



## Perspective

Reservoirs of *Acinetobacter baumannii* outside the hospital and potential involvement in emerging human community-acquired infections

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## SUMMARY

The objective of the present report was to review briefly the potentially community-acquired *Acinetobacter baumannii* infections, to update information on the reservoirs of *A. baumannii* outside the hospital, and to consider their potential interactions with human infections. Most reports on potentially community-acquired *A. baumannii* have been published during the last 15 years. They concern community-acquired pneumonia, infections in survivors from natural disasters, and infected war wounds in troops from Iraq and Afghanistan. Although the existence of extra-hospital reservoirs of *A. baumannii* has long been disputed, the recent implementation of molecular methods has allowed the demonstration of the actual presence of this organism in various environmental locations, in human carriage, in pets, slaughter animals, and human lice. Although the origin of the *A. baumannii* infections in soldiers injured in Southwestern Asia is difficult to determine, there are some arguments to support the involvement of extra-hospital reservoirs in the occurrence of community-acquired infections. Overall, the emergence of community-acquired *A. baumannii* infections could be associated with interactions between animals, environment, and humans that are considered to be potentially involved in the emergence or re-emergence of some infectious diseases.

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## 1. Introduction

*Acinetobacter baumannii* has emerged as an important nosocomial pathogen, and nosocomial infection with this organism has been associated with increased patient morbidity, mortality, and health care costs.<sup>1–3</sup> Recent studies have improved our knowledge about the virulence of *A. baumannii* by using animal models, adherence to cell cultures, and phenotypic and molecular studies of the bacteria.<sup>4–10</sup> This organism has been responsible for numerous hospital outbreaks, presents a remarkable ability to upregulate or acquire resistance determinants,<sup>11</sup> and has a remarkable ability to survive for prolonged periods throughout the hospital environment,<sup>12–14</sup> which can be involved in the duration of certain outbreaks,<sup>15</sup> some of them lasting several years.<sup>16</sup> The cross-transmission of this organism from patient to patient and the possibility of outbreak extension by patient transfer has been demonstrated.<sup>17</sup> In contrast, the existence of an

extra-hospital reservoir of *A. baumannii* and the implication of this potential reservoir in the occurrence of certain infections due to this organism are still controversial.<sup>12,18</sup> It is well established that the members of the genus *Acinetobacter* are considered ubiquitous microorganisms. However, according to most authors, *A. baumannii* being considered ubiquitous or highly prevalent in nature is an important misconception due to the difficulties encountered in accurately identifying this organism.<sup>12,18</sup>

The implementation of molecular methods (e.g. identification of the *bla*OXA-51-like gene by PCR, or the *rpoB* gene by sequencing) has recently improved the identification of *A. baumannii*.<sup>19,20</sup>

The objective of the present report was to review briefly the potentially community-acquired *A. baumannii* infections, to update information on the reservoirs of *A. baumannii* outside the hospital, and to consider their potential interactions with human infections.

2. Potentially community-acquired *A. baumannii* infections

Although the first reports of respiratory community-acquired *Acinetobacter* infections were published in 1977 in two alcoholic

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patients<sup>21</sup> and in 1981 in three foundry workers,<sup>22</sup> most reports concerning potentially community-acquired *A. baumannii* infections have been published during the last 15 years.

*A. baumannii* is increasingly recognized as an uncommon but important cause of community-acquired pneumonia.<sup>23–26</sup> In addition, most of these infections have occurred during the warmest and wettest seasons.<sup>26</sup> This strongly marked seasonal and geographical distribution could be related to some climatic or environmental features involved in the acquisition of these bacteria by patients. Other studies have demonstrated summer peaks and temperature-associated increases in Gram-negative infections among hospitalized patients.<sup>27,28</sup> According to a time-series analysis reported in a study conducted at the University of Maryland Medical Center, the increase in the monthly *A. baumannii* infection rate was 17% for each 10 °F increase in outdoor temperatures.<sup>27</sup> According to Anstey et al.,<sup>23</sup> community-acquired pneumonia could occur following microaspiration of *A. baumannii* in patients with preceding throat colonization with this species.

