A phase 2 trial of daclatasvir for the treatment of hepatitis C infection
See page 671

Modelling of global mortality associated with 2009 pandemic influenza
See page 687

Household transmission of MRSA and other staphylococci
See page 703
Household transmission of meticillin-resistant Staphylococcus aureus and other staphylococci

Meghan F Davis, Sally Ann Iverson, Patrick Baron, Aimee Vasse, Ellen K Silbergeld, Ebbing Lautenbach*, Daniel O Morris*

Although the role of pets in household transmission of meticillin-resistant Staphylococcus aureus (MRSA) has been examined previously, only minor attention has been given to the role of the abiotic household environment independent of, or in combination with, colonisation of pets and human beings to maintain transmission cycles of MRSA within the household. This report reviews published work about household transmission of S aureus and other staphylococci and describes contamination of household environmental surfaces and colonisation of pets and people. Household microbial communities might have a role in transfer of antimicrobial resistance genes and could be reservoirs for recolonisation of people, although additional research is needed regarding strategies for decontamination of household environments. Household-based interventions should be developed to control recurrent S aureus infections in the community, and coordination between medical and veterinary providers could be beneficial.

Introduction

The large rise in prevalence of community-associated meticillin-resistant Staphylococcus aureus (MRSA) has focused attention on control of community sources of MRSA.1–3 Households are a potential point of exchange, not only for MRSA, but also for other staphylococci of veterinary and human clinical importance, including meticillin-resistant Staphylococcus pseudintermedius, Staphylococcus schleiferi, and coagulase-negative staphylococci.4,4 Outbreaks of MRSA infection can occur within and among related households.5–7 In some cases, strain characteristics, such as biofilm production and presence of Panton-Valentine leukocidin, have been implicated in persistent colonisation within households.7 Such factors are important to consider for investigation of the epidemiology and control of MRSA in community settings.8–10,11 Reports of isolation of MRSA from pets (mainly dogs and cats) have also increased.12,13 Some researchers speculate that the human epidemic is driving the veterinary epidemic;13,14 however, transmission can occur in both directions between people and many species of pets and food animals.15,16 The potential for pets within the household to help transmission of MRSA1,15 and veterinary staphylococci1,15 has been reviewed, although more recent research adds to our understanding of concurrent human and pet colonisation within households. The ability of staphylococcal species to survive on environmental surfaces17,18 increases the likelihood of strains establishing populations that persist in household settings. Although reports have previously addressed environmental contamination in health-care settings,19,20 little attention21–25 has been given to environmental surface contamination in households. Moreover, non-aureus staphylococci might have a role in horizontal transfer of genes—for example, genes for antimicrobial resistance—within household microbial communities.26,27 We review the roles of indoor home environments and colonisation of people and pets in maintenance of transmission cycles for staphylococci within households.

Environmental survival of staphylococci

The genus Staphylococcus includes environmentally hardy microorganisms that remain viable in dry environments for periods of at least a week to 3 months or longer.28,29 Survival seems to be dependent on dust composition, temperature, humidity, surface material, and strain.28–31 all of which might affect the longevity of staphylococci in individual households. Gram-positive bacteria are predominant in indoor dust,19 and most household bacteria are of human origin.32 Colonised or infected people and pets can shed bacteria into the environment through direct contact with surfaces,33 shedding of skin cells with adherent bacteria,34 aerosol discharge by cloud carriers35—particularly sneezing—and gastrointestinal routes.36 S aureus and other staphylococci are usually transmitted by direct person-to-person contact; however, routes of indirect transmission might include environmental exposure through aerosols, settled dust, and fomites.37–40

Various human,41–43 pet,44,45,46 and environmental47–49 pathways of introduction of staphylococci into the household exist (figure 1). For example, genetic relatedness of S aureus USA300 (strain ST8) isolates collected from domestic and public surfaces on a university campus suggests transfer between the community and the household.50 People in Canada and the USA report...

Figure 1: Potential points of entry of staphylococci and genes for antimicrobial resistance into households
spending more than 60% of their time indoors at home and children younger than 11 years can spend more than 70% of their time indoors at home.\(^{18}\) As a result of both the time people and animals spend indoors at home, and the frequency of contact within a household, an understanding of these potential exposure pathways is crucial for the study of community dynamics of staphylococcal transmission.

The household environment has been a generally unrecognised reservoir for *S aureus*, although environmental transmission to people has been documented in hospitals\(^{18-21,56}\) and community\(^{17}\) settings. Several case reports\(^{20-23}\) and one study\(^{19}\) have implicated positive home environments as persistent sources for recolonisation of people after decolonisation therapy. Results of a case-control study show that household environmental contamination with community-acquired MRSA USA300 was associated with re-infection of index case patients,\(^{18}\) suggesting that contamination of the household also has consequences for clinical disease. Whereas hospital and public community settings are characterised by transient contact by a diverse population, households have high-intensity contact between the same individuals. As a result, transmission dynamics within households might differ from those of public settings, with implications for control measures. Additionally, indoor home settings are usually beyond administrative authority, placing a burden on the patient to ensure that the environment is hygienic.

