

Non-respiratory infections – specific considerations in care homes

Victoria Ewan¹ and Adam Gordon²

¹Institute for Ageing and Health, Newcastle University, Newcastle-upon-Tyne, UK

²Division of Rehabilitation and Ageing, University of Nottingham, Nottingham, UK.

Correspondence to:

Dr Victoria Ewan,
Institute for Ageing and Health,
Newcastle University
Campus for Ageing and Vitality
Newcastle upon Tyne
NE4 5PL

Email: victoria.ewan@newcastle.ac.uk

Tel: 0191 248 1301

This paper has been accepted for publication and will appear in a revised form, subsequent to peer review and/or editorial input by Cambridge University Press, in **Reviews in Clinical Gerontology** published by Cambridge University Press. Cambridge University Press hold the copyright.

Summary

This review provides an update on current evidence surrounding the epidemiology, treatment and prevention of non-respiratory infections in care homes. It covers urinary tract infection (UTI), methicillin-resistant staphylococcus aureus (MRSA), decubitus ulcers, scabies, tinea infections and viral and bacterial gastroenteritis. The care home sector provides a unique ecological niche for infections, housing frail older people with multiple co-morbidities and frequent contact with healthcare services in a semi-closed environment. This leads to differences in the diagnosis and management of infections – particularly of outbreaks – when compared with community-dwelling counterparts. It is essential that care home staff play a role in the early recognition, isolation and treatment of infections but they are often not trained as healthcare professionals – this presents a challenge to systematised response. Effective interface between care homes, public health and infection control services are essential to the delivery of care, yet it is not clear how most-effectively to structure such links.

Keywords: Nursing homes, residential facilities, Homes for the Aged, Infection, Infection Control

Introduction

Older people living in care homes are more dependent, have more co-morbidities and use health services more than age-matched community-dwelling counterparts^{1, 2}. As a consequence, they are more vulnerable to infections and more likely to suffer significant sequelae³. Their living facilities may provide both opportunities for transmission of infection and the possibility of early identification and treatment⁴.

Pneumonia and influenza constitute the most important infections in care home residents in terms of incidence, morbidity and mortality, and we have discussed these in a previous article⁵. We move on here to discuss important non-respiratory infections in this cohort. As with our previous article, we focus on considerations in the classification, epidemiology, diagnosis and management of these which are particular, or particularly relevant, to the care home setting.

Search Strategy

We searched Medline (1950-present) to week 4 January 2010. We used the search terms “nursing homes” OR “homes for the aged” OR “residential care facilities” AND “urinary tract infections” OR “gastroenteritis” OR “colitis” OR “soft tissue infections” OR “skin infections”. We found 294 articles, of which 7 were duplicate entries, 20 were not relevant to care homes and 2 were about children. This left 265 articles which were read in full. Relevant publications from the reference lists of these articles were also reviewed.

What is a care home?

A care home is defined in the United Kingdom (UK) as “an establishment [which] provides accommodation, together with nursing or personal care, for persons who are or have been ill, who have or have had a mental disorder, who are disabled or infirm, or are or have been dependent on alcohol or drugs”⁶. All care homes provide support with activities of daily living but are classified as either residential care homes or care homes with nursing depending on whether they provide dedicated 24-hour professional nursing care. Considering the whole sector, only 8.6% of residents

are <70, with 76% overall requiring assistance with their mobility or being immobile, 78% having at least one form of mental impairment and 71% suffering incontinence².

Although most developed countries have a care home equivalent, the model of separate residential and nursing care is far from universal^{7, 8}. Further, models of care differ between countries, ranging from the highly medicalised, where medical support is provided in hospital-style surroundings, to the highly socialised, where a primarily residential ethos is adopted, such as in the UK.

This residential ethos presents a number of challenges regarding infection control in the UK. Firstly homes are small, with a mean number of residents of 36 in 2009⁹, which facilitates containment of infections but makes a structured response in the face of outbreaks difficult¹⁰. Secondly, most homes are decorated with carpets and soft furnishings which are difficult to keep clean¹¹. Finally, the residential ethos of homes makes hospital-style isolation procedures difficult.

Not all aspects of the literature on infections in long-term care can readily be translated across national boundaries. We attempt here to present findings of sufficient generalisability to be useful in most contexts.

What is the prevalence of infection in care homes?

Data on the prevalence of infections in care homes come from the USA^{3, 12, 13}, Norway^{14, 15}, Germany¹⁶, the Netherlands¹⁷ and Belgium¹⁸. These studies adopted differing designs, ranging from point-prevalence studies to rolling surveillance programmes, identified and reported infections in different ways and used different classification systems for infection. All were compromised by the fact that acute infections resulting in hospital admission, such as viral gastroenteritis, are readily identified whereas endemic but unobtrusive infections, such as tinea corporis, are not. Regardless of these concerns, a clear hierarchy is evident with urinary tract infection (UTI) the most prevalent non-respiratory infection, followed by soft tissue and gastrointestinal infections, as summarised in table

1.

Table 1 – Point prevalence of common infections in care home populations(3, 13-15)	
Type of infection	Point prevalence (% of care home population)
Respiratory	0.3-3.7
Upper Respiratory Tract	0.13
Lower Respiratory Tract	0.3-1.6
Urinary Tract	0.6-21.8
Skin and Soft Tissue	1.0-8.8
Gastrointestinal	0.50

Urinary tract infections (UTI)

UTI is common amongst care home residents^{15, 19} and is a major source of antibiotic prescribing in this group. Incidence and prevalence rates vary according to the diagnostic criteria used and whether they include patients with asymptomatic bacteriuria or catheters²⁰. A study in Californian nursing homes using resident records and laboratory reports determined an incidence of 34.2%²¹, whilst a prospective year-long surveillance study in a 103-bedded nursing home in Germany reported an incidence of 1 infection per 1000 resident-days¹⁶. Point prevalence rates have been reported at 1.58-3.8 in nursing homes^{13, 15}. The prevalence of asymptomatic bacteriuria is much higher, with rates of 15-50% in non-catheterised US residents of long term care facilities²² and similar rates documented in other countries^{23, 24}.

Bacteriology

The commonest causes of UTI in older people are *Escherichia coli* and *Proteus mirabilis*^{22, 25}. However, care home residents more frequently experience UTI due to bacteria which are antibiotic-resistant or more commonly associated with hospital infection, including *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Citrobacter spp.* or *Providencia stuartii*²⁶. This may, in part, be attributable to the high incidence of antibiotic prescribing in the older population, with studies suggesting a strong

link between the frequency with which an antibiotic is prescribed and subsequent antimicrobial resistance amongst urinary pathogens^{25, 27}.