*A. baumannii* is one of the microorganisms that are most commonly isolated in wound cultures from survivors of natural disasters. It has been demonstrated in earthquakes<sup>29,30</sup> and a tsunami.<sup>31</sup> In the Wenchuan (China) earthquake that occurred in 2008, *A. baumannii* was isolated from wounds, sputum, and blood of some injured persons.<sup>30</sup>

Since 2003 the incidence of *A. baumannii* infection in US military hospitals has increased, primarily among wounded troops from Southwest Asia.<sup>32</sup> A microbiological study concerning war wounds in US troops from Iraq and Afghanistan upon arrival at the National Naval Medical Center (USA) demonstrated that *A. baumannii* was the most prevalent organism isolated, representing 63.0% of all isolates.<sup>33</sup> Similarly, bacteria belonging to the *A. calcoaceticus*–*baumannii* complex (ABC) were the most prevalent microorganisms isolated in the cultures obtained from casualties coming from Iraq and Afghanistan who had open tibial fractures that were complicated by infection.<sup>34</sup>

Overall, studies reporting community-acquired *A. baumannii* infections are based on single events or case series. It is noticeable that, to the best of our knowledge, no study estimating the evolution of the incidence of community-acquired *A. baumannii* infections has been published. Moreover, in most published studies, details concerning the methods used for bacterial identification are limited. Considering the difficulties encountered in the identification of *A. baumannii*, this lack of information is an important limitation for the interpretation of those study results.

### 3. Extra-hospital reservoirs of *A. baumannii*

*A. baumannii* has been isolated from various environmental locations. Indeed, it has been recovered from soil contaminated with petroleum hydrocarbons in countries presenting different climatic conditions, like India<sup>35</sup> and France.<sup>36</sup> A lot of *Acinetobacter* species, including *A. baumannii*, are able to degrade diesel fuel.<sup>37</sup> Among 66 soil samples analyzed in Hong Kong, more than a third contained *Acinetobacter* sp, with *A. baumannii* representing 23% of these bacteria.<sup>38</sup> *A. baumannii* has also been isolated from vegetables collected in supermarkets, greengrocers, and private gardens.<sup>39</sup> Lastly, a recent study conducted in South Korea recorded the presence of *A. baumannii* on inanimate surfaces that are often in contact with humans, like tables in parks and a game console.<sup>40</sup> *A. baumannii* was also recovered from manured agricultural soil and pig slurry in the UK,<sup>41</sup> and in aquaculture environments (fish and shrimp farms) in Southeast Asia.<sup>42</sup> It is noticeable that for all these environmental studies, molecular techniques for identification (amplified ribosomal DNA restriction analysis and analysis of the 16S rRNA gene) were used.

Data concerning human community carriage of *A. baumannii* are scarce. A study performed in New York City reported that 10.4% of community residents carried *A. baumannii* on their hands.<sup>43</sup> A prevalence study conducted among 102 healthy nondeployed US Army soldiers in training reported that 17 individuals (17%) carried ABC on their skin (forehead or feet).<sup>44</sup> Concurrently, a study performed by the same authors among US Army soldiers stationed in Iraq failed to find any skin carrier.<sup>45</sup> Previous surveys in Europe have also found the rates of *A. baumannii* skin carriage to be very low (<1%).<sup>46,47</sup>

During the last decade, *A. baumannii* strains have been isolated from animals. Most of the studies reporting animal cases have been conducted in veterinary clinics or hospitals.<sup>48–51</sup> Nosocomial spread has been described.<sup>49</sup> The *A. baumannii* infections were reported in horses and pets, with a slight predominance in wound infections and abscesses. For those studies performed in animal health care settings, it is difficult to demonstrate the extra-hospital origin of *A. baumannii*. However animals can still be colonized after discharge, then constituting a reservoir for potential transmissions to the environment, other animals, or humans.