In households, several surfaces are potential sites of *S aureus* contamination (table 1). Common room surfaces such as cabinet tops or shelves\(^{50,51,54}\) can be contaminated with *S aureus*. These sites are handled infrequently, so bacteria identified there might have survived for long periods or bacteria adhered to airborne dust might have settled. Two studies have examined airborne *S aureus*,\(^{49,62}\) one of which\(^{49}\) identified *S aureus* in household kitchen air near rubbish. Researchers have speculated that food items might introduce *S aureus* to the household by aerosolisation during handling or bacterial contamination of sinks, kitchen sponges, and other kitchen surfaces.\(^{49-52}\)

Commonly touched sites (figure 2) have high rates of contamination and could have an important role in indirect household transmission. Such fomites include toys,\(^{50,52,61}\) television remote controls or similar objects,\(^{12,19,60}\) telephones,\(^{54}\) door knobs,\(^{52,59,61}\) pillows and bedding,\(^{52,58}\) hand towels,\(^{53,54,61}\) and taps.\(^{52,63}\) In a

<table>
<thead>
<tr>
<th>Region</th>
<th>Study design</th>
<th>Households</th>
<th>Sampling method and sites</th>
<th>Results</th>
<th>Additional notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masterton and colleagues (1995)(^{20})</td>
<td>London, UK</td>
<td>Case report</td>
<td>One household</td>
<td>Sampling method not reported, many sites, negative sites not reported</td>
<td>MRSA-positive sites included door handles, computer desk surface, and computer joystick</td>
</tr>
<tr>
<td>Allen and colleagues (1997)(^{24})</td>
<td>Merseyside, UK</td>
<td>Case report</td>
<td>One household</td>
<td>Sweep plates: carpets, bedding, pillows, curtains, soft furnishings, and clothes</td>
<td>20 (48%) of 42 sweep plates were positive for MRSA, including pillows, bedding, carpets, and clothing; no swabs were MRSA positive</td>
</tr>
<tr>
<td>Ojima and colleagues (2002)(^{25})</td>
<td>Japan</td>
<td>Cross-sectional</td>
<td>Five households</td>
<td>Agar stamping used for 90 sites</td>
<td><em>S aureus</em> identified on sites touched by children and cloth toys, kitchen sponges and sink, taps, and refrigerator handle</td>
</tr>
<tr>
<td>Ojima and colleagues (2002)(^{25})</td>
<td>Japan</td>
<td>Cross-sectional</td>
<td>86 households</td>
<td>Agar stamping used for 100 sites</td>
<td><em>S aureus</em> identified at less than 20% of sites, but some sites were positive, including kitchen (drain traps, sponges, sinks, towels), sites touched by children (pillows, toys), bathroom sites (taps, toilet seats), and common room sites (television remote controls, door/knobs)</td>
</tr>
<tr>
<td>Kneihl and colleagues (2005)(^{26})</td>
<td>Germany</td>
<td>Prospective cohort</td>
<td>Eight health-care worker homes</td>
<td>Contact plates or swabs: bed, baths, carpets, chairs, telephone or mobile telephone, light switch, television remote or switches, and tuner switches</td>
<td>88% of households were positive for MRSA</td>
</tr>
<tr>
<td>Gandara and colleagues (2006)(^{27})</td>
<td>El Paso, TX, USA</td>
<td>Cross-sectional</td>
<td>24 households, part of a larger study</td>
<td>Aerosol (Anderson impactor or agar media collection); duplicate samples taken (two 10-15 min periods per home) in human breathing zone (1 m above floor) in living room</td>
<td>100% of households were positive for <em>S aureus</em> and nine (33%) of 23 homes were positive for multidrug-resistant <em>S aureus</em></td>
</tr>
</tbody>
</table>

(Continues on next page)
Laboratory study, transfer efficiencies for non-staphylococcal bacteria from hands to fomites was high for taps and telephone receivers, but low for textiles. This difference might be caused by different household conditions, or by frequent handling of cloth substrates in homes. Bedroom surfaces such as pillows and bedding might be especially important sites of indirect movement of such as pillows and bedding might be especially important sites of indirect movement of S aureus. Transmission. Crowding, residence in subsidised housing in the USA or Mexico, or frequent contact more frequently, increasing the likelihood of re-infection. Relatedness might vary by strain type, and many strains can be present. The number of household contacts might also affect transmission rates. A study reports that 25% of households had at least one contact colonised with MRSA and 9% had more than one contact colonised. Some, but not all, studies have also noted that households in which transmission occurs have a higher than average number of human occupants. People in large households might have direct contact more frequently, increasing the likelihood of transmission. Crowding, residence in subsidised housing or shelter, and residence in regions with high rates of incarceration are risk factors for MRSA skin and soft-tissue infections.

