JOURNAL OF PURE AND APPLIED MICROBIOLOGY, March 2018. Vol. **12**(1), p. 181-187

The Domestic Kitchen – The 'Front Line in the Battle Against Foodborne Disease'

Karim Hassan[1]* and Marwan ElBagoury[2]

[1]School of Pharmacy and Pharmaceutical Sciences, The University of Manchester, UK.
[2]UniversityUlm - Medizinische Fakultät, Ulm, Deutschland.

http://dx.doi.org/10.22207/JPAM.12.1.23

(Received: 15 January 2018; accepted: 21 February 2018)

Foodborne disease is linked to noteworthy morbidity and mortality all over the world, and is thus a serious public health issue. In spite of the many recorded cases, it is generally accepted that actual figures may be orders of magnitude higher than those recorded due to significant under-reporting amongst other factors. This article sheds the light on bacterial contamination in domestic kitchen, and some of the commonly used biocides.

Keywords: Domestic Kitchen, Foodborne disease**,** *Campylobacter jejuni*, *Salmonella* spp., Sodium hypochlorite, Chloroxylenol, Benzalkonium chloride.

Foodborne disease is becoming a serious public health issue. *Campylobacter jejuni* and *Salmonella* spp. are the main culprits responsible for a significant proportion of reported cases of foodborne disease in the UK[1-4]. This is explained by the close association of these species with chicken, a common component of the UK diet. Numerous surveys have been conducted to assess the extent of contamination of raw poultry sold in the UK and they have revealed that up to 30% is contaminated with salmonellae and up to 90% with campylobacters[3]. *Salmonella* spp. and *Campylobacter* spp. are widely recognised as the most common pathogens isolated in human cases of bacterial gastroenteritis[4, 5] although it has been reported that *Campylobacter* is more often implicated in gastrointestinal infection than *Salmonella*[6]. *Campylobacter* is also considered to be the leading bacterial cause of diarrhoeal disease/ infectious intestinal disease (IID) in the developed world[5].

* To whom all correspondence should be addressed.
E-mail: karimhassan66@outlook.com

In addition to *Salmonella* and *Campylobacter*, it has been reported that *Shigella* and Shiga toxin and *Escherichia coli* (STEC) O157 significantly contribute to the incidence of foodborne disease in the United States[7]. Other foodborne pathogens include *Staphylococcus aureus*, rotavirus and hepatitis A virus[8].

Foodborne outbreaks of *Listeria monocytogenes*, responsible for septicaemia and meningoencephalitis in the immunocompromised and the elderly, have been reported[9]. *L. monocytogenes* is common in the environment and has caused several cases of hospital-acquired/ nosocomial listeriosis, including in neonates, in the UK. Nosocomial outbreaks of *Salmonella* infection, particulary *Salmonella enteritidis*, have also been recorded[10]. The domestic kitchen is increasingly being identified as the most important area associated with the cross contamination of foodborne pathogens in the home, in addition to the harbouring and transferring of infection[11, 12]. This is despite estimates of the proportion of food poisoning cases initiating in the household ranging from 12-17% to 50-80%[13]. Contamination of several foods, particularly raw foods, consumed in the domestic kitchen with naturally occurring

pathogenic microorganisms is inevitable[4, 14]. Accordingly, cross contamination of foodborne pathogens has been recognised as the chief hazard in the household, and can be either direct or indirect[15]. Direct cross contamination includes the transfer of microorganisms directly from raw food, whereas the indirect route uses a vehicle such as kitchen cloths and sponges, hands, utensils and surfaces as an intermediate. The latter route has been shown to be more common. The aim of this article is to explore microbial contamination of the domestic kitchen, in addition to reviewing some of the commonly used biocides.

Previous studies of bacterial contamination in the domestic kitchen

Bacterial contamination in the domestic environment was first comprehensively investigated in 1978 by Finch *et al*[16]. In this study, samples were isolated from various sites in the kitchen, bathroom, living room, toilet and hall of 21 homes and the bacterial flora therein examined. Several studies followed assessing the levels of microbial contamination in the domestic kitchen and the home environment[2, 11, 13, 17, 18] highlighting the growing significance of the home, and the kitchen in particular, as locations for harbouring pathogens responsible for foodborne disease.