In a study conducted in Scotland,<sup>52</sup> pigs and cattle slaughtered for human consumption and coming from different farms were sampled (feces, skin, nostril, ear) for *A. baumannii* isolation. The prevalence of *A. baumannii* carriage was 1.2%. The 16 *A. baumannii* isolated were grouped into three different clusters, but had different pulsed-field gel electrophoresis (PFGE) patterns compared with human isolates of the three major European clones ECI, ECII, and ECIII. Multi-resistant strains have been identified in other studies. Zordan et al.<sup>53</sup> reported that the 56 *A. baumannii* isolates obtained from several German veterinary clinics were highly resistant to antibiotics, a large proportion of them were genetically congruent with the European clones I, II, or III, and most of them belonged to the same cluster. These findings indicate that, as in human medicine, *A. baumannii* is also an emerging opportunistic pathogen in veterinary medicine, especially in hospitalized animals. However, no cohort or case–control study has investigated if contact of humans with animals increases the risk of acquiring *A. baumannii* infections.

Apart from vertebrate animals, *A. baumannii* strains have also been identified in human lice. La Scola and Raoult<sup>54</sup> demonstrated that the average prevalence of *A. baumannii* DNA among 622 body lice sampled from various continents (Europe, Africa, and South America) varied with a range from 3% to 58%. A recent study conducted in Ethiopia showed the presence of *A. baumannii* in human body lice and also head lice.<sup>55</sup> In another recent study conducted in Paris,<sup>56</sup> *A. baumannii* was isolated in 33% of head lice from elementary schoolchildren. However, whether lice could be a preferential host for *A. baumannii* has not been established.

### 4. Potential interactions between extra-hospital reservoirs and infections

According to a study recorded in Singapore, *A. baumannii* was found to colonize burns very early in the course of the infected patient's hospital stay (median 2 days).<sup>57</sup> Therefore, the importation from the community of some *A. baumannii* strains by the patient is a possibility not to be overlooked. This hypothesis is also supported by the small size of the outbreaks that occurred in this unit during the study. Indeed, the largest one included only five patients, indicating a low level of cross-transmission. Moreover, this outbreak could be traced to a patient following the Jakarta Marriott terrorist bombing, underscoring its community origin.<sup>57</sup>

The origin of the *A. baumannii* infections in soldiers injured in Afghanistan and Iraq is difficult to determine. The infections may have been acquired from environmental sources in the field, during evacuation from the operating theatres to the military field

hospitals, within these military field hospitals, or during treatment at hospitals after evacuation outside of the conflict area. In a study of skin and soft tissue infections due to *A. baumannii* identified among American soldiers and Iraqi nationals in a hospital ship, the mean time to diagnosis of the infection was 12.8 days, a duration that is more consistent with nosocomial acquisition.<sup>58</sup> Based on the absence of a correlation between the geographic location of the traumatic injury and the genotype of *A. baumannii*, Petersen et al. suggested that the environment at the time of wounding was an unlikely source of infection.<sup>59</sup> Concurrently, several studies showed that at the time of presentation to the hospital, higher rates of multi-resistant ABC infections or colonizations were observed in the Iraqi non-US patient population than in US patients.<sup>60–62</sup> The observations of Ake et al., which were recorded at the beginning of the casualty evacuation chain, suggest that multi-resistant ABC colonization or infection of host nation patients exists before their entry into deployed US military facilities. Therefore, this could at least partially reflect community carriage of these organisms.<sup>60</sup>

The role of a previous colonization with *A. baumannii* in the development of community-acquired infections is supported by the difference in rates of *A. baumannii* carriage identified in non-hospitalized patients (some high rates of skin carriage (4%) during the wet and warm season in Hong Kong<sup>63</sup> as compared to the low rates (<1%) of skin or fecal colonization described in Europe), which is consistent with the seasonal differences recorded for community-acquired *A. baumannii* pneumonia in Southeast Asia and their scarcity or their absence in Europe. Further studies are needed to determine whether lice can act as a hidden extra-human reservoir for bacteria and can promote the spread of *A. baumannii* among humans. In a broader perspective, it would be useful to study the potential role of other vectors (cockroaches, flies, etc.) in *A. baumannii* transmission.

