

Pillows: The Forgotten Fomite

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Abstract

The pillow is an oft-overlooked link in a chain of cross-infection between patients. In this review, various links of that chain are examined. Evidence is presented to support the existence of viable transmission routes of infection between patients, pillows, and the ambient environment. Increased environmental contamination is found to increase infection rates and cleaning of the surface environment while the pillow interior remains infected is found to be ineffective in controlling contamination. The efficacy of barrier pillows in controlling contamination is considered.

Introduction

A growing body of observational evidence supports the intuitive view that the hospital environment plays a role in the transmission of nosocomial pathogens (Gerding et al, 2008, Weber et al 2010). Routes of transmission between patients (fomites) have been postulated as contamination and contact with contaminated surfaces, either directly or via healthcare workers (Chacko et al, 2003). It has been demonstrated that patients who occupy rooms whose previous occupant was infected with an organism have a significantly increased risk of themselves becoming infected with the same organism (Huang et al, 2006). This elevated risk is mitigated, but not eliminated, by enhanced cleaning regimens (Datta et al, 2011). One explanation for this incomplete eradication of risk is that surfaces remain colonised because of incomplete adherence to cleaning protocols (Nseir et al 2011). In this paper, we suggest an alternative or additional explanation: a significant reservoir for infection – the patient's pillow interior – is routinely escaping the cleaning regimen, and is continuously recolonizing the patient and the environment by multiple routes including direct contact and aerosolisation.

Does the patient environment become contaminated?

Oie et al (2003) found MRSA on the door-handles of 19% of rooms housing MRSA patients, compared with 7% of rooms housing non-MRSA patients. Boyce et al (1997) found that 42% of nurses contaminated their gloves with MRSA while performing tasks with no direct patient contact, but involving touching objects in the rooms of MRSA patients. French et al (2004) found 74% of sites surrounding the patient tested positive for MRSA. *Clostridium difficile* has been found to colonise surfaces including floors, hoppers, toilets, bedding, mops, scales, and furniture (Fekety et al, 1981). Other prevalent organisms include norovirus and actinobacter (Weber, 2010). Mattress interiors have been found to be contaminated with MRSA (Ndawula et al 1991). French et al (2004) sampled the environment surrounding

patients known to be MRSA positive, and found that 74% of sites samples tested positive for MRSA.

Does this include the Pillow?

Pillows present an ideal reservoir for pathogens, for a number of reasons. Typically, a hospital pillow will consist of an interior comprising either polyester fibre filling, or open-cell polyurethane foam. Given favourable conditions, either of these media present an ideal habitat for harbouring and culturing microorganisms (Woodcock et al, 2006). The porosity of the filling material provides a high surface area for colonisation, and a high capillarity for moisture retention. Favourable conditions are indeed provided by the patient: ideal temperature for incubation, humidity, and nutrition by body fluids and detritus, and inoculation by the nose, mouth, eyes, ears, and hair of the patient.

Lange et al (2014) found that 38% of hospital pillows were colonised with MRSA and coliforms, and concluded that disposable pillows might be an option worth exploring. Shik et al (2014) cut open nominally fluid-proof (stitched seam) pillows in a burns unit, and found that many were visibly contaminated with body fluids. Mottar et al (2006) observed a noticeable discrepancy in the weight of pillows in a burns centre. Examination revealed the seams as being a source of leakage, and multiple pathogens were isolated from the pillow interiors which correlated with patient infections. Lippmann et al (2014) sought reservoirs of infection to explain a large outbreak of *Klebsiella pneumoniae carbapenemase* (KPC) in Germany. They found that positioning pillows were internally contaminated, and remained so for at least 6 months.

The common practice of encasing the pillow in a waterproof cover does not solve the problem. Since the pillow must necessarily compress and expand to function as a pillow in accommodating the patient's head (or other part of the anatomy), it follows that air in the order of 2 litres must enter and exit the pillow in a few seconds when the pillow is unloaded or loaded, respectively. In the case of a simple waterproof pillow, this air will take the route of the opening flap, or, if the cover is stitched on, through the stitching holes of the cover seam. This latter scenario is especially troublesome. High concentrations of contaminants can be introduced to the pillow interior just inside the stitched seam (Dewhurst et al 2012). Here, they persist and incubate. Subsequently, contaminated air being driven from the pillow is forced at speed through the small stitching holes, creating an aerosol which may persist in the ambient air for many hours, and which has the capacity to recolonize not only the patient, or the subsequent patient, but also the patient environment (Kalogerakis 2005).