### Household members

Rates of transmission between positive case patients and household members range from less than 10% to 43% (table 2). Molecular characterisation of isolates is important, because household studies have reported lower rates of transmission when household members were assessed for strains related to the one identified in the index patient than when assessed for colonisation with any strain. Relatedness might vary by strain type, and many strains can be present. The number of household contacts might also affect transmission rates. A study reports that 25% of households had at least one contact colonised with MRSA and 9% had more than one contact colonised. Some, but not all, studies have also noted that households in which transmission occurs have a higher than average number of human occupants. People in large households might have direct contact more frequently, increasing the likelihood of transmission. Crowding, residence in subsidised housing or shelter, and residence in regions with high rates of incarceration are risk factors for MRSA skin and soft-tissue infections.
9 months in index patients, with much the same time for household contacts, but with a wide range; in one study, 43% of case patients were colonised for 2 months or less. In another study, having household contacts (for whom MRSA colonisation was not assessed) was associated with long duration of colonisation of the index case-patient, potentially related to re-exposure from colonised household members. A study of patients colonised with MRSA suggests carriers had intermittent negative results 26% of the time and intermittent carriers more frequently developed clinical MRSA infection during the study than did other carrier types, which emphasises the importance of longitudinal monitoring and of re-exposure from household sources.

An estimated 20% of people are persistent carriers who remain positive for MRSA for months or years. Even after decolonisation treatment, these people might be recolonised preferentially with the previously persistent strain if exposed to multiple strains. This effect makes decontamination of the home particularly crucial for strategies to decolonise persistent carriers. Potential exists for colonisation via transfer event or loss by treatment or natural clearance (figure 3).

Choice of anatomical sampling sites might affect surveillance estimates of transmission rates. The highest household transmission rates were from studies that tested multiple sites on all individuals, including nose, throat, skin, or perineum, and clinical lesions. Studies using nasal swabbing only estimated lower rates of transmission. Results of one study show that a survey sampling nostrils only would miss 48% of *S aureus*-positive and 51% of MRSA-positive household contacts. Non-nasal colonisation rates might be high (particularly for community-acquired MRSA strains), might affect transmission dynamics within households, and might help to explain epidemiological differences between patients with community-acquired MRSA, hospital-acquired MRSA, and meticillin-sensitive *S aureus*.

Colonisation of non-nasal sites—for example, the pharynx—has been implicated in transmission to household contacts. Other risk factors include increased duration of exposure (ie, time spent in the household), having more household contacts, having eczema or a previous skin infection, previously taking cefalexin, being younger or in the same age group as a child index patient, and being a partner of the index patient. Colonisation of groin or vaginal sites might
lead to sexual transmission\textsuperscript{77} or vertical transmission to infants during childbirth.\textsuperscript{68} Mother–infant pairs have been colonised\textsuperscript{69,70} or infected\textsuperscript{69,70} simultaneously with \textit{S aureus}.

**Children**

Children, through behaviour or susceptibility, might contribute to household transmission of \textit{S aureus} differently to adults. Children (aged 0–17 years) might be colonised with \textit{S aureus} more commonly\textsuperscript{44,93} and for longer\textsuperscript{80} than adults. Child family members of health-care workers have been implicated in transfer of MRSA involving household spread.\textsuperscript{41} In a day-care study, two children had an MRSA strain that matched one from staff members or household contacts.