The presence of strains belonging to the European clones I–III in animals raises concerns about the possibility of spread from humans to animals and vice versa. In addition, PCR and DNA sequencing for various genes of resistance to antibiotics demonstrated that the molecular backgrounds of resistance to aminoglycosides and quinolones and the hyperproduction of chromosomal beta-lactamases had patterns common to those described in *A. baumannii* of human origin.<sup>52</sup>

In conclusion, it has been demonstrated by molecular identification that *A. baumannii* is actually present outside the hospital. Several arguments are consistent with the involvement of extra-hospital reservoirs of *A. baumannii* in the occurrence of community-acquired infections. However additional studies are needed for a better estimation of the incidence of community-acquired *A. baumannii* infections and a better understanding of the mechanisms of interactions between the different potential reservoirs and humans.

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## References

1. Gaynes R, Edwards JR. Overview of nosocomial infections caused by Gram-negative bacilli. *Clin Infect Dis* 2005;**41**:848–54.
2. Lee NY, Lee HC, Ko NY, Chang CM, Shih HI, Wu CJ, et al. Clinical and economic impact of multidrug resistance in nosocomial *Acinetobacter baumannii* bacteremia. *Infect Control Hosp Epidemiol* 2007;**28**:713–9.
3. Falagas ME, Rafailidis PI. Attributable mortality of *Acinetobacter baumannii*: no longer a controversial issue. *Crit Care* 2007;**11**:134.
4. Eveillard M, Soltner C, Kempf M, Saint-André JP, Lemarié C, Randrianarivelo C, et al. The virulence variability of different *Acinetobacter baumannii* strains in experimental pneumonia. *J Infect* 2010;**60**:154–61.
5. De Brij A, Eveillard M, Dijkshoorn L, van den Broek PJ, Nibbering PH, Joly-Guillou ML. Differences in *Acinetobacter baumannii* strains and host innate immune response determine morbidity and mortality in experimental pneumonia. *PLoS One* 2012;**7**:e30673.
6. Vallenet D, Nordmann P, Barbe V, Poirel L, Mangenot S, Bataille E, et al. Comparative analysis of acinetobacters: three genomes for three lifestyles. *PLoS One* 2008;**19**:e1805.
7. Adams MD, Goglin K, Molyneaux N, Hujer KM, Lavender H, Jamison JJ, et al. Comparative genome sequence analysis of multidrug-resistant *Acinetobacter baumannii*. *J Bacteriol* 2008;**190**:8053–64.
8. Lee HW, Kah YM, Kim J, Lee JC, Seol SY, Cho DT, Kim J. Capacity of multidrug-resistant clinical isolates of *Acinetobacter baumannii* to form biofilm and adhere to epithelial cell surfaces. *Clin Microbiol Infect* 2008;**14**:49–54.
9. Cehavir N, Demir M, Kaleli I, Gurbuz M, Tikvesli S. Evaluation of biofilm production, gelatinase activity, and mannose-resistant haemagglutination in *Acinetobacter baumannii* strains. *J Microbiol Immunol Infect* 2008;**41**:513–8.
10. de Brij A, Gaddy J, van der Meer J, Koning R, Koster A, van den Broek P, et al. CsuA/BABCDE-dependent pili are not involved in the adherence of *Acinetobacter baumannii* ATCC19606(T) to human airway epithelial cells and their inflammatory response. *Res Microbiol* 2009;**160**:213–8.
11. Higgins PG, Dammhayn C, Hackel M, Seifert H. Global spread of carbapenem-resistant *Acinetobacter baumannii*. *J Antimicrob Chemother* 2009;**65**:233–8.
12. Peleg AY, Seifert H, Paterson DL. *Acinetobacter baumannii*: emergence of a successful pathogen. *Clin Microbiol Rev* 2008;**21**:538–82.
13. Thom KA, Johnson JK, Lee MS, Harris AD. Environmental contamination because of multidrug-resistant *Acinetobacter baumannii* surrounding colonized or infected patients. *Am J Infect Control* 2011;**39**:711–5.
14. Weber DJ, Rutal WA, Miller MB, Huslage K, Sickbert-Bennett E. Role of hospital surfaces in the transmission of emerging health care-associated pathogens: norovirus, *Clostridium difficile*, and *Acinetobacter* species. *Am J Infect Control* 2010;**38**:S25–33.