Filling materials within soft furnishings have been demonstrated to provide available nutrition to support the growth of bacteria (Jenkins et al, 2008). Polymer materials used for the filling provide an available source of carbon and nitrogen to support growth (Jenkins et al, 2005). Woodcock et al (2006) also found that 47 species of fungus including *Aspergillus fumigatus*, *Aureobasidium pullulans*, *Rhodotorula mucilaginosa* were endemic in pillows.

Is there a viable transmission route?

As early as 1979 (Reiss-Levy et al 1979) argued that pillows spread MRSA. Sherburn et al (2005) identified aerial release of bacteria from mattress interiors as body weight was applied. Movement on used cot mattresses, simulating that of an infant's head, significantly enhanced aerial release of naturally acquired bacteria from the polyurethane foams (total count data, $P = 0.008$; *Staphylococcus aureus*, $P = 0.004$) or from polyvinyl chloride covers (total count data, $P = 0.001$) (Sherburn et al, 2005). They found that levels of airborne bacteria were proportional to bacterial population levels inoculated into the inner pillow.

Shiomori et al (2002) quantified airborne MRSA before, during and after bedmaking. Air was sampled with an Andersen air sampler in the rooms of 13 inpatients with MRSA infection or colonization. Sampling of surfaces, including floors and bedsheets, was performed by stamp methods. Levels of MRSA colonisation were significantly increased after bedmaking and MRSA was detected on many surfaces. The results suggest that MRSA was recirculated in the air, especially after movement.

Boore et al (2014) conducted full-scale particle resuspension experiments in an environmental chamber, where volunteers performed a prescribed movement routine on an artificially seeded mattress. Human movements in bed, such as rolling from the prone to supine position, were found to resuspend settled particles, leading to elevations in airborne particle concentrations. Resuspension increased with the intensity of a movement, as characterized by surface vibrations, and decreased with repeated movement routines. Inhalation exposure ranged from 102 to 104 inhaled particles per million resuspended, demonstrating that a significant fraction of released particles can be inhaled by sleeping occupants.

When weight is removed from a pillow, air is drawn into the interior. In the proximity of a contaminated patient, being in close proximity to the mouth and nose, this air is especially likely to be contaminated also. As discussed above, the pillow provides a humid, warm, nutrient-rich environment for incubation and survival of microorganisms. Subsequently, when the pillow is squashed, an aerosol of contaminated droplet vapour is expelled from the interior, typically through the stitching holes of the pillow seams. This provides a means for recolonizing the environment over an indefinite future period, which is especially alarming considering the likely proximity of the pillow to subsequent patients' faces.

Does environmental contamination result in increased infection risk?

The most common nosocomial pathogens may well survive or persist on surfaces for months and can thereby be a continuous source of transmission if no regular preventive surface disinfection is performed. (Rutala et al, 2011). A review has been conducted (Rutala et al 2006) which determined that most gram-positive and many gram-negative bacteria will survive for many months on a dry hard surface. Numerous prospective trials have established a link between environmental colonisation by patients infected with a specific organism, and infection risk for subsequent patients with the same organism.

Tsakiridou et al (2014A) prospectively examined the rates of pneumonia due to *Acinetobacter baumannii* in an intensive care unit, and found that previous occupancy by a patient infected with *Acinetobacter baumannii* increased the risk 12-fold (95% CI 2.3-19.5). Huang et al (2006) found that admission to a room previously occupied by an MRSA-positive patient or a VRE-positive patient significantly increased the odds of acquisition for MRSA and VRE. Drees et al (2008) found that prior room contamination, whether measured via environmental cultures or prior room occupancy by VRE-colonized patients, was highly predictive of VRE acquisition. Factors increasing infection risk were VRE-colonized prior room occupant (HR, 3.1; 95% CI, 1.6-5.8), any VRE-colonized room occupants within the previous 2 weeks (HR, 2.5; 95% CI, 1.3-4.8), and previous positive room culture results (HR, 3.4; 95% CI, 1.2-9.6). Nseir et al (2011) found that Independent risk factors for ICU-acquired *A. baumannii* were prior occupant with *A. baumannii* (OR 4.2, 95% CI 2-8.8, $p < 0.001$).

A review of the topic conducted in 2013 (Otter et al 2013) considered evidence that contaminated surfaces contribute to the transmission of hospital pathogens, including studies modeling transmission routes, microbiologic studies, observational epidemiologic studies, intervention studies, and outbreak reports. The review concluded that contaminated surfaces contribute to transmission.

Is cleaning effective as a control mechanism?

