The Environment as a Factor in Methicillin-Resistant *Staphylococcus aureus* Transmission

Tara C. Smith,[1,2] Erin D. Moritz,[1,2,5] Kerry R. Leedom Larson,[3,5] and Dwight D. Ferguson[1,3,4]

[1]*Center for Emerging Infectious Diseases,* [2]*Department of Epidemiology,* [3]*Department of Occupational & Environmental Health,* [4]*Great Plains Center for Agriculture Health, and* [5]*Heartland Center for Occupational Health and Safety; University of Iowa College of Public Health, Iowa City, IA 52242, U.S.A.*

Abstract: In recent years, methicillin-resistant *Staphylococcus aureus* (MRSA) has become a leading cause of infectious disease morbidity and mortality in the United States. The epidemiology of the organism has changed, with novel strains emerging in the community among individuals lacking any healthcare contact. Although direct human-to-human transmission via skin contact is one way for this organism to spread, transmission via environmental contamination of fomites or through air are other potential ways that the organism can be acquired. As such, an improved understanding of MRSA transmission is needed to implement maximally effective control and prevention interventions. We review the research documenting the role of the environment in MRSA spread.

Key words: MRSA, agriculture, zoonosis, antibiotic resistance, fomites

Correspondence: Tara C. Smith, Center for Emerging Infectious Diseases, 2501 Crosspark Rd., MTF B-168, Coralville, IA 52241-3471, USA; Phone: 319-384-5755.S; E-mail: tara-smith@uiowa.edu

---

INTRODUCTION

Staphylococcus aureus (*S. aureus*) is a ubiquitous bacterium that colonizes approximately one third of the human population /1/. The most common site for colonization is the anterior nares, but the throat, skin, and gastrointestinal tract can also be colonized /2,3/. Although the majority of individuals who are colonized will remain asymptomatic, colonization is a risk factor for the development of symptomatic infection /4,5/. Of increasing concern is the spread of strains of *S. aureus* that are resistant to the antibiotic methicillin (methicillin-resistant *S. aureus* or MRSA). Initially these strains were a problem limited to hospitals (hospital-associated MRSA, HA-MRSA), but in recent years novel strains of MRSA that are distinct from the most common hospital strains have spread in the community /6,7/. Such strains are referred to as community-associated MRSA (CA-MRSA) /8,9/. A third group of MRSA strains has been recognized more recently in association with livestock such as pigs and cattle (LA-MRSA) /10/.

Transmission of this organism can occur in a number of different ways. Direct contact between infected and/or colonized individuals can facilitate the spread of this organism from one person to another, and the bacterium can also be spread indirectly via contaminated fomites or even via air. The existence of 'supershedders' of this organism has been identified. Individuals with respiratory infections can temporarily increase the transmission rate /11/, spreading the organism via direct contact or contaminated fomites. Thus, to be successful, any control or eradication measure implemented must take into account the varied transmission mechanisms present in the particular environment. Here we review the research examining MRSA in the environment and the impact this route may have on human MRSA colonization and infection.

MSRA IN THE HOSPITAL ENVIRONMENT

To date, the majority of studies investigating environmental contamination with MRSA have been carried out in healthcare facilities, in which the presence of MRSA in rooms where MRSA-positive patients are housed has been well documented. The prevalence of contamination in such rooms varies but is often higher than the prevalence in non-clinical areas or in areas where MRSA patients are not housed. In 2006, Sexton et al. /12/ reported that 53.6% of surface samples collected from the rooms of MRSA patients were positive for this bacterium. In a study conducted in an intensive care unit (ICU) over a two-year period during which an MRSA-positive patient was present 95.8% of the time, MRSA was present in 21.8% of the environmental samples collected from bed spaces, workstations, and monitors /13/. At the beginning of an MRSA outbreak at a general surgical ward, 32% of environmental samples yielded MRSA /14/. During an outbreak period, Rutala et al. /15/ found that the total bacterial growth isolated from air, elevated surfaces, and floor surfaces, respectively, comprised 16%, 31%, and 40% MRSA /15/. Finally, 58.8% of surfaces in rooms of patients testing positive for gastrointestinal MRSA were contaminated /16/.

The bedding areas of MRSA-positive patients appear to be 'hot spots' for MRSA environmental contamination. Cloth bedding accessories have often been reported to have high rates of MRSA contamination, with rates ranging from 14.6% to over 50% found on pillows, mattresses, and bed linen /12,17-19/. Solid surfaces associated with beds (such as over-bed tables, bed frames, bedside rails, ledges behind beds, and floors next to a patient's bed) have also been found to have high rates of contamination /13,16,18-20/. A partial list of environmental components that have shown to be positive for MRSA in MRSA-positive patient rooms includes floors, faucet handles, patient gowns, blood pressure cuffs, door handles, radiators, medical equipment, television remote controls, and toilet seats /14,16, 17,19,21,22/.

Even rooms and areas that house non-MRSA patients can be contaminated; yet, the rates in such areas are generally lower than those in the rooms of MRSA-positive patients. In a study conducted at a London teaching hospital, communal bathroom areas and bathrooms for non-MRSA patients had high levels of contamination (67% and 50%, respectively), although the number of sites sampled was fairly small /20/. Oie et al. /22/ report that 7.4% of door handles of rooms with non-MRSA patients harbored MRSA, a rate that was not significantly different from that found on handles of rooms housing MRSA-positive patients (19%). The lack of statistical significance, however, could again be due to the limited sample size.

The microorganism has also been found, albeit at much lower levels, in 'non-clinical' areas of health-care facilities where patients rarely spend time. Lu et al. /23/ reported a 1.1% contamination rate on ward computers. Brown et al. /24/ sampled non-clinical areas with high hand contact (door handles, push plates, stair rails, elevator buttons, and equipment carts) but did not isolate any MRSA. In a study of an urban emergency department, only one surface (an ambulance bay security door pad) was found to harbor MRSA /25/. These contamination rates are much lower than those in patient rooms. In 1983, Rutala et al. /15/ reported that contamination levels in work areas adjacent to rooms occupied by MRSA-infected burn patients were lower than the levels in actual patient rooms.

In the healthcare setting, transmission has been shown to occur directly between patients and the environment, particularly in outbreak situations. Numerous studies have demonstrated transmission between the inanimate environment and patients by comparing molecular signatures /12,14,16,19,21, 26-28/. Two such studies found indistinguishable strains of MRSA in patients and on environmental surfaces, but not in healthcare workers, suggesting a direct transmission between patients and fomites /14,27/.