Scott *et al*.'s study[17] study was similar to Finch *et al*[16] but on a much larger scale, looking at microbial contamination in 251 domestic houses by sampling 60 sites in the bathroom, toilet and kitchen. Speirs *et al*[18] presented findings from the investigation of a wider range of sites (76 in total) in 13 domestic kitchens together with focusing on specific sites with potential for cross-contamination in a further 33 kitchens. Josephson *et al*[2] looked at the effectiveness of different cleaning regimes through a two year study that involved the quantification of specific bacterial pathogens and indicator organisms in 10 household kitchens in the United States in the presence, both 'casual' and 'targeted', and absence of disinfectant cleaner use. Rusin *et al*[11] examined fourteen sites in the kitchen and bathroom of 15 households over an extended period of time to determine which of these had the highest levels of contamination with faecal coliform, coliform and heterotrophic plate count (HPC) bacteria. Their study also included an investigation of the effectiveness of various cleaning and disinfection regimes using hypochlorite cleaners. Finally, Haysom[13] recognised that previous kitchen studies only looked at samples taken from one point in time and decided to investigate the change of microbial load in domestic kitchens over a 24-hour period.

The above studies provide substantial evidence confirming the presence of viable microorganisms across various sites in domestic kitchens. However, the type of organism together with its likelihood of contaminating food very much determine how significant this microbial contamination is in relation to human health[19].

Sources of entry

A variety of routes exist by which potential pathogens can enter domestic kitchens[4]. Raw foods, as mentioned previously, are an important route and include meat and poultry, fish and shellfish, raw eggs and fruits and vegetables[8]. It is widely recognised that poultry is a significant vehicle for the transmission of *Campylobacter* and *Salmonella* infections for example[4, 6, 20]. A wide range of non-food activities take place in the domestic kitchen[4, 13] that may contribute to the associated microbiological hazards by providing an additional route or acting as vehicles for microbial entry. Indeed, domestic kitchens have also been found to be used for repairing bicycles, washing pets' water bottles, combing children's hair, gardening, motor vehicle maintenance and even more surprisingly breeding of chickens. Haysom[13] also points out that children playing in the kitchen can also contribute to the microbial load observed. Additionally, domestic pets, such as cats and dogs, have also been recognised as sources of foodborne pathogens, such as *Salmonella* spp. and other eneteropathogens[8]. Since cats and dogs may also act as reservoirs for *Campylobacter* spp. and over half of the English-speaking world own cats and dogs, with 14 million of these pets in the United Kingdom alone; they cannot be excluded as potential sources of infection. Another source of foodborne pathogens may come from the human occupants of the household themselves who may serve as symptomatic or non-symptomatic carriers.

It is therefore clear that the domestic kitchen is a multi-functional setting[4] and, as a result, both food and non-food activities must be taken into account when considering sources of contamination.

Important sites in the kitchen and types of bacteria isolated

Several studies investigating bacterial contamination in the domestic environment have confirmed the presence of pathogenic and non-pathogenic microorganisms on the majority of household surfaces[4]. Surprisingly, Rusin *et al*[11] found heavier bacterial contamination in the kitchen than the bathroom, with the toilet seat exhibiting the lowest level of contamination across all sites sampled. This study found the following sites in the domestic kitchen to be the most heavily contaminated with faecal coliforms, coliforms and heterotrophic plate count (HPC) bacteria: the sponge, dishcloth, kitchen sink drain, kitchen faucet handle(s) and the cutting board. The refrigerator handle, kitchen counter top and the floor in front of the kitchen sink were also found to be contaminated, although to a lesser extent.

A significant finding in the study presented by Finch *et al*[16] was the isolation of a large number of Enterobacteriaceae, such as *Klebsiella* spp. and *Escherichia coli*, from kitchen sinks. The authors noted the speed with which colonization of kitchen sinks with Enterobacteriaceae takes place, with large numbers of E. coli being isolated within one week of occupation of new, previously unoccupied houses. *Staphylococcus aureus* was isolated from almost half of the 47 tea towels/cloths tested, albeit in small numbers. Additionally, the authors found *Bacillus* spp. and *Micrococcus* spp. in all sites of the home that were sampled and the prevalence of Gram-negative bacteria in wet areas was highlighted. It was concluded that a wide variety of bacterial species appeared to be supported by the normal domestic environment.

A study by Scott *et al*[17] expressed concern regarding the prevalence of enteric microorganisms in the predominantly wet areas of the domestic kitchen such as U-tubes, sink surfaces, draining boards and wet cloths, which were associated with high microbial counts. The authors suggested that these sites may harbour enterobacteria and encourage their proliferation by acting as reservoirs. This study also recognised dishcloths and other wet cleaning utensils as disseminators of contamination in the kitchen due to their consistently high microbial counts which included large numbers of enterobacteria.