Dancer et al (2009), implemented an enhanced cleaning regimen in 2 wards for 6 months in a prospective cross-over design. Improved cleaning resulted in 33% reduction in colony counts on hand-touch surfaces, and 27% reduction in MRSA infections. Microbial culture and genotyping positively matched MRSA strains between touch sites and patients.

Datta et al (2007) found that enhanced intensive care unit cleaning reduced MRSA and VRE transmission. and may reduce the risk of MRSA acquisition due to an MRSA-positive prior room occupant. Acquisition of MRSA and VRE was lowered from 3.0% to 1.5% for MRSA and from 3.0% to 2.2% for VRE ($P < .001$ for both). Patients in rooms previously occupied by MRSA carriers had an increased risk of acquisition during the baseline (3.9% vs 2.9%, $P = .03$) but not the intervention (1.5% vs 1.5%, $P = .79$) period. In contrast, patients in rooms previously occupied by VRE carriers had an

increased risk of acquisition during the baseline (4.5% vs 2.8%, $P = .001$) and intervention (3.5% vs 2.0%, $P < .001$) periods.

However, Andrada et al (2000), found that increased levels of cleaning in their setting did not result in reduced environmental colonisation, and in another study (Nseir et al, 2011) it was found that in spite of an aggressive terminal cleaning regimen, the probability of infection with an organism was significantly increased by the previous occupant's being infected with the same organism. They concluded that cleaning of the ICU rooms was probably not efficient in eradicating MDR *P. aeruginosa* and *A. baumannii*. supposing that compliance with cleaning protocol was not optimal.

An alternative explanation is that, in spite of cleaning, the immediate environment is immediately and repeatedly recolonized by contaminated aerosol expelled from the pillow.

Efficacy of 'barrier' pillows

Recently, a new category of medical device has been introduced: the barrier pillow [CE mark IE/CAO1/M/GM/0889]. So far, the only example of this available to hospitals is the Pneumapure pillow (Gabriel Scientific, Dublin). This pillow differs from standard occlusive pillows in that the seams are high frequency welded to make a seal, as opposed to stitched. The absence of stitching holes prevents ingress of contaminated air via the seams. Instead, air passes in and out of the pillow via a waterproof microbial filter, which has been tested to prevent ingress of bacteria, fungi, and viruses as small as 25 nm (Airmid Laboratories report, 2013). The pillow cover is cleaned and disinfected between patients according to standard mattress-cleaning protocols.

In a trial in a multi-centre hospital setting (Dewhurst et al, 2012), 100 new Pneumapure pillows were compared with 100 new nominally occlusive (stitched seams) pillows. After 3 months use, pillow interiors were sampled. Sixty percent of the standard pillows were internally contaminated, versus 0% of the Pneumapure pillows. An additional finding in this study was that, even where pillows were visibly damaged, soiled, and unserviceable, no system appeared to be in place for removing them from service. These findings together resulted in a Trust-wide introduction of the Pneumapure pillow in Liverpool and Broadgreen NHS trust, alongside a regular pillow audit regimen, and an accompanying reduction in MRSA and CDiff infections.

Conclusion

The infected pillow has several things in common with the infected human in terms of its capacity to spread infection. The human being coughs and sneezes, so generating a periodic aerosol of contaminated material into the ambient surroundings. The conventional coated pillow with stitched seams, as we have discussed, does something very similar: approximately 2 litres of humid air contaminated with microorganisms carried in droplet suspension are aerosolised into the environment, every time the patient's head is placed on the pillow. This has the capacity to recolonize the nearby environment, negating the effectiveness of any cleaning regimen. Also in common with the human vector is the tendency for pillows to circulate around hospitals, or even between hospitals (Turk *et al*, 2017).

McDonald and Arduino (2013) proposed an evidentiary hierarchy for the adoption of measures to combat infection control, beginning with (I) laboratory validation, rising through (II) demonstration of in-use bio-burden, (III) demonstration that the reduction is clinically relevant, (IV) demonstration of reduced pathogen transmission, and ultimately (V) demonstration of reduced infections. This hierarchy is necessarily difficult to climb: numerous patient and practice factors confound the relationship between environmental bioburden reductions and interruption of transmission. Correlating infection reductions to environmental bioburden reductions is even more challenging. However, in the case of introducing barrier pillows as a method of interrupting infection, there is a good prospect of progress towards level V of this ladder.

Laboratory validation shows the barrier pillows to be impervious to pathogens, where the standard pillows become quickly colonised by organisms with many months longevity. Samples taken from pillows in service demonstrate a

bioburden in standard pillow interiors which is complexly eliminated in barrier pillows. Environmental contamination is known to take place by aerosolisation via the pillow, and increased environmental contamination is known to result in increased transmission, and increased rates of infection.