Other patient characteristics have also been found to predict environmental contamination, although the direction of transmission has not often been clarified. Although some evidence suggests that the number of colonized patients on a ward is not correlated with the number of environmentally contaminated sites, the location of colonization/ infection on the patient has been shown to be related to contamination rates /13/. In 1997, Boyce et al. /19/ reported that positive wound or urine cultures in a patient were more predictive of environmental contamination than positive cultures isolated from sputum, the nares, blood, and other sites (OR = 10.1, CI 1.6-69) /19/. The same lead author reported in 2007 /16/ that surface MRSA was more often recovered in the rooms of patients with heavily colonized gastrointestinal tracts (58.8%, CI 47.8-68.9) than in rooms of patients with MRSA isolated from sputum, wounds, blood, and the nares (23.3%, CI 14.3-35.5). Another study in 2007 /18/ found that MRSA environmental contamination was most often predicted by the presence of MRSA on the palms of patients' hands (as opposed to sputum, open and closed pus, nasal discharge, pharyngeal mucosa, urine, bronchial aspirates, IVH catheters, and bile).

The hands of healthcare workers have long been recognized as potential vehicles for MRSA transmission in the healthcare setting. Hands can become contaminated by direct contact with MRSA in patients or via environmental reservoirs. One report showed that during the routine care of MRSA-positive patients, 65% of nurses had contaminated their apparel with MRSA /19/. The same study revealed that even after activities that required no direct patient contact, 42% of nurses' gloves tested positive for MRSA. In investigators following hand contact with surfaces near patients, Bhalla et al. /29/ reported a hand contamination rate of 30% with *S. aureus* (of which 35% were MRSA), providing evidence that environment-to-hand transmission does occur. The same type of transmission was documented (although at a lower rate of 10%), even after patient discharge and terminal cleaning had occurred /29/.

Gloves are not the only apparel that can become contaminated with MRSA. High levels of MRSA contamination have been demonstrated on the uniforms of long-term care facility personnel. If no protection was worn by the worker, up to 80% of the 'waist zones' and 60% of 'pocket zones' of uniforms became contaminated, even in those who cared for patients who had not been identified as having MRSA. The same study showed that using plastic aprons and controlling the contents of pockets decreased the rates of contamination /30/.

MRSA can also be airborne, although the significance of airborne transmission of MRSA within the healthcare environment is controversial. Air-circulation systems (for example, exhaust ducting in an adjacent isolation room and ventilation grills) have been implicated in outbreaks of MRSA /31-33/. During an outbreak, Rutala et al. /15/ found that the total bacterial growth isolated from air comprised 16% MRSA. Airborne MRSA has also been demonstrated in the rooms of patients carrying MRSA. In 2006, Sexton et al. /12/ reported that MRSA grew on 28% of air samples and 40.6% of settle plates collected from the rooms of MRSA-colonized patients. Shiomori et al. /34/ reported the expulsion of airborne MRSA in rooms containing colonized patients during bed-making. The same group reported that the circulation of MRSA among patients, air, and environmental surfaces was particularly prevalent when movement occurred in the room /35/. Other data on the acquisition of MRSA from the air remain scarce.

Whereas MRSA has been uncovered in rooms of MRSA-positive patients, non-MRSA patients, and non-clinical areas, the former appears to be the most important contributor to the environmental load of MRSA, with bedding areas being frequently contaminated. Environmental surfaces play a role in the MRSA ecologic behavior in both patients and healthcare workers, and patient characteristics have been shown to predict MRSA contamination. Evidence suggests that airborne MRSA could be an important player in transmission in the healthcare setting, but the extent of that contribution remains to be seen.

MRSA OUTSIDE THE HOSPITAL ENVIRONMENT

In addition to hospitals, MRSA has increasingly been found in individuals with no recent history of hospitalization or other healthcare contracts. Recent surveys have found that approximately 1.5% of the United States (U.S.) population is colonized with MRSA /36/. Several studies have been carried out specifically to examine the prevalence of MRSA carriage in individuals lacking healthcare contact. A 2005 study carried out in New Orleans found that 1.2% of the population sampled were positive for MRSA; all positive individuals were enrolled at a student recreation center where bacterial acquisition could have occurred /37/. A similar cross-sectional study in Malaysia found that of 346 individuals, only one was an MRSA carrier /38/. Additional studies in the U.S. and internationally have found MRSA rates in the population ranging from 0.12% to 5.3% /39-43/. Therefore, MRSA prevalence still remains low outside the hospital environment, which in turn reduces the potential for contamination of the general environment by individuals colonized with this organism. How particular strains of MRSA spread within populations and the importance of direct human-to-human transmission versus environmental acquisition remains to be determined.

In the hospital setting, gastrointestinal (GI) colonization with MRSA has been shown to be associated with greater contamination of the environment /16/; yet whether GI colonization may also increase the spread of MRSA in the community at large or the frequency of MRSA GI colonization in the community in general is unknown because most population-based MRSA studies have focused on nasal carriage.

MRSA IN THE HOME ENVIRONMENT

Regarding the role of the home environment in MRSA transmission, sharing within families has been examined in a number of publications. A study surveying home surfaces in 35 households was carried out, sampling 32 home surfaces including kitchen, bathroom, office, infant, and pet surfaces /44/. The most common sites for MRSA isolation were dish towels, faucet handles, and infant high chair trays. In one home, MRSA was isolated from five different sites: kitchen sponge, garbage bin, dishtowel, bathtub, and infant high chair. Three of four individuals living in this house reported experiencing diarrhea and vomiting in the week before sampling, and six months before, one occupant had been prescribed amoxicillin for an ear infection. Interestingly, the presence of a cat in the house significantly correlated with the finding of MRSA, suggesting the possibility of pets as a secondary reservoir for MRSA after acquiring the bug from a colonized or infected human /44/. Other studies have also suggested a potential role for pets in the maintenance or transmission of MRSA within households /45-47/.

Towels have also been suggested to play a role in MRSA transmission within the household. Experimental inoculation with *S. aureus* has shown that the bacterium can survive on towels and be transferred to individuals for at least 48 hours /48/. Towels have been implicated as potential environmental vectors in outbreaks of MRSA infection within households /49,50/, as well as in athletic facilities (see below).

The home environment of healthcare workers has been found to be contaminated and can serve as a reservoir of transmission to other family members /51,52/. The microorganisms can survive for a period of several weeks on such cleaning items as dry mops /53/, which can serve to spread the bacteria throughout the home or hospital environment.

MRSA IN THE PUBLIC ENVIRONMENT

Several studies have examined the prevalence of MRSA on fomites in the community. A 2008 study in Serbia examined a large urban public transport system, collecting samples from hand rails in 55 vehicles /54/. Although all were negative for MRSA, 30% were positive for methicillin-resistant coagulase-negative staphylococci (MR CoNS), suggesting that resistance genes can also be transferred via environmental contamination /54/.