**Table 2:** Reports of human household transmission of \textit{Staphylococcus aureus}

<table>
<thead>
<tr>
<th>Region</th>
<th>Study design</th>
<th>Index patient enrolment</th>
<th>Householder enrolment (n)</th>
<th>Locations sampled</th>
<th>Estimates of transmission rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lautenbach and colleagues (2010)\textsuperscript{79}</td>
<td>PA, USA Longitudinal</td>
<td>Eight patients with MRSA skin and soft tissue infection</td>
<td>7</td>
<td>Nostrils, armpits, throat, groin, perineum</td>
<td>43% of household members colonised with an MRSA strain related to that of the index patient</td>
</tr>
<tr>
<td>Johansson and colleagues (2007)\textsuperscript{71}</td>
<td>Sweden Cross-sectional</td>
<td>125 patients with MRSA infection or colonisation (51 with participating household contacts)</td>
<td>114</td>
<td>Nostrils, throat, perineum, any skin lesions</td>
<td>36% of household members (43% of households) with an MRSA strain related to that of the index patient</td>
</tr>
<tr>
<td>Mollema and colleagues (2010)\textsuperscript{69}</td>
<td>Netherlands Cross-sectional</td>
<td>46 patients and 16 health-care workers colonised with MRSA</td>
<td>160</td>
<td>Nostrils, throat, perineum, and any skin lesions</td>
<td>35% of household members (47% of households) colonised with an MRSA strain related to that of the index patient</td>
</tr>
<tr>
<td>Eveillard and colleagues (2004)\textsuperscript{75}</td>
<td>France Cross-sectional</td>
<td>Ten health-care workers colonised with MRSA</td>
<td>21</td>
<td>Nostrils</td>
<td>29% of household members (40% of households) colonised with an MRSA strain related to that of the index patient</td>
</tr>
<tr>
<td>Hewlett and colleagues (2009)\textsuperscript{76}</td>
<td>TX, USA Cross-sectional</td>
<td>Seven children and one adult employee colonised with MRSA (104 children and 32 employees screened) at a university day-care centre</td>
<td>17</td>
<td>Nostrils and oropharynx; additional sites in children (armpits, groin, perineal area)</td>
<td>Four (25%) of 16 of household members colonised with an MRSA strain related to that of the index patient; 36% of household members colonised with MRSA overall</td>
</tr>
<tr>
<td>Miller and colleagues (2012)\textsuperscript{78}</td>
<td>CA, USA and IL, USA Cross-sectional</td>
<td>350 patients with \textit{S aureus} skin and soft tissue infection</td>
<td>812</td>
<td>Nostrils, oropharynx, inguinal area</td>
<td>20% of household members colonised with a community-acquired MRSA strain and 17% with \textit{S aureus} related to that of the index patient; 22% of household members colonised with MRSA and 50% with any \textit{S aureus}</td>
</tr>
<tr>
<td>Huang and colleagues (2007)\textsuperscript{79}</td>
<td>Taiwan Cross-sectional</td>
<td>57 children with community-acquired MRSA skin and soft tissue infection</td>
<td>121</td>
<td>Nostrils</td>
<td>Roughly 15% of household members (64% of 28 index-household member pairs) colonised with an MRSA strain related to that of the index patient; 25% of household members (40% of households) colonised with MRSA overall</td>
</tr>
<tr>
<td>Calfee and colleagues (2003)\textsuperscript{80}</td>
<td>VA, USA Cross-sectional</td>
<td>88 patients with MRSA infection or colonisation undergoing eradication therapy</td>
<td>172</td>
<td>Nostrils, groin, axillae, perineal area, and non-intact skin also often cultured</td>
<td>11% of household members (43% of nine case-contact groups) colonised with an MRSA strain related to that of the index patient; 15% of household members colonised with any MRSA</td>
</tr>
<tr>
<td>Zafar and colleagues (2007)\textsuperscript{81}</td>
<td>Detroit, MI, USA Cross-sectional</td>
<td>51 patients with community-acquired MRSA infections (31 with household contacts)</td>
<td>49</td>
<td>Nostrils</td>
<td>10% of household members colonised with an MRSA strain related to that of the index patient; 20% of household members (36% of households) colonised with any MRSA</td>
</tr>
<tr>
<td>Faires and colleagues (2009)\textsuperscript{82}</td>
<td>Canada and USA Cross-sectional</td>
<td>Eight households with pets for which ≥1 person had ≥2 MRSA infections in the past year</td>
<td>36</td>
<td>Nostrils</td>
<td>6% of household members colonised with an MRSA strain related to that of the index patient</td>
</tr>
<tr>
<td>Nerby and colleagues (2011)\textsuperscript{83}</td>
<td>MN, USA Cross-sectional</td>
<td>232 children with community-acquired MRSA skin and soft tissue infection</td>
<td>699</td>
<td>Nostrils</td>
<td>Roughly 2% of household members (65% of household members in 11 households with index-household member pairs) colonised with an MRSA strain related to that of the index patient; 12% of household members (52% of households) colonised with any MRSA</td>
</tr>
<tr>
<td>Fritz and colleagues (2012)\textsuperscript{84}</td>
<td>MO, USA Cross-sectional (in randomised controlled trial)</td>
<td>187 children with \textit{S aureus} skin and soft tissue infection</td>
<td>609</td>
<td>Nostrils, armpits, inguinal folds</td>
<td>Index MRSA: 27% of household members colonised with MRSA; 24% colonised with MSSA; index MSSA: 9% of household members colonised with MRSA, 44% colonised with MSSA (strain identities not tested)</td>
</tr>
<tr>
<td>Lucet and colleagues (2009)\textsuperscript{85}</td>
<td>France Longitudinal</td>
<td>148 patients discharged to home care</td>
<td>188</td>
<td>Nares and (for index patients only) any skin lesions</td>
<td>19% of household members colonised with any MRSA (strain identities not tested)</td>
</tr>
</tbody>
</table>

\textit{MSSA}=meticillin-sensitive \textit{S aureus}. \textit{MRSA}=meticillin-sensitive \textit{S aureus}.
Infants might develop gastrointestinal colonisation with _S aureus_ shortly after birth and can stay positive for months. They also have high rates of perineal infection compared with other age groups. Because patients admitted to hospital with _S aureus_ gastrointestinal colonisation might contaminate their hospital room, gastrointestinal colonisation or perineal infection of infants might be a source of home environmental contamination. In households, infant changing areas and similar locations can be contaminated with _S aureus_. Children tend to interact closely with their environments, suggesting the potential to establish a cycle of environmental contamination and re-exposure to _staphylococci_, with possible implications for duration or risk of colonisation.