Infected pillows are likely to be a significant vector for infection, and the introduction of a CE marked, validated barrier pillow, alongside an audit protocol which considers pillows and their serviceable condition, is a simple, inexpensive, and prudent measure.

References

Airmid Health Group Report ASCR092029 Pneumapure pillow, Airmid Health Group March 2013

Andrade D, Angerami EL, Padovani CR. Microbiological conditions of hospital beds before and after terminal cleaning

Boor BE, Spilak MP, Corsi RL, Novoselac A. Characterizing particle resuspension from mattresses: chamber study. *Indoor Air*. 2014 Jul 31. doi: 10.1111/ina.12148. [Epub ahead of print]

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 Sep;18(9):622-7.

Chacko L, Jose S, Isac A, Bhat KG. Survival of nosocomial bacteria on hospital fabrics. *Indian J Med Microbiol*. 2003 Oct-Dec;21(4):291.

Dancer SJ, White LF, Lamb J, Girvan EK, Robertson C. Measuring the effect of enhanced cleaning in a UK hospital: a prospective cross-over study. *BMC Med*. 2009 Jun 8;7:28. doi: 10.1186/1741-7015-7-28.

Datta R1, Platt R, Yokoe DS, Huang SS. Environmental cleaning intervention and risk of acquiring multidrug-resistant organisms from prior room occupants. *Arch Intern Med*. 2011 Mar 28;171(6):491-4. doi: 10.1001/archinternmed.2011.64.

Dewhursts et al The Pillow is a vector for infection, Proceedings IPS 2012

Drees M1, Snyderman DR, Schmid CH, Barefoot L, Hansjosten K, Vue PM, Cronin M, Nasraway SA, Golan Y. Prior environmental contamination increases the risk of acquisition of vancomycin-resistant enterococci. *Clin Infect Dis*. 2008 Mar 1;46(5):678-85. doi: 10.1086/527394.

Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr. Epidemiology of antibiotic-associated colitis; isolation of *Clostridium difficile* from the hospital environment. *Am J Med*. 1981 Apr;70(4):906-8.

French GL1, 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 May;57(1):31-7.

Gerding DN1, Muto CA, Owens RC Jr. Measures to control and prevent *Clostridium difficile* infection. *Clin Infect Dis*. 2008 Jan 15;46 Suppl 1:S43-9. doi: 10.1086/521861.

Huang SS1, Yokoe DS, Hinrichsen VL, Spurchise LS, Datta R, Miroshnik I, Platt R. Impact of routine intensive care unit surveillance cultures and resultant barrier precautions on hospital-wide methicillin-resistant *Staphylococcus aureus* bacteremia. *Clin Infect Dis*. 2006 Oct 15;43(8):971-8. Epub 2006 Sep 14.

Huang SS, Datta R, Platt R. Risk of acquiring antibiotic-resistant bacteria from prior room occupants. *Arch Intern Med*.

2006 Oct 9;166(18):1945-51.

Jenkins, R. O., & Sherburn, R. E. (2008). Used cot mattresses as potential reservoirs of bacterial infection: nutrient availability within polyurethane foam. *Journal of applied microbiology*, 104(2), 526-533.

Jenkins RO, Sherburn RE. Growth and survival of bacteria implicated in sudden infant death syndrome on cot mattress materials. *J Appl Microbiol*. 2005;99(3):573-9.

Kalogerakis, N., Indoor air quality - bioaerosol measurements in domestic and office premises. *Journal of Aerosol Science*, 2005. 36(5-6): p. 751-761.

Kramer A, Schwebke I, Kampf G. How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. *BMC Infect Dis*. 2006 Aug 16;6:130.

Lange V American Reusable Hospital Pillows - A Reservoir for Hospital Acquired Pathogens: The Importance of Adequate Decontamination *Journal of Infection Control* Volume 42, Issue 6, Supplement, Pages S34–S35, June 2014

Lippmann N Lübbert C, Kaiser T, Kaisers UX, Rodloff AC. Clinical epidemiology of *Klebsiella pneumoniae* carbapenemases *Lancet Infect Dis*. 2014 Apr;14(4):271-2. doi: 10.1016/S1473-3099(14)70705-4.

McDonald LC, Arduino M. Editorial commentary: climbing the evidentiary hierarchy for environmental infection control. *Clin Infect Dis*. 2013 Jan;56(1):36-9. doi: 10.1093/cid/cis845. Epub 2012 Oct 5.