Asymptomatic bacteriuria describes a positive urine culture, with or without pyuria and without associated clinical symptoms. It is commoner in women, with advancing age²⁸, is associated with pyuria in over 90% of cases²², is inevitable in long-term catheterisation and has an incidence of 3-6% per day with short-term catheters²⁹. Treatment has no effect on mortality or incidence of symptomatic infection and leads to morbidity from drug side effects and a higher incidence of antimicrobial resistance²². The number needed to harm (NNH) for antibiotic treatment of asymptomatic bacteriuria is 3 (95%CI 2-10)²⁹.

Diagnosis

The diagnosis of UTI is frequently overestimated in care home residents³⁰. Asymptomatic bacteriuria should not be treated and symptomatic enquiry should therefore guide diagnosis. Symptoms are commonly divided into lower urinary tract symptoms (LUTS) – dysuria, urgency and frequency – and upper urinary tract symptoms (UUTS) – LUTS plus loin pain and fever.

Diagnostic criteria for UTI in residents of long term care facilities were developed in 2000 by a US expert-consensus panel using a modified Delphi approach³¹. They suggested, for patients without a catheter, that a UTI could be diagnosed on the basis of acute dysuria alone, or a fever of 37.9 °C plus any of new or worsening urgency, frequency, suprapubic pain, macroscopic haematuria, urinary incontinence or costovertebral angle tenderness. In those with a catheter, LUTS were held to be less useful and it was therefore suggested that diagnosis be on the basis of a fever of 37.9 °C or 1.5 °C over baseline, new costovertebral angle tenderness, rigors or new onset of delirium. It is likely, however, that the false attribution of delirium to urinary tract infection because of the high prevalence of asymptomatic bacteriuria results in much of the overtreatment previously described³² and these guidelines should therefore be applied with some degree of caution.

A study of 551 non-catheterised nursing home residents found dysuria (RR 1.58 95%CI 1.10-2.03), change in character of urine (RR 1.42 95%CI 1.07-1.79) and change in mental status (RR1.38 95%CI 1.03-1.74) to be associated with bacteriuria and pyuria³³. Dysuria plus one or both other symptoms was the highest predictor of bacteriuria/pyuria, and identified 63.2% of people with a UTI.

Obtaining samples

Obtaining samples of urine from care home residents may be difficult due to frailty, incontinence and cognitive impairment. A mid-stream urine is non-invasive but is associated with a high contamination rate which, contrary to received wisdom, is not reduced by cleansing the urethra with water³⁴. In men a urethral sheath can be used in patients who cannot produce a specimen whilst, in women, an in-and-out catheter may be the best option²². Newcastle urine collection pads (UCPs) represent a non-invasive way of collecting urine, however specimens collected from these are much more useful for dipstick urinalysis than microbiological analysis^{35,36}.

Bedside tests

Bedside tests are useful only as an adjunct to clinical diagnosis. Visual inspection of urine has a 90.4% sensitivity but only a 66.4% specificity for bacteriuria, is dependent upon the experience of the observer and is not, therefore, a useful test²⁹. Dipstick tests, meanwhile, are frequently positive for leucocytes due to the high prevalence of asymptomatic bacteriuria in the care home population. A dipstick positive for leucocytes and nitrites has a disappointingly low positive predictive value of 44%³³. Dipstick tests can also miss UTI as a consequence of the fact that some organisms, including *Streptococcus pneumoniae*, *Enterococcus* or *Pseudomonas aeruginosa*, do not express bacterial nitrate reductase³⁷ and are therefore of limited value in care homes. Men have a high incidence of UTI secondary to organisms which may not form nitrites and should have urine sent for culture if they have symptoms of UTI, regardless of dipstick result²⁹. Dipstick tests should not be performed in patients with long-term catheters as constant bacteriuria and pyuria means that the test is not useful.

Laboratory diagnosis

The threshold for laboratory diagnosis of UTI varies between countries. In the UK, 10^4 colony forming units per ml (cfu/ml) is used as a lower threshold of a positive culture³⁴. However, counts as low as 10^2 cfu/ml may be accepted in women with definite symptoms of urinary tract infection (“low-count” UTI)²⁹. In men, while there is less evidence to guide laboratory diagnosis, a lower cut-off of 10^3 cfu/ml with 80% predominance of one organism may be diagnostic of UTI²⁹. This is lower than the current UK laboratory standard of 10^4 cfu/ml, and samples with 10^3 cfu/ml may be reported “no significant growth”. Liaison with a microbiologist may be indicated if clinically a UTI is suspected but a sample is negative.

In catheterized patients, surveillance cultures are not necessary and may lead to harm of the patient through unnecessary treatment. Similarly, follow-up cultures after successful treatment are not required³⁴.

Prevention

A recent Cochrane review considering prevention of UTI suggested that antibiotic prophylaxis was beneficial in non-pregnant patients^{29, 38}. The benefit of antibiotics ceased after discontinuation. However, the majority of studies were in younger patients and no studies were conducted specifically in the care home setting. Further, the incidence of clostridium difficile enteritis in care homes is high and the role of antibiotics in the aetiology of such infections well demonstrated.

The Scottish Intercollegiate Guideline Network (SIGN) suggest that high-dose cranberry tablets can be used to prevent recurrent UTI²⁹. Only one head-to-head trial has compared the effectiveness of antibiotic prophylaxis with cranberry products, showing a modest but statistically non-significant advantage for antibiotics, with a relative risk of symptomatic UTI during treatment of 1.62 in the cranberry group compared with antibiotics³⁹. The number needed to treat to prevent one infection with cranberry is 6.4 compared with for 1.1 antibiotics but the number of adverse events is

significantly lower⁴⁰. Care is needed when advising cranberry for those on warfarin as the INR (international normalized ratio) may be increased.

Oral oestrogens appear to be less effective than antibiotic prophylaxis and are not recommended for prevention of UTI²⁹. Vaginal oestrogens may be of some benefit.

Treatment

Women with LUTS may be treated with three day courses of antibiotics²⁹, though some older women will relapse and in this case longer courses of up to 10 days may be needed. A Cochrane review compared single dose, 3-6 days and 7-9 days of antibiotic therapy for UTI and concluded that 3-6 days was associated with fewer adverse effects than longer courses but had similar benefits⁴¹. Single dose therapy was least effective.

Men usually need longer courses of antibiotics (7-14 days) due to high rates of prostatic involvement²⁹. Some UTIs are described as “complicated” because bacteria are more difficult to eradicate, and longer courses of antibiotics are necessary. Examples of abnormalities which cause complicated UTI include renal or bladder stones, the presence of a urinary catheter, diabetes mellitus, neurogenic bladder, bladder outflow obstruction and ureteric reflux, all of which are common in care home residents. Because patients with catheters commonly present with multi-resistant organisms, treatment should be given according to antibiotic sensitivities. If treatment cannot be delayed, then antibiotic choice should take into account any previous positive urine cultures.