**Pets**

39% of US households has at least one dog, and 33% of households has at least one cat. Pets become colonised or infected with _S aureus_, including hospital-acquired MRSA strains (eg, ST22, EMRSA-15) and community-acquired MRSA strains (eg, Canadian MRSA-2, ST80) that cause disease in people. This finding has led some researchers to maintain that pets have the so-called humanosis of MRSA and are not true reservoirs of _S aureus_. Differentiation of true colonisation from transient carriage or superficial contamination is a challenge in veterinary studies. A greater proportion of dogs than cats seem to carry _S aureus_, and transmission between people and pets is probably bidirectional.

Like people, pets can become colonised or infected through nosocomial transmission in veterinary or human health-care settings where, for example, therapy pets reside with or visit patients. Repeated, intermittent carriage of particular MRSA strains noted in pets might result from poor sensitivity of testing protocols or might be because pets are recolonised from reservoirs, such as people or environmental surfaces, after natural clearance or decolonisation.

**Transmission between pets and people**

Transient carriage by or infection of pets might provide a conduit for transmission to people if animals are positive for long enough to expose people after decolonisation or natural clearance. Studies have bolstered earlier case reports examining the relation between MRSA positivity in people and pets within households. Prevalences of _S aureus_ and MRSA in people tend to equal or surpass prevalences in pets. Prevalence of MRSA in 122 households with 242 people living with pets (132 dogs and 161 cats) was 3·3% for people, 1·5% for dogs, and 0·0% for cats, whereas the prevalence of meticillin-susceptible _S aureus_ was 27·7% for people, 14·4% for dogs, and 4·3% for cats. Prevalence in people was consistent with US population-based data. A study of owners and their cohabiting dogs at a Berlin dog show notes a prevalence of _S aureus_ of 18·5% for people and 1·8% for dogs. In another study, one person and one pet were sampled from each of 586 households. 5·6% of people were colonised with MRSA and 21·5% of dogs, whereas the prevalence of _S aureus_ was 27·7% for people, 14·4% for dogs, and 4·3% for cats. Households of non-health-care workers, veterinary health-care workers, or human health-care workers did not differ, which is unexpected in view of previous studies of occupational risks for acquisition of MRSA in human and veterinary health-care environments. However, sampling just one person and one pet from each house might have affected the findings.

Strain relatedness between _staphylococci_ isolates from people and animals within households tends to be similar to those between human household members. 50–67% of households have indistinguishable strains of either _S aureus_ or MRSA. Although discovery of related strains in both people and animals suggests that transmission has occurred, it does not show the direction of movement (from people to animals, vice versa, or from a common source). Additionally, some _staphylococcal_ lineages might be better adapted to multispecies colonisation than are others, although more research is needed. Cross-sectional studies cannot be used to distinguish whether households are discordant because of low transmission or simply chance temporal dynamics (figure 3).

In addition to contact with veterinary clinics, surgery, and antimicrobial use, risk factors for pet colonisation with _S aureus_ include contact with children and licking behaviours. Interactions between children and pets within households might involve direct face-to-face contact through licking and biting, or indirect contact through shared environments. One study enrolled households with an infant and a pet, and reports an association between household environmental
contamination and presence of cats. In a longitudinal study of 29 pets living with MRSA-infected children, a dog and a cat were both positive for MRSA at only one of three visits 6 months apart, and each animal was colonised with the same USA300 strain as the child. Whether presence of pets and children together confers greater risk for household contamination is unknown. A case report of a persistently colonised health-care worker notes that neither her child nor her cat was a carrier, but that her home environment was contaminated; decontamination led to successful decolonisation.

Several studies have estimated transmission rates between people and pets. In households with an MRSA-positive pet, the prevalence of human carriage was 27%. In a case-control study of 49 MRSA-positive people with skin and soft-tissue infection and 50 MRSA-negative controls, colonisation with an identical MRSA strain occurred in four in-contact pets (two dogs, a cat, and a hamster), but in none of the pets from control households. The low transmission rates might be a result of the methods; pets were sampled by nasal swab only.

As in people, non-nasal anatomical sites—such as the mouth, perineum, and inguinal skin—are often colonised or contaminated with *S aureus* in dogs and cats. A positive dorsal fur site is probably a result of contamination from human hand or mouth contact; such contamination might be important for transmission but might not be indicative of pet colonisation status. Furthermore, differentiation of colonisation or decolonisation from transient contamination might be important for household transmission. Pets other than dogs and cats might also be important for transmission and should not be ignored in household studies. Clinical *S aureus* isolates, including MRSA, have been identified in parrots and other birds, hamsters and guineapigs, rats, small ruminants, iguanas, a turtle, and bats. Reptiles seem to be resistant to colonisation or contamination by staphylococci, perhaps because they are ectothermic; however, they might have less frequent contact with people than do furry pets, which could lessen transmission.

**Veterinary staphylococci**

Although *S aureus* is a leading cause of disease in people and can spill over into pet populations, *S pseudintermedius* (reclassified from *Staphylococcus intermedius*) and *S schleiferi* do the reverse, and predominantly cause disease in animals. Coagulase-positive staphylococci, particularly the typically multidrug-resistant and meticillin-resistant *S pseudintermedius*, are leading bacterial causes of skin infections in dogs and cats.