Mottar, R., et al. "Pillow talk: examining pillow cores in a regional burn center." *American Journal of Infection Control* 34.5 (2006): E107-E108.

Ndawula EM, Brown L. Mattresses as reservoirs of epidemic methicillin-resistant *Staphylococcus aureus*. *Lancet*. 1991 Feb 23;337(8739):488.

Nseir S, Blazejewski C, Lubret R, Wallet F, Courcol R, Durocher A. Risk of acquiring multidrug-resistant Gram-negative bacilli from prior room occupants in the intensive care unit. *Clin Microbiol Infect*. 2011 Aug;17(8):1201-8. doi: 10.1111/j.1469-0691.2010.03420.x. Epub 2010 Dec 13.

Oie S, Hosokawa I, Kamiya A. Contamination of room door handles by methicillin-sensitive/methicillin-resistant *Staphylococcus aureus*. *J Hosp Infect*. 2002 Jun;51(2):140-3.

Otter JA, Yezli S, Salkeld JA, French GL. Evidence that contaminated surfaces contribute to the transmission of hospital pathogens and an overview of strategies to address contaminated surfaces in hospital settings. *Am J Infect Control*. 2013 May;41(5 Suppl):S6-11. doi: 10.1016/j.ajic.2012.12.004.

Otter JA, Yezli S, French GL. The role played by contaminated surfaces in the transmission of nosocomial pathogens. *Infect Control Hosp Epidemiol*. 2011 Jul;32(7):687-99. doi: 10.1086/660363

Reiss-Levy E, McAllister E Pillows spread methicillin-resistant staphylococci. *Med J Aust*. 1979 Feb 10;1(3):92.

Rutala WA, Weber DJ. Surface disinfection: should we do it? *J Hosp Infect*. 2001 Aug;48 Suppl A:S64-8.

Sherburn RE, Jenkins RO. Cot mattresses as reservoirs of potentially harmful bacteria and the sudden infant death syndrome. *FEMS Immunol Med Microbiol*. 2004 Sep 1;42(1):76-84.

Sherburn RE, Jenkins RO. Aerial release of bacteria from cot mattress materials and the sudden infant death

syndrome. J Appl Microbiol. 2005;98(2):293-8.

Shik N.F., , Ford S., Thompson R., , Pena M., , Luchi M., The Heat Is On: Control of Community-Acquired MRSA in a Burn Center American Journal of Infection Control June 2006Volume 34, Issue 5, Page E100, ;

Shiomori T, Miyamoto H, Makishima K, Yoshida M, Fujiyoshi T, Udaka T, Inaba T, Hiraki N. Evaluation of bedmaking-related airborne and surface methicillin-resistant *Staphylococcus aureus* contamination. J Hosp Infect. 2002 Jan;50(1):30-5

Tsakiridou E, Makris D, Daniil Z, Manoulakas E, Chatzipantazi V, Vlachos O, Xidopoulos G, Charalampidou O, Zakynthinos E. *Acinetobacter baumannii* infection in prior ICU bed occupants is an independent risk factor for subsequent cases of ventilator-associated pneumonia. Biomed Res Int. 2014;2014:193516. doi: 10.1155/2014/193516. Epub 2014 Jul 2.

Turk S, Christersson J, Rööp T Comparison of bacterial loads of two types of hospital pillows: Perspectives of improving hospital hygiene standards. Canadian Journal of Infection Control (in press) 2017

Weber D, Rutala W, Miller M, Huslage K, Sickbert-Bennet E. Role of hospital surfaces in the transmission of emerging healthcare-associated pathogens; norovirus, *Clostridium difficile*, and *Acinetobacter* species. American Journal of Infection Control, 2010 Jun; 35 (5 Suppl 1) S25-33.

William A. Rutala, PhD, MPH; David J. Weber, MD, MPH Are Room Decontamination Units Needed to Prevent Transmission of Environmental Pathogens? Infection Control and Hospital Epidemiology, Vol. 32, No. 8 (August 2011), pp. 743-747

Woodcock, AA., Steel, N., Moore, CB., et al. Fungal contamination of bedding North West Lung Centre, Wythenshawe Hospital and University of Manchester, Manchester, UK. Allergy, 2006 Jan;61(1):140-2

Youngster I, Berkovitch M, Heyman E, Lazarovitch Z, Goldman M. The stethoscope as a vector of infectious diseases in the paediatric division. Acta Paediatr. 2008 Sep;97(9):1253-5. doi: 10.1111/j.1651-2227.2008.00906.x. Epub 2008 Jun 12.

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