SIGN guidelines recommend ciprofloxacin for 7 days as first line treatment for patients with UUTS because of the possibility of bacteraemia²⁹. Quinolones may be particularly useful in the care home setting because they produce similar plasma levels whether given orally or intravenously³⁷ and thus can mitigate the need to admit to hospital. It should be noted that quinolones should not be used for treatment of UTI in general due to a high associated incidence of clostridium difficile²⁹ and should probably only be commenced following discussion with a microbiologist.

Men in care homes should be referred to urology if they fail to respond to antibiotics, develop two UTIs within three months, or manifest UUTS²⁹.

Skin and soft-tissue infections

The prevalence of skin infections is 1.0-8.8% amongst the care home population but they may account for up to 50% of infections^{15, 20}. Infections with Methicillin-Resistant *Staphylococcus aureus* (MRSA), infected decubitus ulcers, scabies and tinea raise specific issues for consideration in the care home setting.

Methicillin-Resistant Staphylococcus Aureus (MRSA)

22-23% of UK care home residents are colonised with MRSA^{42, 43}, compared with 0.8% of community-dwellers²² and 15% of hospital in-patients >65^{44, 45}. Colonisation is seen in 1.1-35% of residents of long-term care facilities depending on country⁴⁶⁻⁵⁰; though the reasons for such wide-ranging prevalence are unclear, they may include institutional factors such as the frequency of transfer to acute hospital, local infection control and antibiotic prescribing policies, and also the frequency of instrumentation.

Patients colonized with MRSA have a higher mortality than those colonised with methicillin-sensitive *Staphylococcus aureus* (MSSA), with an odds ratio of death of 1.93 (95% CI 1.54–2.42)⁵¹, though this difference is less marked in the care home population than in hospitalised patients⁵² possibly due to lower rates of instrumentation. Care-home residents are more likely to become colonized if they have invasive devices (such as indwelling catheters or gastrostomy)⁵³, decubitus ulcers⁵⁴, have previously been colonised with MRSA or have recently been on antibiotics⁵⁵.

The interaction between reservoirs of MRSA in care homes and hospitals is well recognised with recent transfer representing an independent risk factor for MRSA infection⁵⁶⁻⁵⁸. Failure to control infection rates in one reservoir can overwhelm the other⁵⁹ and controlling colonisation rates in care homes has therefore been seen as possible way to influence infection rates in acute hospitals⁶⁰. Unfortunately, a Cochrane review revealed no evidence for effectiveness of eradication of MRSA

using either topical or systemic antimicrobial therapy in any setting, including care homes⁶¹. A second Cochrane review, which sought randomized-controlled trials evaluating barrier methods, hand-washing or environmental hygiene in a care home setting, found no studies to meet its inclusion criteria⁶⁰. Clearly, further care home specific trials of MRSA-eradication and prevention strategies are needed if a cogent argument is to be made that such measures are either effective or beneficial.

Decubitus ulcers

Decubitus ulcers are common in care homes, with a prevalence of 11.9-39% compared to 8.3-23% of acute hospital inpatients⁶². There is considerable international variation with prevalence being six times higher, for example, in the Netherlands than in Germany, with no apparent explanation for the difference⁶³. Incidence rates are at their highest in the first few days following admission to a home⁶⁴, suggesting that many ulcers are sustained⁶⁴ during acute admissions, prior to transfer to the home, or in the recovery period immediately following acute illness. The higher prevalence of MRSA and other antibiotic resistant organisms⁶⁵ in care homes increases the likelihood that these ulcers will become colonized by multi-resistant bacteria. MRSA is more virulent and more likely to cause acute bacteraemia in active wounds such as decubitus ulcers than other bacteria⁵⁰.

Signs of infection in pressure ulcers are erythema at the ulcer margin, malodour, new pain, warmth or purulent discharge. The International Guidelines on Pressure Ulcer Management recommend that infection be confirmed by either tissue biopsy or quantitative swab techniques before commencing antibiotics⁶⁶. Randomized controlled-trial level evidence to guide treatment is unavailable but expert consensus suggests use of silver and iodine impregnated dressings as a first-line, with topical antibacterial agents reserved for patients slow to respond^{66, 67}. Systemic sepsis as a complication of decubitus ulcer has a high mortality in the care home population, and physicians should have a low threshold for instituting hospital admission in patients who develop signs of sepsis⁶⁸.

Osteomyelitis secondary to decubitus ulcer is significantly more likely in full-thickness ulcers involving bone⁶⁸. It cannot be diagnosed clinically in the care home setting, and when suspected, patients should be admitted to hospital for definitive imaging, usually Magnetic Resonance Imaging, followed by deep bone biopsy to guide treatment⁶⁹. Clearly, such decisions need to be shaped by an understanding of a resident's overall prognosis.

Scabies

Scabies is common in care homes: in one survey of 179 Canadian long-term care facilities, 25% reported a scabies outbreak over a 1-year period⁷⁰. It is caused by irritation from the eggs, faeces and saliva of the *sarcoptes scabiei* mite, which reproduces in the interdigital spaces of the hands and feet, axillae and genital regions of infected patients. The usual response is for a patient to scratch, killing the mite and therefore keeping the number of infesting organisms low.

The classical presentation is of intense pruritis, widespread papules, vesicles and excoriations and, most characteristically, serpiginous burrows a millimetre or two in length in the interdigital spaces or on the forearm. On close inspection, the lesion can sometimes be seen to have a burrow entrance, with a vesicle (and sometimes a mite, visible as a black dot) at the opposite end⁷¹. Scabies can be sexually transmitted and it is important to remember this as a differential diagnosis for genital pruritis in the care home population⁷².

Norwegian, or crusted, scabies is a fulminant version of the infection, characterised by high levels of infestation with thousands of mites on a single patient⁷¹ due to frailty, immunosenescence and a reduced ability to scratch^{71, 73}. A consequence of such high levels of infestation is the formation of atypical crusted skin lesions which can be indistinguishable from psoriasis or eczema. Eosinophilia is seen in just over half of cases, which may raise suspicion⁷⁴. Serum IgE levels are increased in up to 98% of cases but this test is not routinely conducted in clinical practice.

Classical (non-Norwegian) scabies is contagious where there is prolonged skin-to-skin contact⁷⁵, which is common in care homes. Further, patients are often asymptomatic but contagious during the

3-week incubation period⁷⁶ – during which time they could easily infect staff members or other residents. Norwegian scabies is highly contagious because of the high parasite load and can spread by even transient skin-to-skin contact^{73,76}.