Although infection of people is rare, pet owners and veterinarians can become colonised, often transiently. Like *S aureus*, when humans are colonised with meticillin-resistant *S pseudintermedius*, they tend to share the same strains as their pets. Prevalences in people exposed to meticillin-resistant *S pseudintermedius* are estimated to be 4–12% in household studies and occupational cohorts. Nasal positivity in veterinary dermatologists in a conference setting, away from clinic sources of exposure, suggests that people can become truly colonised, not just transiently contaminated, with meticillin-resistant *S pseudintermedius*. In an occupational cohort of veterinary dermatologists and their pets, four (2%) of 171 households had related staphylococcal strains in both people and pets (one MRSA and three meticillin-resistant *S pseudintermedius*), and an additional seven households (4%) had related strains in pets only.

Few studies of household transmission of non-aureus staphylococci exist (table 3). The only study that assessed concurrent environmental contamination, human colonisation, and animal colonisation for any staphylococcal species included 16 households, visited at 6 monthly intervals, exposed to meticillin-resistant *S pseudintermedius*. The researchers report that, although some animals were persistently colonised with the same genetic strain, many were intermittently colonised or became negative. Household environments were positive even in the absence of concurrent human or animal colonisation. Such events (figure 3) might be important for recolonisation of pets or people after natural clearance or treatment.

**Horizontal genetic transfer in household staphylococci**

MRSA, meticillin-resistant *S pseudintermedius*, and other meticillin-resistant staphylococci share the staphylococcal chromosomal cassette, which contains the mecA gene conferring β lactam resistance through an altered penicillin-binding protein (SCCmec). Transfer of an SCCmec element from *Staphylococcus epidermidis* to meticillin-susceptible *S aureus* during antimicrobial treatment of a patient colonised with both, creating an MRSA strain, has been reported. Many SCCmec elements in meticillin-resistant *S pseudintermedius* are of distinct types or are combinations of elements from *S aureus* and other staphylococci; however, at least one (SCCmec type V) is largely homologous to the type in MRSA. In an outpatient study, a dog living with an MRSA-positive case-patient was not colonised with MRSA but was colonised with meticillin-resistant *S pseudintermedius*; the frequency of such joint colonisation of different staphylococci species within households is unknown. When multiple species of staphylococci are present simultaneously, horizontal gene transfer of SCCmec and other genes for resistance can occur within household microbial communities. However, results of one study show that SCCmec types were different in meticillin-resistant *S pseudintermedius* isolated from pets and
### Table 3: Reports of indoor household environmental contamination with meticillin-resistant, non-aureus staphylococci

<table>
<thead>
<tr>
<th>Region</th>
<th>Species</th>
<th>Study design</th>
<th>Households</th>
<th>Locations sampled</th>
<th>Results</th>
<th>Additional notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancholi and colleagues (2005)</td>
<td>Coagulase-negative staphylococci, unknown antimicrobial susceptibility</td>
<td>Cross-sectional</td>
<td>238 caregivers</td>
<td>Carer hand (glove juice method); moistened swabs taken from table surface</td>
<td>29% of households had related bacterial isolates on carer hands and table surfaces</td>
<td>21% of isolates were coagulase-negative staphylococci and the remainder were Gram-negative rods</td>
</tr>
<tr>
<td>Lis and colleagues (2009)</td>
<td>Meticillin-resistant coagulase-negative staphylococci</td>
<td>Cross-sectional</td>
<td>14 households</td>
<td>Aerosol (Anderson impactor with agar media collection)</td>
<td>Staphylococcus isolates from homes with health-care contact had higher prevalence of meticillin-resistance than did reference homes</td>
<td>24/47 (51%) of all pets were positive for meticillin-resistant S pseudintermedius, including nine pets that lived with an index case who was positive at the time of sampling. 2/45 (4%) people were nasally colonised</td>
</tr>
<tr>
<td>van Duijkeren and colleagues (2011)</td>
<td>Meticillin-resistant Staphylococcus pseudintermedius</td>
<td>Cross-sectional</td>
<td>20 households</td>
<td>Person, pet (index case and in-contact pets), and environmental samples taken concurrently; gauze samples taken from five to eight environmental surfaces; sleeping place of the index case (animal bedding), feeding site, floor underneath sofa, door mat, windowsill, cupboard</td>
<td>70% of households were contaminated with meticillin-resistant S pseudintermedius, particularly homes with concurrent index-patient colonisation or infection; feeding sites and bedding were frequently contaminated</td>
<td>24/57 (42%) intermittently positive for meticillin-resistant S pseudintermedius and 42% intermittently positive in-contact animals (n=7): 96% positive at least once during the study</td>
</tr>
<tr>
<td>Laarhoven and colleagues (2011)</td>
<td>Metillin-resistant S pseudintermedius</td>
<td>Longitudinal</td>
<td>16 households with dogs positive for meticillin-resistant S pseudintermedius - index case</td>
<td>Human (nasal swabs), pet (nasal and perineal samples from index case plus in-contact pets), and environmental samples taken concurrently at monthly intervals for 6 months; most environmental wipes were taken from 400 cm² areas from three sites (sleeping place of the index case, feeding place, and a site physically inaccessible to the animals)</td>
<td>0–59% of households were contaminated at visits, and sometimes households were contaminated in absence of positivity in people or animals; feeding sites were positive in 69% of households, sleeping places in 56% of households, and inaccessible sites in 38% of households</td>
<td>Index dogs (n=12): 8% continuously positive for meticillin-resistant S pseudintermedius and 42% intermittently positive</td>
</tr>
</tbody>
</table>

