

readmitted 18 days later with nasal congestion, cough, and high fever. PCR results were again positive for pandemic (H1N1) 2009, and the patient was successfully treated with oseltamivir.

Patient 2 and probably patient 3 acquired their infections while hospitalized, suggesting potential nosocomial transmission. No other respiratory viruses were found in any of these patients. The viral isolates were all tested (LightMix for detection of influenza A virus oseltamivir resistance [H274Y]; TIB MOLBIOL) for possible resistance to oseltamivir, but none had the resistance-implicated H274Y mutation in the neuraminidase gene.

Shedding of seasonal influenza A virus ceases within 5–7 days during natural infection and during infections treated with neuraminidase inhibitors (4). Although clearing of virus after the first infection was not documented in the 3 patients described here, it is unlikely that virus persisted between the 2 episodes of influenza since each of the patients fully recovered after specific antiviral drug treatment. However, we cannot rule out that patient 2 may have never cleared the virus due to her immune suppression.

As described by mathematical modeling (5), the 3 patients described were susceptible to reinfection with pandemic (H1N1) 2009 due to the high rate of community infection and to their incomplete immunologic protection within the period of reexposure. During the current pandemic of influenza subtype H1N1, healthcare workers and patients should be aware that symptomatic reinfection might occur after a first episode of infection.

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Skin Lesion Caused by ST398 and ST1 MRSA, Spain¹

To the Editor: Human infections caused by methicillin-resistant *Staphylococcus aureus* (MRSA) sequence type 398 (ST398) have been emerging in recent years in Europe (1,2). Pigs represent a common reservoir of MRSA ST398, and working with these animals may constitute a risk factor for MRSA carriage and possible infection (2–4). We report a case of human infection caused by MRSA ST398 in Spain.

A 12-year-old girl living close to a pig farm, where her father and mother worked, sought treatment for a skin lesion on her chin. Two types of MRSA were recovered from the lesion, and it resolved after topical

treatment with mupirocin over 10 days. MRSA isolates recovered were characterized by multilocus sequence typing (MLST) and by staphylococcal cassette chromosome (SCC) *mec*, *spa*, and *agr* typing as described (3). The presence of genes encoding Pantón-Valentine leukocidin (PVL) (*lukF/lukS*), toxic shock syndrome toxin-1 (*tsst1*), and exfoliative-toxin A and B (*eta*, *etb*) was investigated by PCR (2,3). Antimicrobial susceptibility tests were carried out by using the VITEK-2 system (bioMérieux, Marcy l’Étoile, France), and *mecA*, *ermA*, *ermB*, *ermC*, *mrsA*, *tetK*, *tetL*, *tetM*, *ant(4’)-Ia*, *aph(3’)-III*, and *aph(2’)-aac(6’)* resistance genes were studied by PCR (5). *dfiK* gene detection was performed by using primers designed from the sequence FM207105 included in GenBank (*dfiK*-F 5'-GAGAATCCCAGAGGATTGGG; *dfiK*-R, 5'-CAAGAAGCTTTTCGCTCATAAA), and the obtained amplicons were sequenced for confirmation. Mutations in quinolone targets were determined by sequence analysis of *grrA* and *gyrA* genes (6). In addition, MRSA isolates were typed by pulsed-field gel electrophoresis (PFGE) (www.harmony-microbe.net/microtyping.htm).

One of the clinical MRSA isolates recovered from the lesion and typed as ST398-*spa*-t011 showed resistance to tetracycline, macrolides, and lincosamides and harbored 5 antimicrobial resistance genes. The second MRSA strain was typed as ST1-*spa*-t127 and showed a multiresistance phenotype with 11 resistance genes, as well as the Ser80Phe and Ser84Leu amino acid changes in GrlA and GyrA proteins, respectively.