Current guidelines tend to favour the use of topical permethrin on the basis of proven efficacy and a favourable side-effect profile^{77, 78}. Malathion is probably better tolerated but has less evidence supporting its efficacy and, in particular, no head-to-head trial with permethrin is available⁷⁸. Effective application, with complete body coverage below the neck and concordance with treatment is essential for efficacy – permethrin requires two applications lasting twelve hours and malathion, one application lasting 24 hours. A key issue in care homes is that all residents with scabies, along with all residents or staff who have had contact with them, are treated simultaneously – this in practice often means treating all residents and staff in the home⁷⁹.

Oral ivermectin is an effective but controversial treatment against scabies⁸⁰ because of excess deaths following treatment amongst 47 residents of a Canadian long-term care facility⁸¹. However, most authorities assert that the treatment is safe, as these findings were not reproduced in cohorts of 47 Colombian⁸² and 220 Dutch⁸³ nursing home residents. It is available in the UK on a named patient basis for treatment of Norwegian scabies⁷⁷ but is not recommended by the Food and Drug Administration in the US⁸⁴.

Following treatment, the patient's bedding and clothing should be washed at minimum 50°C and all soft furnishings and carpets vacuumed⁷⁷. The scabies mite is incapable of surviving outside of the body for longer than 24 to 36 hours in conditions typically found in centrally-heated care homes⁸⁵, and is dependent on shed skin cells for survival, which are readily removed by modern vacuum cleaners.

Tinea

Tinea pedis, corporis and capitis describe fungal infections of the feet, body and scalp respectively. These are characteristically superficial skin infections of trichophyton or microsporum species and are

usually sporadic and readily controlled by routine topical antifungal therapy⁸⁶. Both outbreaks and epidemics of tinea have been described in care homes^{87, 88} and involved atypical fungal organisms, spread by staff and personal hygiene utensils (combs and razors). Staff also contracted the infection. Staff therefore need to ensure both prompt medical attention and close attention to barrier nursing methods when index cases of tinea are identified.

Gastrointestinal infections

Although the incidence of gastroenteritis is relatively low in older patients, care home residents are much more likely than their community-dwelling counterparts to be hospitalised or die as a consequence of infection⁸⁹. A US review conducted 1995-1997 reported the death rate in nursing home residents due to gastroenteritis as 38.91 (95% CI, 38.55–39.27) per 100,000 persons, compared with 8.50 (95% CI, 8.47–8.53) for all over-65s⁹⁰. Gastroenteritis can be split into viral and bacterial aetiology, with special consideration given to *Clostridium difficile* given its high mortality in this group.

Viral gastroenteritis

Epidemiology

The incidence of viral gastroenteritis, of which the two most common causes are norovirus (synonyms “Norwalk-like viruses” and “small, round-structured viruses”) and rotavirus, considerably outstrips that of bacterial gastrointestinal infections in care homes⁹¹. In 2006, norovirus was the cause of 96% of acute gastroenteritis outbreaks in the US. Of these, 50% of the non-food related outbreaks occurred in long-term care facilities⁹². In Europe, 34-39% of norovirus outbreaks occur in care homes^{93, 94}.

Norovirus infections in care homes are commonly associated with person-to-person spread, predominantly via the faecal oral route, even when the index case is acquired from food^{92, 95}. A characteristic winter peak in incidence occurs, which coincides almost exactly with the winter peak in respiratory infections⁹⁴.

Diagnosis

Kaplan et al⁹⁶ suggested that viral gastroenteritis can be diagnosed clinically and is characterised by a short incubation period (24-60 h), a short infection duration (12–60 h) and a high frequency of vomiting (>50% of cases). These criteria are, however, based upon studies in healthy volunteers and critics suggest that infections are both less typical and less benign in care home residents⁹⁴.

Infection Control

Because norovirus is environmentally very stable, difficult to eradicate and highly infectious, particular attention is required to infection control measures. Chadwick et al⁹⁷ provided useful recommendations for the containment of norovirus outbreaks in the hospital setting, most of which also apply to the care home setting, and these are summarised in box 1.

Box 1: Measures for containment of norovirus infection, from Chadwick et al(97)

- Isolate or cohort symptomatic residents.
- Wear gloves and apron for contact with affected patients and change these between patients.
- Wash hands with soap and water after contact with an affected patient.
- Exclude affected staff from duties until symptom-free for 48 hours.
- Close of the facilities to new admissions.
- Limit visits and advise visitors on handwashing.
- Promptly clean body fluid spillages.
- Increase the frequency of routine cleaning.
- Use 0.1% (1000 ppm) hypochlorite to disinfect hard surfaces and clean soft furnishings with either steam or detergent and hot water.

Chlorhexidine and alcohol are ineffective against norovirus and therefore bleach must be used for cleansing surfaces, and soap and water for washing hands^{97, 98}. Staff must wear protective clothing

during contact with infected residents, as exposure to vomitus increases the risk of contracting the illness, though concordance with wearing gowns in particular appears poor⁹⁵.

Isolation and cohorting of residents can prove difficult in care homes due to shared bathroom facilities and wandering residents. Minimising the amount of time for which residents are isolated helps considerably. For norovirus, viral shedding in stool normally peaks between 24 and 72 hours⁹⁹ but can last longer, and has been reported up to 45 days following resolution of symptoms in care home residents¹⁰⁰. On the basis of pragmatism, most authors recommend isolating patients for 48-72 hours after symptom resolution⁹⁸.

The recommendation that staff be excluded from work for 48 hours following resolution of symptoms is similarly pragmatic, however longer periods of staff exclusion may be both desirable and cost-effective. A UK case-control study comparing staff exclusion for either 48 or 72 hours showed significantly lower infection and attack rates in the 72 hour group and suggested that the net effect on staff availability was minimal¹⁰¹. Staff education and a no-blame culture are essential to maintain staff compliance with such protocols, as staff presenteeism during illness is common⁹⁵.

Treatment

Much of the excess morbidity and mortality associated with viral gastroenteritis in the care home cohort is likely to be due to the effects of dehydration⁹⁴ and treatment largely focuses on avoiding this. UK care homes are unable to provide intravenous fluids and such therapy necessitates transfer to hospital. Clearly such transfer is problematic both because it spreads infection and exposes a frail patient to the risks of the acute hospital. An alternative is to use antimotility drugs, such as loperamide or racecadotril¹⁰², whilst pursuing aggressive oral rehydration.