**Table 3: Reports of indoor household environmental contamination with meticillin-resistant, non-aureus staphylococci**

Meticillin-resistant coagulase-negative staphylococci isolates from people in the same household, indicating no common source of SCCmeC for household staphylococci. Previous use of antimicrobials—especially fluoroquinolones and cephalosporins—has been associated with later colonisation or infection with meticillin-resistant staphylococci in both people and animals. Frequent multidrug resistance is a concern, and particularly, the potential for selection or induction of drug resistance by antimicrobial use in *S. pseudintermedius* and potentially in other staphylococci. Widespread use of antimicrobials in kennels fosters persistence of carriage of meticillin-resistant *S. pseudintermedius* in dogs. Clinical reports of susceptible *S. pseudintermedius* or *S. schleiferi* becoming resistant after antimicrobial treatment in the same animal might suggest either that use of antimicrobials selected for the resistant strains, or that *S. pseudintermedius* is receptive to gene transfer within household or veterinary clinic microbial communities. Clinical infection of animals with multidrug-resistant meticillin-resistant *S. pseudintermedius* or MRSA in pets could necessitate prescription of antimicrobial drugs more commonly used in people, thus exposing the household microbiome to these drugs. The household microbiome should be investigated as a source of resistance genes, including quantification of genetic transfers for drug resistance and other virulence genes among species of staphylococci.

**Intervention strategies**

The effectiveness of hospital cleaning strategies to eradicate environmental staphylococci has been studied; however, few decontamination strategies have been tested in home environments. Reports suggest that heavy household environmental contamination might contribute to colonisation and re-colonisation of health-care workers. Gaseous ozone, replacement of carpets, commercial steam cleaning, general disinfection of hard surfaces, and replacement of mattresses have been used in households. Low-temperature home laundering is effective for clothing. Case studies of home environmental decontamination and a human decolonisation trial report laundering of bedding and towels as an intervention to curb transmission of MRSA.
However, laundered clothing can become recontaminated within hours if exposed in a health-care setting.28 Thus, laundering might need to be done frequently during decolonisation of household members.

*S aureus* and other bacteria, while susceptible to detergents under laboratory conditions, might not be inhibited by normal household use of dishwashing liquid in the presence of food residues.136 However, 2-1% sodium hypochlorite with a degreaser effectively reduces *S aureus* on kitchen items.95 Additionally, combination of chlorine and quaternary ammonium-based cleaners for disinfection of multiple sites in the kitchen, bathroom, and pet-associated areas reduced log colony-forming units of *S aureus* by 99-99%.95 Some strains of *S aureus* might contain mutations conferring resistance to household disinfectants,143,145 although the ability of such mutations to compromise household decontamination has not been reported.

More studies should assess the effectiveness of cleaning and disinfection methods. As shown by hospital disinfection studies21 and intervention studies examining staphylococci in conjunction with other bacteria,96 strategies targeting staphylococci might be relevant to other bacterial pathogens that spread within households, such as outbreak strains of *Escherichia coli.*97 Since many contaminated sites in the household are frequently touched, good hand hygiene practices might help to reduce contamination of these sites.92 A national study of recommendations given for household decolonisation in Scotland reports that less than half of regions did recommended housekeeping measures, such as changing bed linen daily.102 Furthermore, hospital studies have shown that environments can be rapidly recontaminated after decontamination.141 Decontamination of home environments will probably be needed in conjunction with human and animal treatment for some households. A general recommendation for household cleaning and laundering of human and animal bedding is warranted when people or animals are diagnosed with staphylococcal infection or colonisation.

Decolonisation strategies for people have been well described.103 Short-term treatment with the topical nasal antibiotic mupirocin can temporarily eradicate nasal carriage of MRSA,145,146 but the long-term value of this treatment is unclear.103,104 Exposure within the household or among other networks of human or animal contact outside the home might be sources for recolonisation of treated patients.