To establish the MRSA nasal colonization status of the patient and of her relatives (father, mother, and brother, all of whom had contact with

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animals) and to elucidate the possible origin of these strains, we analyzed nasal swabs for MRSA recovery. Nasal samples were plated in oxacillin-resistant *S. aureus* agar media (ORSA, Termofisher, UK), colistin nalidixic acid agar media (bioMérieux), and blood agar media (Oxoid); colonies suggestive of *S. aureus* were initially selected, further identified, and characterized for bacterial typing and antimicrobial resistance mechanisms. Samples from all relatives and the patient showed that all 4 persons were MRSA nasal carriers presenting the following genetic lineages: patient girl, ST398-t108; mother, ST398-t108 and ST1-t127; father, ST398-t108; and brother, ST398-t011 (2 variants) (Table). MRSA t127 and t011 variants detected in the skin lesion of the patient were not found in her nasal sample but were found in the nasal samples of her mother and brother. Another study has been initiated to analyze the presence of MRSA in nasal swabs of swine on the farm.

The ST1-t127 strains obtained from the skin lesion of the girl (C1569)

and from the mother's nasal sample (C1578) showed the same multiresistance phenotype and genotype (Table) and an indistinguishable PFGE pattern. This clonal type seems to be associated with community-acquired MRSA isolates circulating in Europe (7). The animal origin of this MRSA type and its possible transmission from horses and cows to humans has been suggested in previous reports (8,9).

In addition, all family members were colonized by MRSA ST398. All recovered nasal MRSA ST398 strains showed resistance to β -lactams, macrolides, lincosamide, and tetracycline, and 3 strains also showed diminished susceptibility to quinolones. Strain ST398 C1576, recovered from the brother, showed a multiresistance phenotype (Table).

The ST398 strains of our study were classified in the *spa*-types t011 and t108, 2 of the most frequently described types of the clonal complex 398 (I,4) and were untypeable by PFGE. Most MRSA ST398 strains in our study showed a resistance phenotype that included tetracycline, mac-

rolides, and lincosamides. Tetracycline resistance is a common trait of ST398 and suggests that its use in veterinary medicine may have been implicated in selection of this resistance. The first reports, describing ST398 strains, indicated β -lactams and tetracycline as the unique resistance markers, which were susceptible to other antimicrobial agents. Nevertheless, reports of ST398 showing resistance to other antimicrobial drugs are increasing. One of the ST398 strains from the brother showed an uncommon multiresistance phenotype. Moreover, all the MRSA strains of this study were negative for all tested toxin genes, included PVL, although some ST398 PVL-positive strains have been occasionally described (10).

We report a skin lesion on the daughter of a pig farmer in Spain associated with ST398-t011 and ST1-t127 MRSA strains. The results obtained suggest the specific animal origin of these strains and subsequent transference among family members. Of special interest is the multiresistance phenotype of the clinical and nasal

Table. Characteristics of the 9 MRSA strains recovered in Spain from a patient's lesion and from nasal samples obtained from patient's family members*