Speirs *et al*[18] echoed these findings reporting that the wet areas around the sink, such as the surface, plughole and draining board, together with cloths used for wiping surfaces and/or drying utensils, such as dishcloths, sponges and tea towels, were associated with the highest microbial loads. They found the sink to be predominantly contaminated with Gram-negative rods and cloths with Gram-positive cocci. *Enterobacter cloacae* and *Bacillus subtilis* were isolated from over half of the 46 kitchens investigated by the authors. *Enterobacter spp.*, *Klebsiella pneumonia* and *Escherichia coli* were among the enterobacteria isolated in the study, with *Pseudomonas aeruginosa* being identified as the predominant pseudomonad isolated. *Staphylococcus* spp. and *Micrococcus* spp. were found in all 46 kitchens examined, being isolated from many of the sampled sites. The authors remarked that although bacteria implicated in foodborne disease were not often discovered, individual isolates of *Listeria monocytogenes*, *Bacillus cereus* and *Yersinia enterocolitica* were recorded.

Josephson *et al*[2] identified the kitchen sink and sponge as the two sites most consistently contaminated and attributed this to the high level of moisture associated with these sites. Isolates included *Staphylococcus* and *Pseudomonas*, albeit at low concentrations, and 'surprisingly high' counts of HPC and total and faecal coliform bacteria were recorded. Pseudomonads were almost exclusively found in the sink and sponge samples and it was suggested that this was due to their high affinity for moisture. Furthermore, *Salmonella* and *Campylobacter* were only identified on one and two occasions respectively.

These studies show that the type and density of bacterial contamination is dependent on the physical nature of the site sampled and that wet to moist areas are more likely to be contaminated due to the preferential survival and proliferation of microorganisms in these conditions[4].

Bacterial resistance

Resistance has been defined as 'the relative insusceptibility of a microorganism to a particular treatment under a particular set of conditions'[21]. Non-antibiotic treatments include antiseptics, disinfectants and preservatives, generally termed 'biocides'[22,23]. The minimum concentration needed to exert a definable effect, in this case growth inhibition, on a cell population is generally used

to quantify bacterial resistance in relation to antimicrobial agents[21]. Two major mechanisms have been used to describe resistance to biocides: Intrinsic and acquired[22, 24]. Bacterial response to biocides will vary depending on the nature of the biocide together with that of the specific organism. Generally speaking, Gram-negative bacteria such as members of the Enterobacteriaceae family tend to be more resistant to biocides than Gram-positive bacteria such as staphylococci and enterococci.

Intrinsic resistance

Intrinsic resistance refers to the innate (natural) ability of a bacterial cell to avoid the action of a biocide via chromosomally controlled mechanisms or properties[22]. This 'intrinsic insusceptibility' is as a result of the failure of the biocide to achieve sufficiently high concentrations at the target site to exert its lethal effect[24]. This usually involves the outer cell layers, which the biocide must traverse in order to reach the target site(s) often found within the cell, acting as a permeability barrier[22]. This results in limited access of the biocide into the cell. Gram-negative bacteria for example, possess outer layers that by nature result in reduced uptake of biocides thus explaining their reduced susceptibility relative to staphylococci. Cell wall composition is therefore important in this respect. Alternatively, intrinsic resistance may be as a result of enzymatic biodegradation (or inactivation) of the biocide compound e.g. via ²-lactamases[25], however this is less commonly observed[22]. Additionally, exposure to certain environmental conditions can trigger phenotypic (or physiological) adaptation resulting in reduced susceptibility.

Acquired resistance

Acquired resistance to biocides arises from changes in the genetic composition within a bacterial cell as a result of either mutation, which can occur following the exposure of bacteria to progressively increasing concentrations of biocide, or via the acquisition of genetic material such as plasmids or transposons from other cells[22]. This type of resistance may occur through modified targets to which the biocides bind, expression of enzymes that inactivate antimicrobial agents, or the expression of efflux pumps which result in limited access of the biocide to its target[25]. The role of both, drug-specific and multidrug, efflux mechanisms as important determinants of both intrinsic and acquired resistance has long been recognised[26]. Generally speaking, resistance arising from mutation or genetic exchange is irreversible[25].

