomatic healthy cats (FECV; test specimens: feces) and 13 dead cats with FIP confirmed through pathology (FIPV; test specimens: organs, ascites). These animals came from 8 catteries. FECV and FIPV cases were found in 7 (designated A to G); the remaining cattery (H) provided 2 cats with FIP. The genomes from individually living cats were from 15 FIPV- and 5 FECV-infected animals.

Using specific primers (sense 5′-CGTCTCAATCAAGGCATATAATC CCGACGAAG-3′, antisense 5′-CAG TTGACGCGTTGTCCCTGTG-3′), we amplified the same 575-bp M gene fragment as studied by Brown et al. (*9*). GenBank accession numbers for the FCoV M gene sequences determined in this study are HQ738691–HQ738733. When compared by phylogenetic analysis, the nucleotide sequences of FIPV and FECV M genes distributed into paraphyletic patterns rather than in monophyletic clusters (Figure, panel A).

Thus, as we observed earlier for the 3c gene (*10*), M gene sequences generally clustered according to the cattery from where they originated, irrespective of their pathotype (e.g., FECV 586 and FIPVs 584 and 585 from cattery A; FECV 620 and FIPVs 615 and 622 from cattery G; FECV 10 and FIPV 8 from cattery F). Such a distribution pattern is consistent with the mutation theory, according to which FIPVs originate from FECVs and are thus closely related (*7*,*9*). Exceptions in this picture were FIPV 9 in cattery F and FECVs 406 and