MRSA outbreaks have also been traced to public operations. An outbreak of CA-MRSA in the Netherlands was related to a beauty salon, where a beautician was found to have recurrent infections. Although instruments and wax were suspected to play a role in the outbreak, all environmental samples tested were found negative /55/.

Recently, MRSA and MRCoNS were recovered from water and sand at public beaches in the West Coast of the U.S. /56/. MRSA has also been isolated from municipal wastewater /57/, representing another potential source of human environmental contamination. Prior studies demonstrated that among residents living in proximity to areas fertilized with treated wastewater, the prevalence of *S. aureus* infections was ~25 times higher than other infections among hospitalized patients /58/. MRSA can survive for extended periods in sea water and river water, although in properly chlorinated pool water, the bacteria are killed within 24 hours /59/.

Staphylococcus aureus has been examined on environmental fomites such as coins. A Canadian study isolated *S. aureus* from circulating coins, but no MRSA were identified /60/. Nevertheless, a followup experimental study showed that MRSA could survive on coins for up to 4 hours, and if the coins were inoculated with biological materials such as pus or blood, then MRSA was able to survive on coins for up to 13 days post-inoculation /61/.

MRSA has also been identified in low levels at childcare facilities. In 2009, Hewlett et al. /62/ collected swabs from environmental surfaces at a childcare facility serving a medical university. MRSA was isolated from 2.0% (4/195) of surface swabs, including samples from two cribs, a cloth toy, and a nap mat.

MRSA IN THE SCHOOL ENVIRONMENT

In the school environment, MRSA has also become a concern, although in the U.S., data on the magnitude and severity of related skin and soft tissue infections (SSTI)—a common manifestation of community-associated MRSA strains—are generally lacking. In India, the prevalence of MRSA colonization in students aged 5-15 years has been estimated at 3% /63/. Over 13% of kindergarten students in Taiwan have also been shown to be colonized /64/. The higher MRSA colonization rates in children may be due to poor hygiene and the sharing of contaminated environments or fomites. Yet, a study of U.S. college students showed that older school groups also have an increased prevalence of MRSA colonization as college students at one State university in Texas had a rate of 7.4% /65/. In comparison, only about 1.5% of the general U.S. population is thought to be colonized with MRSA /36/.

Prevalence studies of nasal colonization are useful for surveillance purposes; yet o utside the hospital environment, an association between colonization and skin infection remains unclear. A recent study of households in New York found no association between CA-MRSA colonization and serious skin infection /66/. In children specifically, a Missouri study showed a weak relationship between SSTI and MRSA nasal colonization /67/. Future studies are necessary to determine the true association between MRSA colonization and risk of SSTI and other types of MRSA infections, not only in children but also in the community at large.

In the school setting, MRSA is transmitted by the same means as in other community venues. Direct skin-to-skin contact with an infected wound is a common route of exposure. The role of the environment in MRSA transmission has been less clear, although two recent studies have shown that surfaces in schools related to athletics (training facilities, locker rooms, wrestling mats, etc.) are commonly contaminated with MRSA /68,69/. The best way to prevent students from contracting an MRSA infection is to follow the general guidelines from the Centers for Disease Control and Prevention (CDC): practice good hand hygiene; cover cuts, wounds, and abrasions; avoid sharing personal items that are in contact with bare skin, such as razors or towels; place a barrier between yourself and shared equipment, such as a towel; and maintain a clean environment by establishing a cleaning protocol (using an Environmental Protection Agency-approved disinfectant for MRSA) for surfaces that are in direct contact with skin /70/. Specific information about the environmental management of MRSA in the community is available at http://www.cdc.gov/ncidod/dhqp/ar_mrsa_Enviro_Manage.html.

In addition to these general guidelines for students, school professionals can help prevent MRSA infections in schools /71/. Teachers should refer children with open wounds to the school nurse for evaluation and enforce hand hygiene in the classroom /72/. Healthcare professionals (school nurses) should use standard precautions when caring for potential infections and refer students to their physicians for further treatment if necessary. Parents should be notified if potential SSTI are identified.

By far, most MRSA SSTI related to schools are those involving athletic activities. Since the first known MRSA sports outbreak in 1993, actual MRSA infections, as opposed to colonization, have been increasingly reported /73/. Recently, a surveillance of Nebraska high schools showed that over 14% of schools reported an MRSA infection in one or more athletes over a 2-year period /74/. Contact sports are the most able to facilitate MRSA transmission due to the increased likelyhood of skin abrasions and direct contact. Most sports-related MRSA SSTI have been reported in football players; high school, and collegiate players, but even professional players have been affected /75-78/.

Non-contact sports have not been immune from CA-MRSA. Sports like fencing and weight lifting have experienced MRSA outbreaks as well /79/. In these cases, epidemiologic studies have shown that contaminated fomites, such as a fencing wire shared beneath the clothing or weights, are the likely cause of infection /79/. Similar to school populations at large, members of sports teams can also be colonized with MRSA at higher rates than the general population /80/.

Many athletic organizations have developed recommendations for the prevention of MRSA on the field or in the locker room. These measures are usually based on the CDC general guidelines described above and may include recommendations targeted for athletes, such as those published by the National Athletic Trainers' Association (http://www.nata.org/statements/official/MRSA_Statement.pdf). Many measures include interventions with the goal of minimizing environmental contamination and transmission, including: immediate showering after athletic activity, no whirlpool use when open wounds are present, no sharing of athletic gear, towels, or razors, and proper washing of all athletic gear and equipment after use.

When MRSA infections are identified in a school, confusion may ensue regarding the proper course of action. Parental notification policies for a single case of MRSA can vary between local and state health departments. In the past, schools have been closed because of MRSA outbreaks—for example, 22 Virginia schools were closed in 2007 after a student died from invasive MRSA /81/. School closures due to a single diagnosed case of MRSA have also been reported /82/. Neither the CDC nor the American Academy of Pediatrics recommends that the entire school be informed about a single MRSA infection, or that the school should be closed for disinfection (http://www.cdc.gov/Features/MRSainSchools, http://www.aap.org/new/mrsa.htm). For the schools, however, checking with their local public health department for additional guidance is advisable. The U.S. Department of Education recommends that schools include MRSA prevention in their emergency response plan (www.ed.gov/admins/lead/safety/emergencyplan/mrsa.doc). Generally, children with MRSA SSTI should not be excluded from school unless directed to do so by their physicians; for cases in which infected lesions are actively draining and cannot be adequately covered by a clean, dry bandage, exclusion from school activities should be considered /83/. Overall, MRSA SSTI in schools has been highly publicized in recent years. School officials must balance parental and public opinion with scientific evidence. Although MRSA infections seem to be increasing, increased surveillance and improved diagnostics may have contributed to this trend.