Biofilms – the predominant lifestyle

A plethora of research into biofilms over the last few decades has resulted in the evolution of our definition of biofilm. Perhaps the most complete definition was one proposed by Donlan and Costerton[27] in 2002 who defined biofilm as "a microbially derived sessile community characterized by cells that are irreversibly attached to a substratum or interface or to each other, are embedded in a matrix of extracellular polymeric substances that they have produced, and exhibit an altered phenotype with respect to growth rate and gene transcription". It is now widely acknowledged as a result of the overwhelming number of studies on the subject that microorganisms in nature predominantly grow, not as free-swimming, planktonic cells, but in association with surfaces[28]. In addition to bacteria; fungi, yeasts, algae, protozoa and viruses have also been shown to form biofilms, and this contributes to the heterogeneity in species composition often observed in biofilm communities[29]. However, in relation to colonisation of surfaces, bacteria are the most extensively studied[28].

Intriguingly, biofilm-associated organisms differ physiologically from their corresponding planktonic counterparts[28, 30, 31]. Their behaviour has been likened to that of multi-cellular organisms in that they are a cooperative functional consortia of cells. More intriguing is the profound recalcitrance of bacteria to biocide treatments conferred by growth in this mode[31, 32]. In fact, reduced susceptibilities of 10-1,000- fold relative to corresponding planktonic (free-floating) counterparts have been reported[29, 32-34].

Although of some benefit to human beings, biofilms are notorious for their associated problems[29]. Not only are they associated with oral disease such as periodontitis, infections relating to indwelling medical devices and industrial biofouling, but they also present a significant public health issue with regards to food hygiene. For example, the persistence and maturity of biofilms in domestic kitchen sink drains has been recognised and, being closely situated to food preparation areas, this presents a risk of contamination of food with potentially pathogenic bacteria[35]. Since

wet environments encourage the adherence of microorganisms to a surface[31], the kitchen is indeed an ideal environment for biofilm development.

Biocides

Chemical agents with antiseptic, disinfectant and/or preservative activity fall under the umbrella term 'biocides'[23]. Biocides are defined as "molecules, generally of synthetic or semisynthetic origin, that, above certain critical concentrations and under defined conditions, will kill living cells within specified times"[21]. It is important to recognise the difference between antibiotics and biocides. Antibiotics work in conjunction with host defence mechanisms to eliminate bacteria, generally acting on a single target to achieve growth inhibition[36]. Conversely, biocides may act on one or multiple cellular targets with the aim of rapid killing. Broad classifications such as oxidative, membrane active etc. have been assigned to biocides as a result[21]. Potential bacterial target sites for biocides include the cell wall or outer membrane, cytoplasmic membrane, DNA and RNA amongst other cytosolic components [31]. Factors that may influence biocide activity include concentration, period of contact, pH, temperature, presence of organic matter and the microorganism's nature, number, location and condition[23,24]. It is worthy to note that there is currently a severe lack of evidence to suggest that the exposure of bacterial cultures to sublethal concentrations of biocide is related to the emergence of antibiotic resistance[21].

Sodium hypochlorite (chlorine releasing agent - CRA)

Sodium hypochlorite, found in household bleach, is commonly used in solution for hard-surface disinfection[37]. It is a commercially available chlorine releasing agent and is a salt of the hypochlorite ion (OCl^-)[38]. Although this biocide has broad antimicrobial activity and has been extensively studied, its full mechanism of action has not yet been confirmed[37, 38]. It is believed to terminate cellular activity of proteins due to its strong oxidising properties. Undissociated hypochlorous acid (HOCl) is thought to be the active moiety which can undergo dissociation into the less microbicidal OCl^- with increased pH[38]. Additionally, the concentration of available chlorine also dictates the effectiveness of sodium hypochlorite[39]. Rutala and Weber[38] reported that concentrations of 1-15% sodium hypochlorite are typically used in commercial products for home or hospital use, with household bleach generally containing concentrations of 4-6% of aqueaous solution.

Chloroxylenol (4-chloro-3,5-dimethylphenol)

Chloroxylenol is among the oldest antimicrobial chemical entities being used today[40]. Despite this, little is known about its mechanism of action [37]. It is a halophenol and is thought to exert its effect on microbial membranes due to its phenolic nature. Phenolic disinfectants have been shown to be effective at significantly reducing bacterial numbers, in particular numbers of enterobacteria, at wet and dry sites in the domestic kitchen under 'in use' conditions[41].