15. Hong KB, Oh HS, Song JS, Lim JH, Kang DK, Son IS, et al. Investigation and control of an outbreak of imipenem-resistant *Acinetobacter baumannii* infection in a pediatric intensive care unit. *Pediatr Infect Dis J* 2012;**31**:685–90.
16. Barnaud G, Zihoune N, Ricard JD, Hippeaux MC, Eveillard M, Dreyfuss D, et al. Two sequential outbreaks caused by multidrug-resistant *Acinetobacter baumannii* isolates producing OXA-58 or OXA-72 oxacillinase in an intensive care unit in France. *J Hosp Infect* 2010;**76**:358–60.
17. Naas T, Coignard B, Carbonne A, Blanckaert K, Bajolet O, Bernet C, et al. VEB-1 extended-spectrum beta-lactamase-producing *Acinetobacter baumannii*, France. *Emerg Infect Dis* 2006;**12**:1214–22.
18. Towner KJ. *Acinetobacter*: an old friend, but a new enemy. *J Hosp Infect* 2009;**73**:355–63.
19. Turton JF, Woodford N, Glover J, Yarde S, Kaufmann ME, Pitt TL. Identification of *Acinetobacter baumannii* by detection of blaOXA-51-like carbapenemase gene intrinsic to this species. *J Clin Microbiol* 2006;**44**:2974–6.
20. Gundi VA, Diskshoorn L, Burignat S, Raoult D, La Scola B. Validation of partial *rpoB* gene sequence analysis for the identification of clinically important and emerging *Acinetobacter* species. *Microbiology* 2009;**155**:2333–41.
21. Goodhart GL, Abrutyn E, Watson R, Root RK, Egert J. Community-acquired *Acinetobacter calcoaceticus* var *anitratus* pneumonia. *JAMA* 1977;**238**:1516–8.
22. Cordes LG, Brink EW, Checko PJ, Lentnek A, Lyons RW, Hayes PS, et al. A cluster of *Acinetobacter* pneumonia in foundry workers. *Ann Intern Med* 1981;**95**:688–93.
23. Anstey NM, Currie BJ, Hassell M, Palmer D, Dwyer B, Seifert H. Community-acquired bacteremic *Acinetobacter* pneumonia in tropical Australia is caused by diverse strains of *Acinetobacter baumannii*, with carriage in the throat in at-risk groups. *J Clin Microbiol* 2002;**40**:685–6.
24. Leung WS, Chu CM, Tsang KY, Lo FH, Lo KF, Ho PL. Fulminant community-acquired *Acinetobacter baumannii* pneumonia as a distinct clinical syndrome. *Chest* 2006;**129**:102–9.
25. Chen MZ, Hsueh PR, Lee LN, Yu CJ, Yang PC, Luh KT. Severe community pneumonia due to *Acinetobacter baumannii*. *Chest* 2001;**120**:1072–7.
26. Ong CW, Lye DC, Khoo KL, Chua GS, Yeoh SF, Lea YS, et al. Severe community-acquired *Acinetobacter baumannii* pneumonia: an emerging highly lethal infectious disease in the Asia-Pacific. *Respirology* 2009;**14**:1200–5.
27. Perencevitch EN, McGregor JC, Shardell M, Furuno JP, Harris AD, Morris Jr JG, et al. Summer peaks in the incidences of Gram-negative bacterial infection among hospitalized patients. *Infect Control Hosp Epidemiol* 2008;**29**:1124–31.
28. Eber MR, Shardell M, Schweizer ML, Laxminarayan R, Perencevitch EN. Seasonal and temperature-associated increases in Gram-negative bacterial bloodstream infections among hospitalized patients. *PLoS One* 2011;**6**:e25298.
29. Öncül O, Keskin Ö, Acar HV, Küçükardalı Y, Evrenkaya R, Atasoyu EM, et al. Hospital-acquired infections following the 1999 Marmara earthquake. *J Hosp Infect* 2002;**51**:47–51.
30. Wang Y, Hao P, Lu B, Yu H, Huang W, Hongliang H, Dai K. Causes of infection after earthquake, China, 2008. *Emerg Infect Dis* 2010;**16**:974–5.
31. Uçkay I, Sax H, Harbarth S, Bernard L, Pittet D. Multi-resistant infections in repatriated patients after natural disasters: lessons learned from the 2004 tsunami for hospital infection control. *J Hosp Infect* 2008;**68**:1–8.
32. Centers for Disease Control and Prevention (CDC). Centers for disease control: *Acinetobacter baumannii* infections among patients at military medical facilities treating injured US service members, 2002–2004. *MMWR Morb Mortal Wkly Rep* 2004;**53**:1063–6.
33. Sheppard FR, Keiser P, Craft DW, Gage F, Robson M, Brown TS, et al. The majority of US combat casualty soft-tissue wounds are not infected or colonized upon arrival or during treatment at a continental US military medical facility. *Am J Surg* 2010;**200**:489–95.