MRSA AND AGRICULTURE

Over the past decade, evidence showing a relationship of MRSA carriage among livestock and animal workers has shifted the focus of MRSA as a hospital-acquired to a potential zoonotic pathogen /84/. A report from the Netherlands showed that pig and cattle farmers had increased odds of being carriers of NT-MRSA ('non-typeable' MRSA, typically strain ST398) compared with other groups in the study, with odds ratios of 12.2 and 19.7, respectively /85/. This study also identified MRSA of animal origin as causing more than 20% of the cases of MRSA in the population. Of special note, the MRSA occurrences in the study population clustered predominantly around pig farming. In another study, farms were considered MRSA positive when a high rate of MRSA positive samples was found in pigs and when dust also had a high MRSA carriage rate among humans /86/. Dust samples collected from pig houses had a high prevalence of ST398, which could indicate possible transmission through the environment /86/.

MRSA ST398 has also been detected in healthcare workers in the Netherlands who have been in contact with livestock /87/. This finding has led to screening healthcare workers in the Netherlands to determine if they are carriers of MRSA to prevent transmission to patients, either directly via skin contact or indirectly via environmental contamination /87/. As studies have shown that antibiotic-resistant *S. aureus* can persist in poultry waste for up to 120 days /88/ and may be spread by flies from contaminated farms or fields /89/, further studies investigating the presence of MRSA from livestock, poultry, and the farming environment must be conducted.

Horses are also being evaluated as a reservoir for zoonotic MRSA /90/. A Canadian group studying the colonization of MRSA among horse personnel and horses identified 79 horses colonized or infected with MRSA during a two year period. Of the 194 persons evaluated in the study, MRSA was identified in 14% (27 of 194) of study participants. Only one of the 27 persons reported having no contact with MRSA-positive horses /91/. MRSA was also isolated from environmental samples within a veterinary teaching hospital /92/. Horses' stalls were identified as the most common source of MRSA contamination. A group from the United Kingdom also isolated MRSA-positive samples from humans, equines, companion animals, and environmental surfaces /93/. Although ST398 has been described in horses /94/, notably most reports of MRSA in horses have been strains other than ST398.

Transmission of MRSA ST1 between cows and humans has also been reported /95/. This study was the first to isolate MRSA from bovines and a human on the same farm, with the implication that zoonotic transmission had occurred. Other studies have identified MRSA in milk /96,97/ as another potential source of MRSA transmission. Although the samples identified were from raw milk, additional studies are needed to explore the role of milk as a potential reservoir for MRSA.

In addition to the findings of MRSA in pigs, horses, and cattle, MRSA has been identified in poultry. A study of broiler flocks and workers in slaughterhouses indicated that 5.6% of personnel were positive for MRSA /98/. In this study, broiler hangers were at higher risk than other personnel in the slaughterhouse. An interesting finding was that MRSA contamination increased in the different areas of the slaughterhouse during the production day. A separate group of investigators isolated livestock-associated MRSA in broiler chickens /99/. With the implication that MRSA found in poultry and meat products could be possible environmental sources of MRSA transmission to humans, more studies are warranted to identify the environmental sources of MRSA in livestock /100-102/.

With the detection of MRSA in the nasal passages of livestock, farmers, veterinarians, and others occupationally exposed to livestock, the question of airborne transmission of MRSA must be evaluated. A study to evaluate the presence of aerosolized antibiotic resistant bacteria in animal feeding operations detected *S. aureus* as the predominant bacteria present /103/. In another study, aerosolized *S. aureus* accounted for 76% of the bacteria detected 150 meters downwind from a confined animal feeding operation /104/. Resistant strains of *S. aureus* were also isolated from air samples taken at dairy cattle feeding operations /105/ and in chicken houses /106/. Not only is *S. aureus* an environmental contaminant in livestock feeding operations, a study in Texas also isolated antibiotic-resistant strains of *S. aureus* from air samples in residential homes /107/. This study detected higher levels of antibiotic-resistant strains of *S. aureus* inside the homes than outside. Although these studies did not examine MRSA specifically, the finding of airborne *S. aureus* in the air surrounding farms and in residential homes suggests that farm-associated MRSA could spread to neighboring residential buildings.

DISCUSSION

As MRSA becomes increasingly common in non-hospitalized human and animal populations, the potential for environmental contamination and therefore additional acquisition of these strains by the general population similarly increases. Indeed, the ability to survive for extended periods in the environment has been suggested as a factor in the generation or selection of strains capable of causing outbreaks /108/.

Although environmental disinfection may seem an appealing and obvious solution to eradicate MRSA, this approach may not always be a sound public health option. Certain MRSA carry genes that confer resistance to biocides, including quaternary ammonium compounds and triclosan /109-111/. Therefore, employing certain biocides could eliminate sensitive strains, thereby increasing the percentage of resistant organisms in the environment. Although guidelines for the control of MRSA include cleaning and disinfection steps /112/, such measures have been developed mainly for hospital use. The implementation of such procedures within other settings like farms requires evidence-based testing to determine their utility. If airborne MRSA is a main route of transmission on farms, for example, then simple surface cleaning and hand hygiene measures will not be adequate to reduce the prevalence of MRSA in these settings.

Because of the increasing ubiquity of MRSA in the population and the common occurrence of asymptomatic colonization without subsequent disease, the association between environmental contamination and the development of MRSA infection remains difficult to determine definitively. In the case of relatively rare strains in human populations, such as the livestock-associated ST398 strain, an association with livestock exposure or potentially, contaminated food products could be hypothesized. With the more common strains like USA300, however, a source may be more difficult to pinpoint. Complicating the matter, the identification of 'human' strains, such as ST5/USA100, both in meat and in food-animal species (including chickens and pigs /100,113,114/), blurs the distinction between common 'animal' and 'human' strains.

To determine instances of MRSA transmission, new research has demonstrated the effectiveness of a whole genome-sequencing strategy /115/, but this method is expensive and currently out of reach for many investigators. Nevertheless, additional genome-based methods must be implemented to better understand the ecology and epidemiology of MRSA, and to clarify further the role of the environment in MRSA transmission.