Benzalkonium chloride

Benzalkonium chloride, a quaternary ammonium compound (QAC) introduced in 1935, was the first in its class to be commercially available[42]. It is a membrane-active cationic agent that has been used in health care e.g. preoperative disinfection of unbroken skin, and for disinfection of hard-surfaces[37]. It exerts its action via disruption of the cytoplasmic membrane of the bacterial cell resulting in loss of structural integrity among other detrimental effects. The C-chain length of benzalkonium chloride has been shown to influence the extent of bacterial resistance in a biofilm[31]. An increase in the C-chain length of benzalkonium chloride from C12 to C18 was shown to enhance the recalcitrance of biofilm-associated *P. aeruginosa* to the molecule as a result of increased hydrophobicity hindering its penetration through the hydrophilic biofilm matrix and hence, its bactericidal efficacy.

REFERENCES

1. Cogan, T., *et al*., *Achieving hygiene in the domestic kitchen: the effectiveness of commonly used cleaning procedures. Journal of Applied Microbiology,* 2002. **92**(5): p. 885-892.
2. Josephson, K., J. Rubino, and I. Pepper, Characterization and quantification of bacterial pathogens and indicator organisms inhousehold kitchens with and without the use of a disinfectant cleaner. *Journal of Applied Microbiology,* 1997. **83**(6): p. 737-750.
3. Meredith, L., R. Lewis, and M. Haslum, Contributory factors to the spread of contamination in a model kitchen. *British Food Journal,* 2001. **103**(1): p. 23-36.

4. Redmond, E.C. and C.J. Griffith, The importance of hygiene in the domestic kitchen: implications for preparation and storage of food and infant formula. *Perspectives in Public Health*, 2009. **129**(2): p. 69-76.
5. Rodrigues, L.C.C., J. M.; Wheeler, J. G.; Sethi, D.; Wall, P. G.; Cumberland, P.; Tompkins, D. S.; Hudson, M. J.; Roberts, J. A.; Roderick, P. J., The study of infectious intestinal disease in England: risk factors for cases of infectious intestinal disease with Campylobacter jejuni infection. *Epidemiology and Infection,* 2001. **127(2)**: p. 185-193.
6. Scott, E., Foodborne disease and other hygiene issues in the home. *Journal of Applied Microbiology,* 1996. **80**(1): p. 5-9.
7. Nyachuba, D.G., Foodborne illness: is it on the rise? *Nutrition Reviews,* 2010. **68**(5): p. 257-269.
8. Scott, E., Food safety and foodborne disease in 21st century homes. *The Canadian Journal of Infectious Diseases & Medical Microbiology*, 2003. **14(5)**: p. 277-280.
9. Graham, J.C., *et al*., Hospital-acquired listeriosis. *Journal of Hospital Infection,* 2002. **51**(2): p. 136-139.
10. Dryden, M.S., *et al*., Asymptomatic foodhandlers as the source of nosocomial salmonellosis. *Journal of Hospital Infection,* 1994. **28**(3): p. 195-208.
11. Rusin, P., P. Orosz-Coughlin, and C. Gerba, Reduction of faecal coliform, coliform and heterotrophic plate count bacteria in the household kitchen and bathroom by disinfection with hypochlorite cleaners. *Journal of Applied Microbiology,* 1998. **85**(5): p. 819-828.
12. Sharma, M., J. Eastridge, and C. Mudd, Effective household disinfection methods of kitchen sponges. *Food Control,* 2009. **20**(3): p. 310-313.
13. Haysom, I.W.S., A.K. , Bacterial contamination of domestic kitchens over a 24-hour period. *British Food Journal,* 2005. **107**(7): p. 453-466.
14. Mattick, K., *et al*., The microbiological quality of washing-up water and the environment in domestic and commercial kitchens. *Journal of Applied Microbiology,* 2003. **94**(5): p. 842-848.
15. Sharp, K. and H. Walker, A microbiological survey of communal kitchens used by undergraduate students. *International Journal of Consumer Studies,* 2003. **27**(1): p. 11-16.
16. Finch, J.E., J. Prince, and M. Hawksworth, A Bacteriological Survey of the Domestic Environment. *Journal of Applied Microbiology,* 1978. **45**(3): p. 357-364.
17. Scott, E., S.F. Bloomfield, and C.G. Barlow, An investigation of microbial contamination in the home. *The Journal of Hygiene,* 1982. **89(2)**: p. 279-293.
18. Speirs, J.P., A. Anderton, and J.G. Anderson, A study of the microbial content of the domestic kitchen. *International Journal of Environmental Health Research,* 1995. **5**(2): p. 109-122.
19. Hilton, A.C. and E. Austin, The kitchen dishcloth as a source of and vehicle for foodborne pathogens in a domestic setting. *International Journal of Environmental Health Research,* 2000. **10**(3): p. 257-261.
20. Humphrey, T.J., *et al*., Campylobacter spp. in the kitchen: spread and persistence. *Journal of Applied Microbiology,* 2001. **90**(S6): p. 115S-120S.
21. Gilbert, P. and A.J. McBain, Potential Impact of Increased Use of Biocides in Consumer Products on Prevalence of Antibiotic Resistance. *Clinical Microbiology Reviews,* 2003. **16**(2): p. 189-208.
22. Russell, A.D., Mechanisms of bacterial resistance to biocides. International Biodeterioration & *Biodegradation,* 1995. **36**(3–4): p. 247-265.
23. Russell, A.D., Biocide use and antibiotic resistance: the relevance of laboratory findings to clinical and environmental situations. *The Lancet Infectious Diseases,* 2003. **3**(12): p. 794-803.
24. Russell, A.D., Bacterial resistance to disinfectants: present knowledge and future problems. Journal of Hospital Infection, 1999. **43, Supplement 1**(0): p. S57-S68.
25. Patel, R. Biofilms and Antimicrobial Resistance. *Clinical Orthopaedics and Related Research* 2005; **437**, 41-47.
26. Poole, K. Efflux pumps as antimicrobial resistance mechanisms. *Annals of Medicine* 2007; **39**, 162-176.
27. Donlan, R.M.C.J.W. Bioûlms: Survival Mechanisms of Clinically Relevant Microorganisms. *Clinical Microbiology Reviews,* 2002; **15**(2), 167-193.
28. Lindsay, D. and A. von Holy, Bacterial biofilms within the clinical setting: what healthcare professionals should know. *Journal of Hospital Infection,* 2006. **64**(4): p. 313-325.
29. Gilbert, P. and McBain, A.J. Biofilms: Their impact on health and their recalcitrance toward biocides. *American Journal of Infection Control,* 2001; **29**, 252-255.
30. Donlan, R. (2002) Biofilms: microbial life on surfaces. *Emerging Infectious Diseases* **8**.
31. Bridier, A., *et al*., Resistance of bacterial biofilms to disinfectants: a review. *Biofouling,* 2011. **27**(9): p. 1017-1032.
32. Davies, D., Understanding biofilm resistance to antibacterial agents. *Nat Rev Drug Discov,* 2003. **2**(2): p. 114-122.
33. Anderson, G.G. and G.A. O'Toole, Innate and Induced Resistance Mechanisms of Bacterial