Food-borne gastroenteritis

Food-borne gastroenteritis outbreaks can readily take place in care homes because food is often prepared in a central kitchen and served communally, and outbreaks due to *Bacillus cereus*¹⁰³,

*Salmonella enteritidis*¹⁰⁴ and verotoxin producing *Escherichia coli* serotype O157¹⁰⁵ have been described. Depending on the organism, these outbreaks can either be self-limiting or perpetuated by person-to-person spread. The potential for spread is greater in care homes than in the community due to the combination of close contact between residents and the high rates of faecal incontinence, especially during diarrhoeal illness. *E. coli* O157 is particularly important because of its association with haemolytic uraemic syndrome and excess mortality is reported over the age of 50¹⁰⁶. It can present non-specifically in care home residents, with visible bloody diarrhoea in only 65-75% of patients¹⁰⁶. Of the four outbreaks reported in the literature, the rate of conversion to haemolytic uraemic syndrome ranges from 0-14.2% and the mortality rate from 3-14.2%¹⁰⁶⁻¹¹⁰.

Clostridium difficile enteritis

Residence in a care home, especially in the first year after admission, is an independent risk factor for *Clostridium difficile* infection, which occurs in 7-9% of residents compared with 2% of healthy adults¹¹¹. Reasons for this include high levels of antibiotic prescribing, the increased incidence of proton-pump inhibitor mediated achlorhydia and the frailty of care home residents^{111, 112}. Once established in a home, *clostridium difficile* can spread via the faecal-oral route and through contact with contaminated surfaces and attention to isolation measures is, again, important. Handwashing using soap and surface cleansing using hypochlorite based substances are essential because these, unlike alcohol, are sporicidal¹¹².

Generic issues around gastroenteritis in care homes

Analysis of stool specimens is particularly important in care home residents because it allows correct selection of treatment with loperamide, for example, which is best avoided in haemorrhagic *E. coli* or *Clostridium difficile* infections¹¹³ but represents an important therapy in viral gastroenteritis. It also allows epidemiological analysis of infections to identify the infecting strain of each organism and track its spread.

Regardless of the cause of enteritis, it is likely that the early parts of recognition and management will be conducted without input from a doctor or possibly even a nurse. Thus, operational definitions for gastroenteritis outbreaks are essential^{98, 104}. These should, ideally, be written in lay language and presented alongside clear guidelines about early isolation, specimen collection and fluid management. Such guidelines are not, at present, universally available.

Conclusion

Because of the concentration of frail older people in a semi-closed environment, care homes present particular challenges in the diagnosis and management of infection. Infections which are frequently inconsequential in community-dwelling adults can be life-threatening in care home residents, as is the case with UTI. Infections can also behave in atypical ways in this cohort, as is the case with Norwegian scabies or epidemic tinea.

A further challenge when dealing with such frail patients is deciding when to admit them to acute hospital care – which is fraught with risk. There is often an alternative, for example by using intramuscular antibiotics or oral quinolones for UTI, or by using antimotility agents in viral gastroenteritis. Occasionally, however, there is none, for example the need for detailed imaging and bone biopsy in suspected osteomyelitis complicating decubitus ulcer. When such difficult decisions are required they should be made with attention to the patient's overall prognosis and stated wishes.

At an individual home level, prompt recognition and treatment coupled to rigorous infection control are needed to minimise the impact of infection. The fact that care, in the UK at least, is often provided by staff without healthcare qualifications is a challenge to such systematic response. Consideration must be given as to what role care home staff can and should play. Guidelines for the sector should be written with them in mind and, almost certainly, with their input.

At a public health level, antibiotic stewardship and improved links from homes to microbiology and infection control services seem intuitive responses. How to provide such support consistently to a highly heterogeneous sector is a genuine challenge for health service providers and an area for research and development going forward.

Conflict of interest

The authors declare that they have no conflict of interests.

References

1. Bajekal M. Health Survey for England 2000: Characteristics of Care Homes and Their Residents. London: The Stationary Office; 2002.
2. Bowman C, Whistler J, Ellerby M. A national census of care home residents. *Age Ageing*. 2004;33(6):561-6.
3. Nicolle LE, Strausbaugh LJ, Garibaldi RA. Infections and antibiotic resistance in nursing homes. *Clin Microbiol Rev*. 1996;9(1):1-17.
4. Nicolle L. Infection Control in Long Term Care Facilities. *Clinical Infectious Diseases*. 2000;31(3):752-6.
5. Gordon A, Ewan V. Pneumonia and influenza: specific considerations in care homes. *Reviews in Clinical Gerontology*. 2010;20(01):69-80.
6. Great Britain Department of Health. Care Standards Act 2000. London: Stationery Office; 2001.
7. Ribbe MW, Ljunggren G, Steel K, et al. Nursing Homes in 10 Nations: A Comparison Between Countries and Settings. *Age Ageing*. 1997 January 1, 1997;26(suppl_2):3-12.
8. Conroy S, Van Der Cammen T, Schols J, et al. Medical services for older people in nursing homes — Comparing services in England and The Netherlands. *The Journal of Nutrition, Health and Aging*. 2009;13(6):559-63.
9. Care of Elderly: UK Market Survey. London: Laing and Buisson 2009.
10. Fell G. Preparedness of Residential and Nursing Homes for Pandemic Flu. *J Public Health*. 2008;30(1):99-102.
11. Cheesebrough JS, Green J, Gallimore CI, Wright PA, Brown DWG. Widespread environmental contamination with Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiology and Infection*. 2000;125(01):93-8.
12. Strausbaugh LJ, Joseph CL. The burden of infection in long-term care. *Infect Control Hosp Epidemiol*. 2000;21(10):674-9.
13. Tsan L, Davis C, Langberg R, et al. Prevalence of nursing home-associated infections in the Department of Veterans Affairs nursing home care units. *American Journal of Infection Control*. 2008 Apr;36(3):173-9.
14. Andersen BM, Rasch M. Hospital-acquired infections in Norwegian long-term-care institutions. A three-year survey of hospital-acquired infections and antibiotic treatment in nursing/residential homes, including 4500 residents in Oslo. *J Hosp Infect*. 2000;46(4):288-96.
15. Eriksen HM, Iversen BG, Aavitsland P. Prevalence of nosocomial infections and use of antibiotics in long-term care facilities in Norway, 2002 and 2003. *J Hosp Infect*. 2004 Aug;57(4):316-20.
16. Engelhart ST, Hanses-Derendorf L, Exner M, Kramer MH. Prospective surveillance for healthcare-associated infections in German nursing home residents. *J Hosp Infect*. 2005 May;60(1):46-50.