REFERENCES

1. Graham PL, 3rd, Lin SX, Larson EL. A U.S. population-based survey of Staphylococcus aureus colonization. Ann Intern Med 2006;144(5):318-25.
2. Lautenbach E, Nachamkin I, Hu B, Fishman NO, Tolomeo P, Prasad P, et al. Surveillance cultures foı detection of methicillin-resistant Staphylococcus aureus: diagnostic yield of anatomic sites and comparison of provider—and patient—collected samples. Infect Control Hosp Epidemiol 2009;30(4):380-2.
3. Shurland SM, Stine OC, Venezia RA, Johnson JK, Zhan M, Furuno JP, et al. Colonization sites of USA300 methicillin-resistant Staphylococcus aureus in residents of extended care facilities. Infect Control Hosp Epidemiol 2009;30(4):313-8.
4. Fritz SA, Epplin EK, Garbutt J, Storch GA. Skin infection in children colonized with community-associated methicillin-resistant Staphylococcus aureus. J Infect 2009;59(6):394-401.
5. Croft CA, Mejia VA, Barker DE, Maxwell RA, Dart BW, Smith PW, et al. Methicillin-resistant Staphylococcus aureus in a trauma population: does colonization predict infection? Am Surg 2009;75(6):458-61; discussion 61-2.
6. Groom AV, Wolsey DH, Naimi TS, Smith K, Johnson S, Boxrud D, et al. Community-acquired methicillin-resistant Staphylococcus aureus in a rural American Indian community. JAMA 2001; 286(10):1201-5.
7. Naimi TS, LeDell KH, Boxrud DJ, Groom AV, Steward CD, Johnson SK, et al. Epidemiology and clonality of community-acquired methicillin-resistant Staphylococcus aureus in Minnesota, 1996-1998. Clin Infect Dis 2001;33(7):990-6.
8. Fey PD, Said-Salim B, Rupp ME, Hinrichs SH, Boxrud DJ, Davis CC, et al. Comparative molecular analysis of community- or hospital-acquired methicillin-resistant Staphylococcus aureus. Antimicrob Agents Chemother 2003;47 (1):196-203.
9. Said-Salim B, Mathema B, Kreiswirth BN. Community-acquired methicillin-resistant Staphylococcus aureus: an emerging pathogen. Infect Control Hosp Epidemiol 2003;24(6):451-5.
10. Wulf M, Voss A. MRSA in livestock animals-an epidemic waiting to happen? Clin Microbiol Infect 2008;14(6):519-21.
11. Sheretz RJ, Reagan DR, Hampton KD, Robertson KL, Streed SA, Hoen HM, et al. A cloud adult: the Staphylococcus aureus-virus interaction revisited. Ann Intern Med 1996;124(6):539-47.
12. Sexton T, Clarke P, O'Neill E, Dillane T, Humphreys H. Environmental reservoirs of methicillin-resistant Staphylococcus aureus in isolation rooms: correlation with patient isolates and implications for hospital hygiene. J Hosp Infect 2006;62(2):187-94.
13. Hardy KJ, Oppenheim BA, Gossain S, Gao F, Hawkey PM. A study of the relationship between environmental contamination with methicillin-resistant Staphylococcus aureus (MRSA) and patients' acquisition of MRSA. Infect Control

Hosp Epidemiol 2006;27(2):127-32.
14. Rampling A, Wiseman S, Davis L, Hyett AP, Walbridge AN, Payne GC, et al. Evidence that hospital hygiene is important in the control of methicillin-resistant Staphylococcus aureus. J Hosp Infect 2001;49(2):109-16.
15. Rutala WA, Katz EB, Sherertz RJ, Sarubbi FA, Jr. Environmental study of a methicillin-resistant Staphylococcus aureus epidemic in a burn unit. J Clin Microbiol 1983;18(3):683-8.
16. Boyce JM, Havill NL, Otter JA, Adams NM. Widespread environmental contamination associated with patients with diarrhea and methicillin-resistant Staphylococcus aureus colonization of the gastrointestinal tract. Infect Control Hosp Epidemiol 2007;28(10):1142-7.
17. Blythe D, Keenlyside D, Dawson SJ, Galloway A. Environmental contamination due to methicillin-resistant Staphylococcus aureus (MRSA) J Hosp Infect 1998;38(1):67-9.
18. Oie S, Suenaga S, Sawa A, Kamiya A. Association between isolation sites of methicillin-resistant Staphylococcus aureus (MRSA) in patients with MRSA-positive body sites and MRSA contamination in their surrounding environmental surfaces. Jpn J Infect Dis 2007;60(6):367-9.
19. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant Staphylococcus aureus: possible infection control implications. Infect Control Hosp Epidemiol 1997;18(9):622-7.
20. French GL, Otter JA, Shannon KP, Adams NM, Watling D, Parks MJ. Tackling contamination of the hospital environment by methicillin-resistant Staphylococcus aureus (MRSA): a comparison between conventional terminal cleaning and hydrogen peroxide vapour decontamination. J Hosp Infect 2004;57(1):31-7.
21. Bures S, Fishbain JT, Uyehara CF, Parker JM, Berg BW. Computer keyboards and faucet handles as reservoirs of nosocomial pathogens in the intensive care unit. Am J Infect Control 2000; 28(6):465-71.
22. Oie S, Hosokawa I, Kamiya A. Contamination of room door handles by methicillin-sensitive/methicillin-resistant Staphylococcus aureus. J Hosp Infect 2002;51(2):140-3.
23. Lu PL, Siu LK, Chen TC, Ma L, Chiang WG, Chen YH, et al. Methicillin-resistant Staphylococcus aureus and Acinetobacter baumannii on computer interface surfaces of hospital wards and association with clinical isolates. BMC Infect Dis 2009;9:164.
24. Brown NM, Lee SD, Duerden BI, Gillanders SA, Cookson B, Neville L, et al. MRSA in non-clinical areas of hospitals. J Hosp Infect 2006;64(4):402-3.
25. Kei J, Richards JR. The prevalence of methicillin-resistant Staphylococcus aureus on inanimate objects in an urban emergency department. J Emerg Med 2008 Dec 24 [Epub ahead of print].
26. Dominguez MA, de Lencastre H, Linares J, Tomasz A. Spread and maintenance of a dominant methicillin-resistant Staphylococcus aureus (MRSA) clone during an outbreak of MRSA disease in a Spanish hospital. J Clin Microbiol 1994;32(9): 2081-7.
27. O' Fallon E, Schreiber R, Kandel R, D'Agata EM. Multidrug-resistant gram-negative bacteria at a long-term care facility: assessment of residents, healthcare workers, and inanimate surfaces. Infect Control Hosp Epidemiol 2009;30(12):1172-9.
28. Schultsz C, Meester HH, Kranenburg AM, Savelkoul PH, Boeijen-Donkers LE, Kaiser AM, et al. Ultra-sonic nebulizers as a potential source of methicillin-resistant Staphylococcus aureus causing an outbreak in a university tertiary care hospital. J Hosp Infect 2003;55(4):269-75.
29. Bhalla A, Pultz NJ, Gries DM, Ray AJ, Eckstein EC, Aron DC, et al. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalized patients. Infect Control Hosp Epidemiol 2004;25(2):164-7.
30. Gaspard P, Eschbach E, Gunther D, Gayet S, Bertrand X, Talon D. Meticillin-resistant Staphylococcus aureus contamination of healthcare workers' uniforms in long-term care facilities. J Hosp Infect 2009;71(2):170-5.
31. Cotterill S, Evans R, Fraise AP. An unusual source for an outbreak of methicillin-resistant Staphylococcus aureus on an intensive therapy unit. J Hosp Infect 1996;32(3):207-16.
32. Kumari DN, Haji TC, Keer V, Hawkey PM, Duncanson V, Flower E. Ventilation grilles as a potential source of methicillin-resistant Staphylococcus aureus causing an outbreak in an orthopaedic ward at a district general hospital. J Hosp Infect 1998;39(2):127-33.
33. Wagenvoort JH, Davies BI, Westermann EJ, Werink TJ, Toenbreker HM. MRSA from air-exhaust channels. Lancet. 1993;341(8848):840-1.