Biofilms, T. Romeo, Editor. 2008, Springer Berlin Heidelberg. p. 85-105.
34. Smith, K. and I.S. Hunter, Efficacy of common hospital biocides with biofilms of multi-drug resistant clinical isolates. *Journal of Medical Microbiology,* 2008. **57**(8): p. 966-973.
35. McBain, A.J., *et al*., Microbial Characterization of Biofilms in Domestic Drains and the Establishment of Stable Biofilm Microcosms. *Applied and Environmental Microbiology,* 2003. **69**(1): p. 177-185.
36. Bloomfield, S.F., Significance of biocide usage and antimicrobial resistance in domiciliary environments. *Journal of Applied Microbiology,* 2002. **92**: p. 144S-157S.
37. McDonnell, G. and A.D. Russell, Antiseptics and Disinfectants: Activity, Action, and Resistance. *Clinical Microbiology Reviews,* 1999. **12**(1): p. 147-179.
38. Rutala, W.A. and D.J. Weber, Uses of inorganic hypochlorite (bleach) in health-care facilities. *Clinical Microbiology Reviews,* 1997. **10**(4): p. 597-610.
39. Fukuzaki, S., Mechanisms of actions of sodium hypochlorite in cleaning and disinfection processes. *Biocontrol Science,* 2006. **11**(4): p. 147-57.
40. Ascenzi, J.M., Chloroxylenol: An Old-New Antimicrobial. Handbook of disinfectants and antiseptics, 1996.
41. Scott, E., S.F. Bloomfield, and C.G. Barlow, Evaluation of disinfectants in the domestic environment under 'in use' conditions. *Epidemiology and Infection,* 1984. **92(2)**: p. 193-203.
42. Rutala, W.A., APIC guideline for selection and use of disinfectants. *American Journal of Infection Control,* 1996. **24**(4): p. 313-342.