17. Cools HJM, van der Meer JWM. Infection control in a skilled nursing facility: a 6-year survey. *Journal of Hospital Infection*. 1988;12(2):117-24.
18. Moens GF, Haenen R, Jacques P. The prevalence of infections in nursing homes in Belgium. *Journal of Hospital Infection*. 1996;34(4):336-7.
19. Beck-Sague C, Banerjee S, Jarvis WR, et al. Infectious diseases and mortality among US nursing home residents. *American Journal of Public Health*. 1993 Dec;83(12):1739-42.
20. Garibaldi RA. Residential care and the elderly: the burden of infection. *J Hosp Infect*. 1999 Dec;43 Suppl:S9-18.
21. Beck-Sague C, Villarino E, Giuliano D, et al. Infectious diseases and death among nursing home residents: results of surveillance in 13 nursing homes. *Infection Control & Hospital Epidemiology*. 1994 Jul;15(7):494-6.
22. Nicolle LE. Urinary tract infection in long-term-care facility residents. *Clinical Infectious Diseases*. 2000 Sep;31(3):757-61.
23. Hedin K, Petersson C, Wideback K, et al. Asymptomatic bacteriuria in a population of elderly in municipal institutional care. *Scandinavian Journal of Primary Health Care*. 2002 Sep;20(3):166-8.
24. Aguirre-Avalos G, Zavala-Silva ML, Daz-Nava A, Amaya-Tapia G, Aguilar-Benavides S. Asymptomatic Bacteriuria and Inflammatory Response to Urinary Tract Infection of Elderly Ambulatory Women in Nursing Homes - molecular pathogenesis and clinical management. *Archives of Medical Research*. 1999;30:29-32.
25. Vromen M, van der Ven AJ, Knols A, et al. Antimicrobial resistance patterns in urinary isolates from nursing home residents. Fifteen years of data reviewed. *Journal of Antimicrobial Chemotherapy*. 1999 Jul;44(1):113-6.
26. Muder RR, Brennen C, Wagener MM, et al. Bacteremia in a long-term-care facility: a five-year prospective study of 163 consecutive episodes. *Clinical Infectious Diseases*. 1992 Mar;14(3):647-54.
27. Cohen AE, Lautenbach E, Morales KH, Linkin DR. Fluoroquinolone-resistant *Escherichia coli* in the long-term care setting. *Am J Med*. 2006;119(11):958-63.
28. Hassanzadeh P, Motamedifar M. The prevalence of asymptomatic bacteriuria in long term care facility residents in Shiraz, Southwest Iran: a cross-sectional study. *Pak J Biol Sci*. 2007;10(21):3890-4.
29. SIGN. Management of suspected bacterial urinary tract infection in adults. A national clinical guideline 2006: Available from: www.sign.ac.uk/pdf/sign88.pdf
30. Stevenson KB, Moore JW, Sleeper B. Validity of the Minimum Data Set in Identifying Urinary Tract Infections in Residents of Long-Term Care Facilities. *Journal of the American Geriatrics Society*. 2004;52(5):707-11.
31. Loeb M, Bentley DW, Bradley S, et al. Development of minimum criteria for the initiation of antibiotics in residents of long-term-care facilities: results of a consensus conference. *Infect Control Hosp Epidemiol*. 2001;22(2):120-4.
32. McMurdo M, Gillespie N. Commentary. Urinary tract infection in old age: over-diagnosed and over-treated. *Age Ageing*. 2000 July 1, 2000;29(4):297-8.
33. Juthani-Mehta M, Quagliarello V, Perrelli E, et al. Clinical features to identify urinary tract infection in nursing home residents: a cohort study. *Journal of the American Geriatrics Society*. 2009 Jun;57(6):963-70.
34. HPA. Diagnosis of UTI. Quick reference guide for primary care 2009: Available from: http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1194947404720.
35. Macfarlane PI, Ellis R, Hughes C, Houghton C, Lord R. Urine collection pads: are samples reliable for urine biochemistry and microscopy? *Pediatric Nephrology*. 2005;20(2):170-9.
36. Farrell M, Devine K, Lancaster G, Judd B. A method comparison study to assess the reliability of urine collection pads as a means of obtaining urine specimens from non-toilet-trained children for microbiological examination. *Journal of Advanced Nursing*. 2002;37(4):387-93.

37. Beier MT. Management of Urinary tract infections in the nursing home elderly: a proposed algorithmic approach. *International Journal of Antimicrobial Agents*. 1999 May;11(3-4):275-84.
38. Albert X, Huertas I, Pereiro, II, et al. Antibiotics for preventing recurrent urinary tract infection in non-pregnant women. *Cochrane Database of Systematic Reviews*. 2004(3).
39. McMurdo MET, Argo I, Phillips G, Daly F, Davey P. Cranberry or trimethoprim for the prevention of recurrent urinary tract infections? A randomized controlled trial in older women. *J Antimicrob Chemother*. 2009;63(2):389-95.
40. Sumukadas D, Davey P, McMurdo MET. Recurrent urinary tract infections in older people: the role of cranberry products. *Age Ageing*. 2009;38(3):255-7.
41. Lutters M, Vogt-Ferrier NB, Lutters M, Vogt-Ferrier NB. Antibiotic duration for treating uncomplicated, symptomatic lower urinary tract infections in elderly women. *Cochrane Database of Systematic Reviews*. 2008(3).
42. Baldwin NS, Gilpin DF, Hughes CM, et al. Prevalence of methicillin-resistant *Staphylococcus aureus* colonization in residents and staff in nursing homes in Northern Ireland. *J Am Geriatr Soc*. 2009;57(4):620-6.
43. Barr B, Wilcox M, Brady A, et al. Prevalence of Methicillin Resistant *Staphylococcus aureus* Colonization Among Older Residents of Care Homes in the United Kingdom. *Infection Control and Hospital Epidemiology*. 2007;28(7):853-9.
44. Maudsley J, Stone SP, Kibbler CC, et al. The community prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) in older people living in their own homes: implications for treatment, screening and surveillance in the UK. *Journal of Hospital Infection*. 2004;57(3):258-62.
45. Hori S, Sunley R, Tami A, Grundmann H. The Nottingham *Staphylococcus aureus* population study: prevalence of MRSA among the elderly in a university hospital. *Journal of Hospital Infection*. 2002;50(1):25-9.
46. Baum H, Schmidt C, Svoboda D, Bockâ Hensley O, Wendt C. Risk Factors for Methicillin Resistant *Staphylococcus aureus* Carriage in Residents of German Nursing Homes. *Infection Control and Hospital Epidemiology*. 2002;23(9):511-5.
47. O'Sullivan NP, Keane CT. The prevalence of methicillin-resistant *Staphylococcus aureus* among the residents of six nursing homes for the elderly. *Journal of Hospital Infection*. 2000;45(4):322-9.
48. Denis O, Jans B, Deplano A, et al. Epidemiology of methicillin-resistant *Staphylococcus aureus* (MRSA) among residents of nursing homes in Belgium. *J Antimicrob Chemother*. 2009 December 1, 2009;64(6):1299-306.
49. Talon DR, Bertrand X. Methicillin Resistant *Staphylococcus aureus* in Geriatric Patients: Usefulness of Screening in a Chronic Care Setting. *Infection Control and Hospital Epidemiology*. 2001;22(8):505-9.
50. Manzur A, Gudiol F. Methicillin-resistant *Staphylococcus aureus* in long-term-care facilities. *Clinical Microbiology and Infection*. 2009;15(s7):26-30.
51. Cosgrove S, Sakoulas G, Perencevich E, et al. Comparison of Mortality Associated with Methicillin Resistant and Methicillin Susceptible *Staphylococcus aureus* Bacteremia: A Meta-analysis. *Clinical Infectious Diseases*. 2003;36(1):53-9.
52. Bradley SF. Methicillin-resistant *Staphylococcus aureus*: : Long-term care concerns. *The American Journal of Medicine*. 1999;106(5, Supplement 1):2-10.
53. Mody L, Maheshwari S, Galecki A, Kauffman CA, Bradley SF. Indwelling Device Use and Antibiotic Resistance in Nursing Homes: Identifying a High-Risk Group. *Journal of the American Geriatrics Society*. 2007;55(12):1921-6.
54. Bradley SF, Terpenning MS, Ramsey MA, et al. Methicillin-resistant *Staphylococcus aureus*: Colonization and Infection in a Long-term Care Facility. *Annals of Internal Medicine*. 1991 September 15, 1991;115(6):417-22.