34. Shiomori T, Miyamoto H, Makishima K, Yoshida M, Fujiyoshi T, Udaka T, et al. Evaluation of bedmaking-related airborne and surface methicillin-resistant Staphylococcus aureus contamination. J Hosp Infect 2002;50(1):30-5.
35. Shiomori T, Miyamoto H, Makishima K. Significance of airborne transmission of methicillin-resistant Staphylococcus aureus in an otolaryngology-head and neck surgery unit. Arch Otolaryngol Head Neck Surg 2001;127(6):644-8.
36. Gorwitz RJ, Kruszon-Moran D, McAllister SK, McQuillan G, McDougal LK, Fosheim GE, et al. Changes in the prevalence of nasal colonization with Staphylococcus aureus in the United States, 2001-2004. J Infect Dis 2008;197(9):1226-34.
37. Malik S, Vranken P, Silio M, Ratard R, Van Dyke R. Prevalence of community-associated methicillin-resistant Staphylococcus aureus colonization outside the healthcare environment. Epidemiol Infect 2009;137(9):1237-41.
38. Choi CS, Yin CS, Bakar AA, Sakewi Z, Naing NN, Jamal F, et al. Nasal carriage of Staphylococcus aureus among healthy adults. J Microbiol Immunol Infect 2006;39(6):458-64.
39. Zanelli G, Sansoni A, Zanchi A, Cresti S, Pollini S, Rossolini GM, et al. Staphylococcus aureus nasal carriage in the community: a survey from central Italy. Epidemiol Infect 2002;129(2):417-20.
40. Furuya EY, Cook HA, Lee MH, Miller M, Larson E, Hyman S, et al. Community-associated methicillin-resistant Staphylococcus aureus prevalence: how common is it? A methodological comparison of prevalence ascertainment. Am J Infect Control 2007;35(6):359-66.
41. Mainous AG 3rd, Hueston WJ, Everett CJ, Diaz VA. Nasal carriage of Staphylococcus aureus and methicillin-resistant S. aureus in the United States, 2001-2002. Ann Fam Med 2006;4(2):132-7.
42. Gorwitz RJ, Kruszon-Moran D, McAllister SK, McQuillan G, McDougal LK, Fosheim GE, et al. Changes in the prevalence of nasal colonization with Staphylococcus aureus in the United States, 2001-2004. J Infect Dis 2008;197(9):1226-34.
43. Saxena S, Singh K, Talwar V. Methicillin-resistant Staphylococcus aureus prevalence in community in the east Delhi area. Jpn J Infect Dis 2003;56(2):54-6.
44. Scott E, Duty S, Callahan M. A pilot study to isolate Staphylococcus aureus and methicillin-resistant S aureus from environmental surfaces in the home. Am J Infect Control 2008;36(6):458-60.
45. Sing A, Tuschak C, Hormansdorfer S. Methicillin-resistant Staphylococcus aureus in a family and its pet cat. N Engl J Med 2008;358(11):1200-1.
46. Faires MC, Tater KC, Weese JS. An investigation of methicillin-resistant Staphylococcus aureus colonization in people and pets in the same household with an infected person or infected pet. J Am Vet Med Assoc 2009;235(5):540-3.
47. Hanselman BA, Kruth SA, Rousseau J, Weese JS. Coagulase positive staphylococcal colonization of humans and their household pets. Can Vet J 2009;50(9):954-8.
48. Oller AR, Mitchell A. Staphylococcus aureus recovery from cotton towels. J Infect Dev Ctries 2009;3(3):224-8.
49. Huijsdens XW, van Santen-Verheuvel MG, Spalburg E, Heck ME, Pluister GN, Eijkelkamp BA, et al. Multiple cases of familial transmission of community-acquired methicillin-resistant Staphylococcus aureus. J Clin Microbiol 2006;44(8): 2994-6.
50. Johansson PJ, Gustafsson EB, Ringberg H. High prevalence of MRSA in household contacts. Scand J Infect Dis 2007;39(9):764-8.
51. Allen KD, Anson JJ, Parsons LA, Frost NG. Staff carriage of methicillin-resistant Staphylococcus aureus (EMRSA 15) and the home environment: a case report. J Hosp Infect 1997;35(4):307-11.
52. Masterton RG, Coia JE, Notman AW, Kempton-Smith L, Cookson BD. Refractory methicillin-resistant Staphylococcus aureus carriage associated with contamination of the home environment. J Hosp Infect 1995 Apr;29(4):318-9.
53. Oie S, Kamiya A. Survival of methicillin-resistant Staphylococcus aureus (MRSA) on naturally contaminated dry mops. J Hosp Infect 1996;34(2): 145.
54. Stepanovic S, Cirkovic I, Djukic S, Vukovic D, Svabic-Vlahovic M. Public transport as a reservoir of methicillin-resistant staphylococci. Lett Appl Microbiol 2008;47(4):339-41.
55. Huijsdens XW, Janssen M, Renders NH, Leenders A, van Wijk P, van Santen Verheuvel MG, et al. Methicillin-resistant Staphylococcus aureus in a beauty salon, the Netherlands. Emerg Infect Dis 2008;14(11):1797-9.
56. Soge OO, Meschke JS, No DB, Roberts MC. Characterization of methicillin-resistant Staphylococcus aureus and methicillin-resistant coagulase-negative Staphylococcus spp. isolated from US West Coast public marine beaches. J Antimicrob Chemother 2009;64(6):1148-55.