In 2010, the Clinical and Laboratory Standards Institute recommended screening and decolonisation for household members, for both people and animals, in cases of recurrent human infection within the household, as part of a household-wide investigation.145 Decolonisation of human household members in conjunction with an index patient identified with *S aureus* skin and soft tissue infection has been tested in a randomised clinical trial (NCT00731783).145 92 households were assigned to decolonisation of the index patient only, 91 were assigned to household-wide decolonisation with the same 5-day protocol of twice-daily 2% nasal mupirocin ointment and daily body washes with 4% chlorhexidine gluconate, along with improved hygiene measures.146 Despite identical efficacy for decolonisation of the index patient (roughly 50%), the household intervention group had significantly lower incidence of recurrent skin and soft tissue infections than did the individual decolonisation group, for both index patients and household contacts.146 Unexpectedly, over the 12 month longitudinal study period after decolonisation, the presence of pets in the household was protective against meticillin-sensitive *S aureus* colonisation in human household members; no relation existed between longitudinal MRSA colonisation in people and presence of pets in the household. Pet colonisation status, however, was not assessed (Fritz S, Washington University School of Medicine, personal communication). A cross-sectional study also provided weak evidence that presence of a pet protected against colonisation of the index patient with the infecting strain type (odds ratio [OR] 0·70; p=0·32), but such presence did not protect household members from colonisation (OR 1·01; p=0·83).13 Colonisation by veterinary staphylococcal species after treatment of the index patient might result in a complex microbial ecology that could offer some protection from recolonisation by pathogenic MRSA strains.

When dogs or cats become persistently infected with *S aureus*, including MRSA, effective treatment must be given in conjunction with decolonisation of people, since people are probably a source of *S aureus* for pets. Infected pets should be treated on a case-by-case basis under veterinary supervision, and clients should be instructed in good hygiene and wound care practices to help reduce potential for environmental contamination and spread to people.13,14 Pets often clear colonisation without drug treatment.14 Reduction of person and environmental contact has been advocated through temporary contact isolation (ie, sleeping in a crate on a surface that can be disinfected or laundered rather than in a bed with people).14 We recommend contact precautions, social distancing, and good hygiene practices for households with MRSA-positive pets. When warranted, pets might need to be included in treatment regimens for the whole household.

Few interventions have been described for animals. Although rifampicin has been used as a systemic treatment for decolonisation of people and animals,147 rifampicin resistance in staphylococci after treatment148 and the drug’s potential for causing toxic effects150 restrict its use. We do not recommend routine use of rifampicin for decolonisation of pets. Chlorhexidine scrub or shampoo use at either 2% or 4% is effective for treatment of canine pyoderma;151 it might also be appropriate for decolonisation of pets. Present
Search strategy and selection criteria

We searched PubMed, ScienceDirect, and GoogleScholar for peer-reviewed articles published in English with no date restrictions. Search terms were combinations of “environment”, “household, house, home, spouse, or family”, “companion animal, dog, cat, canine, feline, pocket pet, pet animal, rodent, rat, mouse, rabbit, reptile, bird, lagomorph, horse, equine, sheep, goat, potbellied pig, hamster, guinea pig, or chinchilla”, and “MRSA, MSSA, Staphylococcus aureus, MRSP, coagulase-negative staphylococcus, CoNS, Staphylococcus pseudintermedius, or Staphylococcus schleiferi”.

The last search was done on June 5, 2012. Articles related only to food animals were not included, unless otherwise indicated (eg, related articles to food animals that might be kept as pets), because the role of food animal production (farms) in household transmission cycles was beyond the scope of the report. We examined references from identified articles and searched PubMed for authors of identified articles. We searched MD’s collection of more than 6000 publications with the same search terms. If necessary, we contacted corresponding authors for further clarification or additional information.

recommendations involve veterinary prescription of topical or systemic antimicrobial drugs on the basis of individual culture and sensitivity reports, avoiding use of β-lactam drugs. In the absence of definitive studies on decolonisation of pets, we recommend initial topical therapy, such as with chlorhexidine and tris-EDTA, to avoid overuse or misuse of oral antimicrobial drugs, particularly in view of concerns about selection for antimicrobial resistance. Intervention treatments for pets with veterinary staphylococci might be especially complex because of the high prevalence of multidrug resistance among isolates of meticillin-resistant S pseudintermedius. Owners and veterinarians might prefer to treat pets rather than remove them permanently from households for two reasons: many owners regard pets as part of the family, and the presence of pets has physical and mental health benefits. For cases of persistent colonisation of people or recurrent infections in either people or animals, we recommend that the patient’s physician and pet’s veterinarian (with the patient’s permission) jointly develop treatment regimens for concurrent person and pet decolonisation when circumstances warrant household-wide intervention.

Understanding transmission dynamics within households needs data from people, pets, and environmental surfaces. Future studies should sample pet species and assess other potential vectors. Variable durations of colonisation in man and pet hosts, and variable survival rates on environmental surfaces, might lead to a dynamic cycle of transmission, making longitudinal assessments essential. Sampling several anatomical locations for both people and animals is crucial, as is further research to assess decolonisation regimens and their indications for use in pets. In view of the potential roles of non-aureus staphylococci and other bacteria as sources of genes for antimicrobial resistance within the household, these bacterial species should be assessed.

Contributors

MFD searched the published work and wrote the first draft. All other authors contributed equally. SA1 and PB assisted with preparation of the tables. All authors assisted with interpretation of published work, edited the report, and assisted with revisions.

Conflicts of interest

We declare that we have no conflicts of interest.

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714 www.thelancet.com/infection Vol 12 September 2012