55. O'Sullivan NP, Keane CT. Risk factors for colonization with methicillin-resistant *Staphylococcus aureus* among nursing home residents. *Journal of Hospital Infection*. 2000;45(3):206-10.
56. Manzur A, Gavalda L, Ruiz de Gopegui E, et al. Prevalence of methicillin-resistant *Staphylococcus aureus* and factors associated with colonization among residents in community long-term-care facilities in Spain. *Clinical Microbiology and Infection*. 2008;14(9):867-72.
57. Brugnaro P, Fedeli U, Pellizzer G, et al. Clustering and Risk Factors of Methicillin-Resistant *Staphylococcus aureus* Carriage in Two Italian Long-Term Care Facilities. *Infection*. 2009;37(3):216-21.
58. Fraise AP, Mitchell K, Brien SJ, Oldfield K, Wise R. Methicillin-resistant *Staphylococcus aureus* (MRSA) in nursing homes in a major UK city: an anonymized point prevalence survey. *Epidemiology and Infection*. 1997;118(1):1-5.
59. Cooper BS, Medley GF, Stone SP, et al. Methicillin-resistant *Staphylococcus aureus* in hospitals and the community: Stealth dynamics and control catastrophes. *Proceedings of the National Academy of Sciences of the United States of America*. 2004 July 6, 2004;101(27):10223-8.
60. Hughes CM, Smith MBH, Tunney MM. Infection control strategies for preventing the transmission of methicillin-resistant *Staphylococcus aureus* (MRSA) in nursing homes for older people. *Cochrane Database Syst Rev*. 2008(1).
61. Loeb M, Main C, Walker-Dilks C, Eady A. Antimicrobial drugs for treating methicillin-resistant *Staphylococcus aureus* colonization. *Cochrane Database Syst Rev*. 2003(4).
62. Vanderwee K, Clark M, Dealey C, Gunningberg L, Defloor T. Pressure ulcer prevalence in Europe: a pilot study. *Journal of Evaluation in Clinical Practice*. 2007;13(2):227-35.
63. Tannen A, Dietz E, Dassen T, Halfens R. Explaining the national differences in pressure ulcer prevalence between the Netherlands and Germany adjusted for personal risk factors and institutional quality indicators. *Journal of Evaluation in Clinical Practice*. 2009;15(1):85-90.
64. Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. *J Am Geriatr Soc*. 1992;40(8):747-58.
65. Wiener J, Quinn JP, Bradford PA, et al. Multiple Antibiotic-Resistant *Klebsiella* and *Escherichia coli* in Nursing Homes. *JAMA*. 1999 February 10, 1999;281(6):517-23.
66. European Pressure Ulcer Advisory Panel and National Pressure Ulcer Advisory Panel. *Treatment of pressure ulcers: Quick Reference Guide*. Washington DC: National Pressure Ulcer Advisory Panel; 2009.
67. UK National Institute of Clinical Excellence. CG29 - The management of pressure ulcers in primary and secondary care: a clinical practice guideline. London: National Institute of Clinical Excellence/Royal College of Nursing; 2005.
68. Smith DM. Pressure Ulcers in the Nursing Home. *Annals of Internal Medicine*. 1995 September 15, 1995;123(6):433-8.
69. Gannon KL, Braun T. Osteomyelitis. *Essential Infectious Disease Topics for Primary Care* 2008. p. 213-23.
70. Holness DL, DeKoven JG, Nethercott JR. Scabies in Chronic Health Care Institutions. *Arch Dermatol*. 1992 September 1, 1992;128(9):1257-60.
71. Tjioe M, Vissers WHPM. Scabies Outbreaks in Nursing Homes for the Elderly: Recognition, Treatment Options and Control of Reinfestation. *Drugs & Aging*. 2008;25:299-306.
72. García RG, López-Areal JdLL, Martínez CA. Scabies in the elderly. *Journal of the European Academy of Dermatology & Venereology*. 2004;18(1):105-7.
73. Heukelbach J, Feldmeier H. Scabies. *The Lancet*. 2006;367(9524):1767-74.
74. Roberts LJ, Huffam SE, Walton SF, Currie BJ. Crusted scabies: clinical and immunological findings in seventy-eight patients and a review of the literature. *Journal of Infection*. 2005;50(5):375-81.
75. Mellanby K. Transmission of Scabies. *Br Med J*. 1941 September 20, 1941;2(4211):405-6.
76. Chosidow O. Scabies and pediculosis. *The Lancet*. 2000;355(9206):818-.