57. Borjesson S, Matussek A, Melin S, Lofgren S, Lindgren PE. Methicillin-resistant Staphylococcus aureus (MRSA) in municipal wastewater: an uncharted threat? J Appl Microbiol 2009 Aug 6.
58. Lewis DL, Gattie DK, Novak ME, Sanchez S, Pumphrey C. Interactions of pathogens and irritant chemicals in land-applied sewage sludges (biosolids). BMC Public Health 2002;2:11.
59. Tolba O, Loughrey A, Goldsmith CE, Millar BC, Rooney PJ, Moore JE. Survival of epidemic strains of healthcare (HA-MRSA) and community-associated (CA-MRSA) meticillin-resistant Staphylococcus aureus (MRSA) in river-, sea- and swimming pool water. Int J Hyg Environ Health 2008;211(3-4):398-402.
60. Xu J, Moore JE, Millar BC. Ribosomal DNA (rDNA) identification of the culturable bacterial flora on monetary coinage from 17 currencies. J Environ Health 2005;67(7):51-5.
61. Tolba O, Loughrey A, Goldsmith CE, Millar BC, Rooney PJ, Moore JE. Survival of epidemic strains of nosocomial- and community-acquired methicillin-resistant Staphylococcus aureus on coins. Am J Infect Control 2007;35(5):342-6.
62. Hewlett AL, Falk PS, Hughes KS, Mayhall CG. Epidemiology of methicillin-resistant Staphylococcus aureus in a university medical center day care facility. Infect Control Hosp Epidemiol 2009; 30(10):985-92.
63. Ramana KV, Mohanty SK, Wilson CG. Staphylococcus aureus colonization of anterior nares of school going children. Indian J Pediatr 2009;76(8):813-6.
64. Lo WT, Lin WJ, Tseng MH, Lu JJ, Lee SY, Chu ML, et al. Nasal carriage of a single clone of community-acquired methicillin-resistant Staphylococcus aureus among kindergarten attendees in northern Taiwan. BMC Infect Dis 2007;7:51.
65. Rohde RE, Denham R, Brannon A. Methicillin resistant Staphylococcus aureus: carriage rates and characterization of students in a Texas university. Clin Lab Sci 2009;22(3):176-84.
66. Miller M, Cook HA, Furuya EY, Bhat M, Lee MH, Vavagiakis P, et al. Staphylococcus aureus in the community: colonization versus infection. PLoS ONE. 2009;4(8):e6708.
67. Fritz SA, Epplin EK, Garbutt J, Storch GA. Skin infection in children colonized with community-associated methicillin-resistant Staphylococcus aureus. J Infect 2009;59(6):394-401.
68. Montgomery K, Ryan TJ, Krause A, Starkey C. Assessment of athletic health care facility surfaces for MRSA in the secondary school setting. J Environ Health 2010;72(6):8-11.
69. Stanforth B, Krause A, Starkey C, Ryan TJ. Prevalence of community-associated methicillin-resistant Staphylococcus aureus in high school wrestling environments. J Environ Health 2010;72 (6):12-6.
70. Centers for Disease Control and Prevention (CDC). Methicillin-resistant Staphylococcus aureus infections among competitive sports participants—Colorado, Indiana, Pennsylvania, and Los Angeles County, 2000-2003. MMWR. 2003;52(33):793-5.
71. Mattern CS, Rotbart HA. Germ proof your school. School Nurse News 2008;25(4):31-4.
72. Alex A, Letizia M. Community-acquired methicillin-resistant Staphylococcus aureus: considerations for school nurses. J Sch Nurs 2007;23(4):210-3.
73. Lindenmayer JM, Schoenfeld S, O'Grady R, Carney JK. Methicillin-resistant Staphylococcus aureus in a high school wrestling team and the surrounding community. Arch Int Med 1998;27; 158(8):895-9.
74. Buss BF, Mueller SW, Theis M, Keyser A, Safranek TJ. Population-based estimates of methicillin-resistant Staphylococcus aureus (MRSA) infections among high school athletes—Nebraska, 2006-2008. J Sch Nurs 2009;25(4):282-91.
75. Rihn JA, Posfay-Barbe K, Harner CD, Macurak A, Farley A, Greenawalt K, et al. Community-acquired methicillin-resistant Staphylococcus aureus outbreak in a local high school football team unsuccessful interventions. Ped Infect Dis J 2005;24(9):841-3.
76. Romano R, Lu D, Holtom P. Outbreak of community-acquired methicillin-resistant Staphylococcus aureus skin infections among a collegiate football team. J Athl Train 2006;41(2):141-5.
77. Bowers AL, Huffman GR, Sennett BJ. Methicillin-resistant Staphylococcus aureus infections in collegiate football players. Med Sci Sports Exerc 2008;40(8):1362-7.
78. Kazakova SV, Hageman JC, Matava M, Srinivasan A, Phelan L, Garfinkel B, et al. A clone of methicillin-resistant Staphylococcus aureus among professional football players. NEJM 2005;352(5):468-75.
79. Cohen PR. The skin in the gym: a comprehensive review of the cutaneous manifestations of community-acquired methicillin-resistant Staphylococcus aureus infection in athletes. Clin Dermatol 2008;26(1):16-26.