Strain	Origin of sample	SCC <i>mec</i> type	MLST	<i>spa</i> type	<i>agr</i>	Antimicrobial resistance phenotype	Resistance genes detected	Amino acid change in:	
								GrlA	GyrA
C1570	Patient/ skin lesion	V	ST398	t011	I	OXA, FOX, TET, ERY, CLI, TEL	<i>mecA, tetK, ermA, ermC, msrA</i>	NP	NP
C1569	Patient/ skin lesion	II	ST1	t127	III	OXA, FOX, TET, ERY, CLI, TEL, GEN, TOB, KAN, CIP, LEV, SXT	<i>mecA, tetL, tetK, ermA, ermB, ermC, msrA, aph(2')-acc(6'), ant(4')-Ia, aph(3'), dfrK</i>	S80F	S84L
C1571	Patient/ nasal swab	V	ST398	t108	I	OXA, FOX, TET, ERY, CLI, TEL, CIP†, LEV†	<i>mecA, tetK, tetL, tetM, ermA, ermC, msrA</i>	Wild	Wild
C1577	Mother/ nasal swab	V	ST398	t108	I	OXA, FOX, TET, ERY, CLI, TEL, CIP†, LEV†	<i>mecA, tetK, tetM, ermA, ermC, msrA</i>	Wild	Wild
C1578	Mother/ nasal swab	II	ST1	t127	III	OXA, FOX, TET, ERY, CLI, TEL, GEN, TOB, KAN, CIP, LEV, SXT	<i>mecA, tetL, tetK, ermA, ermB, ermC, msrA, aph(2')-acc(6'), ant(4')-Ia, aph(3'), dfrK</i>	S80F	S84L
C1574	Brother/ nasal swab	V	ST398	t011	I	OXA, FOX, TET, ERY, CLI, TEL	<i>mecA, tetK, ermA, ermC, msrA</i>	NP	NP
C1576	Brother/ nasal swab	V	ST398	t011	I	OXA, FOX, TET, ERY, CLI, TEL, GEN, TOB, KAN, SXT	<i>mecA, tetL, tetK, ermA, ermB, ermC, msrA, aph(2')-acc(6'), ant(4')-Ia, aph(3'), dfrK</i>	NP	NP
C1572	Father/ nasal swab	V	ST398	t108	I	OXA, FOX, TET, ERY, CLI, TEL, CIP†, LEV†	<i>mecA, tetK, tetL, tetM, ermA, ermC, msrA</i>	Wild	Wild

*MRSA, methicillin resistant *Staphylococcus aureus*; SCC, staphylococcal cassette chromosome; MLST, multilocus sequence typing; ST, sequence type; OXA, oxacillin; FOX, cefoxitin; TET, tetracycline; ERY, erythromycin; CLI, clindamycin; TEL, telithromycin; NP, not performed; GEN, gentamicin; TOB, tobramycin; KAN, kanamycin; CIP, ciprofloxacin; LEV, levofloxacin; SXT, sulfamethoxazole-trimethoprim.

†Intermediate category for the indicated antimicrobial drug.

ST1-t127 MRSA clinical strains and of 1 nasal strain belonging to ST398 lineage. Nasal colonization by different ST398 genetic lineages and by other lineages of MRSA as ST1-t127 seems to be frequent in persons living in close proximity to farm animals. Dissemination of MRSA ST398 (and probably also MRSA ST1) in humans who have contact with farm animals, is an emerging problem in Spain.

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Identification of a Rotavirus G12 Strain, Indonesia

To the Editor: Group A rotaviruses are the most common etiologic agents of acute gastroenteritis in infants and young children, each year resulting in ≈ 100 million diarrhea episodes and 600,000 deaths worldwide (1). The genome of rotavirus comprises 11 segments of double-stranded RNA, which encode 6 structural viral proteins (VPs) and 6 nonstructural proteins (NSPs). Recent scientific reports have identified novel rotavirus strains, such as G12 (2–5), which were first described in 1987 among Filipino children with diarrhea (6). In Indonesia, a rotavirus study showed that a broad variety of VP7 types (G1, G2, G3, G4, G8, G9) and VP4 types (P[4], P[6], P[8], P[9], P[10], P[11]), especially G9 and P[8] and G9P[8], were the genotype combinations most frequently encountered (7).

From 2005 through 2008, we conducted a nationwide surveillance study among children who had diarrhea to determine etiologies among Indonesian children seeking health services for diarrhea at hospitals and health clinics. Patients were enrolled after obtaining consent from parents/guardians of those eligible in accordance with an institutional review board protocol approved by the US Naval Medical Research Unit No. 2 (NAMRU-2) and the Ethical Committee of the Indonesian National Health Research and Development Institute. Stool specimens and clinical enrollment data were collected for each eligible patient, and all collected items were transported to NAMRU-2 in Jakarta, Indonesia. In December 2007, a stool specimen was collected from a 14-day-old afebrile infant brought to Sumber Waras Hospital in West Jakarta with diarrhea, vomiting, moderate dehydration, and malnutrition. This patient was infected with the rotavi-