77. Clinical Effectiveness Group of the British Association of Sexual Health and HIV. United Kingdom National Guideline on the Management of Scabies Infestation, available at <http://www.bashh.org/documents/27/27.pdf> (accessed 20th June 2010).
78. Strong M, Johnstone PW. Interventions for treating scabies. *Cochrane Database Syst Rev.* 2007(3).
79. Health Protection Agency (North West). The Management of Scabies in the Community, available at http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1194947308867 (accessed 14th August 2010). 2007.
80. Meinking TL, Taplin D, Herminda JL, Pardo R, Kerdel FA. The Treatment of Scabies with Ivermectin. *N Engl J Med.* 1995 July 6, 1995;333(1):26-30.
81. Barkwell R, Shields S. Deaths associated with ivermectin treatment of scabies. *The Lancet.* 1997;349(9059):1144-5.
82. Diazgranados JA, Costa JL. Deaths after ivermectin treatment. *The Lancet.* 1997;349(9066):1698-.
83. Reintjes R, Hoek C. Deaths associated with ivermectin for scabies. *The Lancet.* 1997;350(9072):215-.
84. Scheinfeld N. Controlling Scabies in Institutional Settings: A Review of Medications, Treatment Models, and Implementation. *American Journal of Clinical Dermatology.* 2004;5:31-7.
85. Arlian LG, Runyan RA, Achar S, Estes SA. Survival and infectivity of *Sarcoptes scabiei* var. *canis* and var. *hominis*. *J Am Acad Dermatol.* 1984;11(2 Pt 1):210-5.
86. Havlickova B, Czaika VA, Friedrich M. Epidemiological trends in skin mycoses worldwide. *Mycoses.* 2008;51(s4):2-15.
87. Summerbell RC, Krajden S, Kane J. Dermatophytosis in institutions for the elderly. *CMAJ.* 1989;140(2):112-.
88. Kakutani H, Kakutani T, Mochizuki T. *Trichophyton violaceum* infection occurring in a nursing home. *Nippon Ishinkin Gakkai Zasshi.* 2005;46(4):279-84.
89. Kirk MD, Roberts L, Horvath J. Understanding gastroenteritis in elderly residents of aged-care facilities. *Med J Aust.* 2008;189(9):476-7.
90. Frenzen PD. Mortality due to gastroenteritis of unknown etiology in the United States. *J Infect Dis.* 2003;187(3):441-52.
91. Marshall J, Botes J, Gorrie G, et al. Rotavirus detection and characterisation in outbreaks of gastroenteritis in aged-care facilities. *J Clin Virol.* 2003;28(3):331-40.
92. Norovirus activity--United States, 2006-2007. *MMWR Morb Mortal Wkly Rep.* 2007;56(33):842-6.
93. Fretz R, Svoboda P, Thi TM, Tanner M, Baumgartner A. Outbreaks of gastroenteritis due to infections with Norovirus in Switzerland, 2001&2003. *Epidemiology and Infection.* 2005;133(03):429-37.
94. Lopman BA, Adak GK, Reacher MH, Brown DWG. Two epidemiologic patterns of norovirus outbreaks: surveillance in England and wales, 1992-2000. *Emerg Infect Dis.* 2003;9(1):71-7.
95. Recurring norovirus outbreaks in a long-term residential treatment facility - Oregon, 2007. *MMWR Morb Mortal Wkly Rep.* 2009;58(25):694-8.
96. Kaplan JE, Feldman R, Campbell DS, Lookabaugh C, Gary GW. The frequency of a Norwalk-like pattern of illness in outbreaks of acute gastroenteritis. *Am J Public Health.* 1982;72(12):1329-32.
97. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastroenteritis due to small roundstructured viruses. *J Hosp Infect.* 2000;45(1):1-10.
98. Drinka PJ. Norovirus outbreaks in nursing homes. *J Am Geriatr Soc.* 2005;53(10):1839-40.
99. Graham DY, Jiang X, Tanaka T, et al. Norwalk virus infection of volunteers: new insights based on improved assays. *J Infect Dis.* 1994;170(1):34-43.
100. Tu ETV, Bull RA, Kim M-J, et al. Norovirus excretion in an aged-care setting. *J Clin Microbiol.* 2008;46(6):2119-21.

101. Vivancos R, Sundkvist T, Barker D, Burton J, Nair P. Effect of exclusion policy on the control of outbreaks of suspected viral gastroenteritis: Analysis of outbreak investigations in care homes. *Am J Infect Control*.38(2):139-43.
102. Gallelli L, Colosimo M, Tolotta GA, et al. Prospective randomized double-blind trial of racecadotril compared with loperamide in elderly people with gastroenteritis living in nursing homes. *Eur J Clin Pharmacol*.66(2):137-44.
103. DeBuono BA, Brondum J, Kramer JM, Gilbert RJ, Opal SM. Plasmid, serotypic, and enterotoxin analysis of *Bacillus cereus* in an outbreak setting. *J Clin Microbiol*. 1988;26(8):1571-4.
104. Frank C, Buchholz U, MaaSZ M, et al. Protracted outbreak of *S. Enteritidis* PT 21c in a large Hamburg nursing home. *BMC Public Health*. 2007;7(1):243.
105. Hemorrhagic colitis in a nursing home in Ontario. *CMAJ*. 1986;134(1):50-.
106. Reiss G, Kunz P, Koin D, Keeffe EB. *Escherichia coli* O157:H7 infection in nursing homes: review of literature and report of recent outbreak. *J Am Geriatr Soc*. 2006;54(4):680-4.
107. Krishnan C, Fitzgerald VA, Dakin SJ, Behme RJ. Laboratory investigation of outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7. *J Clin Microbiol*. 1987;25(6):1043-7.
108. Ryan CA, Tauxe RV, Hosesk GW, et al. *Escherichia coli* O157:H7 diarrhea in a nursing home: clinical, epidemiological, and pathological findings. *J Infect Dis*. 1986;154(4):631-8.
109. Carter AO, Borczyk AA, Carlson JA, et al. A severe outbreak of *Escherichia coli* O157:H7--associated hemorrhagic colitis in a nursing home. *N Engl J Med*. 1987;317(24):1496-500.
110. Waters JR, Sharp JC, Dev VJ. Infection caused by *Escherichia coli* O157:H7 in Alberta, Canada, and in Scotland: a five-year review, 1987-1991. *Clin Infect Dis*. 1994;19(5):834-43.
111. Al-Tureihi FIJ, Hassoun A, Wolf-Klein G, Isenberg H. Albumin, length of stay, and proton pump inhibitors: key factors in *Clostridium difficile*-associated disease in nursing home patients. *J Am Med Dir Assoc*. 2005;6(2):105-8.
112. Crogan NL, Evans BC. *Clostridium difficile*: An Emerging Epidemic in Nursing Homes. *Geriatric Nursing*. 2007/6//;28(3):161-4.
113. Holtz LR, Neill MA, Tarr PI. Acute Bloody Diarrhea: A Medical Emergency for Patients of All Ages. *Gastroenterology*. 2009;136(6):1887-98.