80. Beam JW, Buckley B. Community-acquired methicillin-resistant Staphylococcus aureus: prevalence and risk factors. J Athl Train 2006;41 (3):337-40.
81. Cutri ght C. Staph death jars county. Roanoke, VA: The Roanoke Times; 2007.
82. A nonymous. More MRSA Cases Reported. 2007 October 20, 2007 [cited; Available from: http:// abclocal.go.com/wpvi/story?section=news/health &id=5715361]
83. Ale x A, Letizia M. Community-acquired methicillin-resistant Staphylococcus aureus: considerations for school nurses. J Sch Nurs 2007;23(4):210-3.
84. Springer B, Orendi U, Much P, Hoger G, Ruppitsch W, Krziwanek K, et al. Methicillin-resistant Staphylococcus aureus: a new zoonotic agent? Wien Klin Wochenschr. 2009;121(3-4):86-90.
85. van Loo I, Huijsdens X, Tiemersma E, de Neeling A, van de Sande-Bruinsma N, Beaujean D, et al. Emergence of methicillin-resistant Staphylococcus aureus of animal origin in humans. Emerg Infect Dis 2007;13:1834-9.
86. Van den Broek IV, Van Cleef BA, Haenen A, Broens EM, Van der Wolf PJ, Van den Broek MJ, et al. Methicillin-resistant Staphylococcus aureus in people living and working in pig farms. Epidemiol Infect 2009;137(5):700-8.
87. Wulf MW, Tiemersma E, Kluytmans J, Bogaers D, Leenders AC, Jansen MW, et al. MRSA carriage in healthcare personnel in contact with farm animals. J Hosp Infect 2008;70(2):186-90.
88. Graham JP, Evans SL, Price LB, Silbergeld EK. Fate of antimicrobial-resistant enterococci and staphylococci and resistance determinants in stored poultry litter. Environ Res 2009;109(6): 682-9.
89. Graha m JP, Price LB, Evans SL, Graczyk TK, Silbergeld EK. Antibiotic resistant enterococci and staphylococci isolated from flies collected near confined poultry feeding operations. Sci Total Environ 2009;407(8):2701-10.
90. Seguin JC, Walker RD, Caron JP, Kloos WE, George CG, Hollis RJ, et al. Methicillin-resistant Staphylococcus aureus outbreak in a veterinary teaching hospital: potential human-to-animal transmission. J Clin Microbiol 1999;37(5):1459-63.
91. Weese JS, Archambault M, Willey BM, Hearn P, Kreiswirth BN, Said-Salim B, et al. Methicillin-resistant Staphylococcus aureus in horses and horse personnel, 2000-2002. Emerg Infect Dis 2005;11(3):430-5.
92. Weese JS, DaCosta T, Button L, Goth K, Ethier M, Boehnke K. Isolation of methicillin-resistant Staphylococcus aureus from the environment in a veterinary teaching hospital. J Vet Intern Med 2004;18(4):468-70.
93. Moodley A, Stegger M, Bagcigil AF, Baptiste KE, Loeffler A, Lloyd DH, et al. Spa typing of methicillin-resistant Staphylococcus aureus isolated from domestic animals and veterinary staff in the UK and Ireland. J Antimicrob Chemother 2006;58 (6):1118-23.
94. van Duijkeren E, Moleman M, Sloet van Oldruitenborgh-Oosterbaan MM, Multem J, Troelstra A, Fluit AC, et al. Methicillin-resistant Staphylococcus aureus in horses and horse personnel: An investigation of several outbreaks. Vet Microbiol 2009 Aug 8.
95. Juhasz -Kaszanyitzky E, Janosi S, Somogyi P, Dan A, van der Graaf-van Bloois L, van Duijkeren E, et al. MRSA transmission between cows and humans. Emerg Infect Dis 2007;13(4):630-2.
96. Lee JH. Methicillin (Oxacillin)-resistant Staphylococcus aureus strains isolated from major food animals and their potential transmission to humans. Appl Environ Microbiol 2003;69(11):6489-94.
97. Moon JS, Lee AR, Kang HM, Lee ES, Kim MN, Paik YH, et al. Phenotypic and genetic antibiogram of methicillin-resistant staphylococci isolated from bovine mastitis in Korea. J Dairy Sci 2007;90(3):1176-85.
98. Mulders MN, Haenen AP, Geenen PL, Vesseur PC, Poldervaart ES, Bosch T, et al. Prevalence of livestock-associated MRSA in broiler flocks and risk factors for slaughterhouse personnel in The Netherlands. Epidemiol Infect 2010;9:1-13.
99. Persoons D, Van Hoorebeke S, Hermans K, Butaye P, de Kruif A, Haesebrouck F, et al. Methicillin-resistant Staphylococcus aureus in poultry. Emerg Infect Dis 2009;15(3):452-3.
100. Pu S, Han F, Ge B. Isolation and characterization of methicillin-resistant Staphylococcus aureus from Louisiana retail meats. Appl Environ Microbiol 2008 Oct 31.
101. van Loo IH, Diederen BM, Savelkoul PH, Woudenberg JH, Roosendaal R, van Belkum A, et al. Methicillin-resistant Staphylococcus aureus in meat products, the Netherlands. Emerg Infect Dis 2007 Nov;13(11):1753-5.
102. de Boer E, Zwartkruis-Nahuis JT, Wit B, Huijsdens XW, de Neeling AJ, Bosch T, et al. Prevalence of methicillin-resistant Staphylococcus aureus in meat. Int J Food Microbiol 2009;134(1-

2):52-6.
103. Gibbs SG, Green CF, Tarwater PM, Scarpino PV. Airborne antibiotic resistant and nonresistant bacteria and fungi recovered from two swine herd confined animal feeding operations. J Occup Environ Hyg 2004;1(11):699-706.
104. Green CF, Gibbs SG, Tarwater PM, Mota LC, Scarpino PV. Bacterial plume emanating from the air surrounding swine confinement operations. J Occup Environ Hyg 2006;3(1):9-15.
105. Alvarado CS, Gandara A, Flores C, Perez HR, Green CF, Hurd WW, et al. Seasonal changes in airborne fungi and bacteria at a dairy cattle concentrated animal feeding operation in the southwest United States. J Environ Health 2009; 71(9):40-4.
106. Zhong Z, Chai T, Duan H, Miao Z, Li X, Yao M, et al. REP-PCR tracking of the origin and spread of airborne Staphylococcus aureus in and around chicken house. Indoor Air 2009;19(6):511-6.
107. Gandara A, Mota LC, Flores C, Perez HR, Green CF, Gibbs SG. Isolation of Staphylococcus aureus and antibiotic-resistant Staphylococcus aureus from residential indoor bioaerosols. Environ Health Perspect 2006;114(12):1859-64.
108. Wagenvoort JH, Sluijsmans W, Penders RJ. Better environmental survival of outbreak vs. sporadic MRSA isolates. J Hosp Infect 2000;45(3):231-4.
109. Noguchi N, Hase M, Kitta M, Sasatsu M, Deguchi K, Kono M. Antiseptic susceptibility and distribution of antiseptic-resistance genes in methicillin-resistant Staphylococcus aureus. FEMS Microbiol Lett 1999;172(2):247-53.
110. Noguchi N, Suwa J, Narui K, Sasatsu M, Ito T, Hiramatsu K, et al. Susceptibilities to antiseptic agents and distribution of antiseptic-resistance genes qacA/B and smr of methicillin-resistant Staphylococcus aureus isolated in Asia during 1998 and 1999. J Med Microbiol 2005;54(Pt 6): 557-65.
111. Bayston R, Ashraf W, Smith T. Triclosan resistance in methicillin-resistant Staphylococcus aureus expressed as small colony variants: a novel mode of evasion of susceptibility to antiseptics. J Antimicrob Chemother 2007;59(5):848-53.
112. Anonymous. Revised guidelines for the control of epidemic methicillin-resistant Staphylococcus aureus. Report of a combined working party of the Hospital Infection Society and British Society for Antimicrobial Chemotherapy. J Hosp Infect 1990; 16(4):351-77.
113. Khanna T, Friendship R, Dewey C, Weese JS. Methicillin resistant Staphylococcus aureus colonization in pigs and pig farmers. Vet Microbiol 2008;128(3-4):298-303.
114. Lowder BV, Guinane CM, Ben Zakour NL, Weinert LA, Conway-Morris A, Cartwright RA, et al. Recent human-to-poultry host jump, adaptation, and pandemic spread of Staphylococcus aureus. Proc Natl Acad Sci USA 2009;106 (46):19545-50.
115. Harris SR, Feil EJ, Holden MT, Quail MA, Nickerson EK, Chantratita N, et al. Evolution of MRSA during hospital transmission and intercontinental spread. Science;327(5964):469-74.