34. Johnson EN, Burns TC, Hayda RA, Hospenthal DR, Murray CK. Infectious complications of open type III tibial fractures among combat casualties. *Clin Infect Dis* 2007;**45**:409–15.
35. Sarma PM, Bhattacharya D, Krishnan S, Lal B. Assessment of intra-species diversity among strains of *Acinetobacter baumannii* isolated from sites contaminated with petroleum hydrocarbons. *Can J Microbiol* 2004;**50**:405–14.
36. Bordenave S, Goñi-Urriza MS, Caumette P, Duran R. Effects of heavy fuel oil on the bacterial community structure of a pristine microbial mat. *Appl Environ Microbiol* 2007;**73**:6089–97.
37. Mara K, Decorosi F, Viti C, Giovannetti L, Papaleo MC, Maida I, et al. Molecular and phenotypic characterization of *Acinetobacter* strains able to degrade diesel fuel. *Res Microbiol* 2012;**163**:161–72.
38. Houang ET, Chu YW, Leung CM, Chu KY, Berlau J, Ng KC, et al. Epidemiology and infection control implications of *Acinetobacter* sp. in Hong Kong. *J Clin Microbiol* 2001;**39**:228–34.
39. Berlau J, Aucken HM, Houang E, Pitt TL. Isolation of *Acinetobacter* spp including *A. baumannii* from vegetables: implications for hospital-acquired infections. *J Hosp Infect* 1999;**42**:201–4.
40. Choi JY, Kim Y, Ko EA, Park YK, Jheong WH, Ko G, et al. *Acinetobacter* species isolates from a range of environments: species survey and observations of antimicrobial resistance. *Diagn Microbiol Infect Dis* 2012;**74**:177–80.
41. Byrne-Bailey KG, Gaze WH, Kay P, Boxall ABA, Hawkey PM, Wellington EM. Prevalence of sulphonamide resistance genes in bacterial isolates from manured agricultural soils and pig slurry in the United Kingdom. *Antimicrob Agents Chemother* 2009;**53**:696–702.
42. Huys G, Bartie K, Cnockaert M, Hoang Oahn DT, Phuong NT, Somsiri T, et al. Biodiversity of chloramphenicol-resistant mesophilic heterotrophs from Southeast Asian aquaculture environments. *Res Microbiol* 2007;**158**:228–35.
43. Zeana C, Larson E, Sahni J, Bayuga SJ, Wu F, Della-Latta P. The epidemiology of multidrug-resistant *Acinetobacter baumannii*: does the community represent a reservoir? *Infect Control Hosp Epidemiol* 2003;**24**:275–9.
44. Griffith ME, Ceremuga JM, Ellis MW, Guymon CH, Hospenthal DR, Murray CK. *Acinetobacter* skin colonization of US Army soldiers. *Infect Control Hosp Epidemiol* 2006;**27**:659–61.
45. Griffith ME, Lazarus DR, Mann PB, Boger JA, Hospenthal DR, Murray CK. *Acinetobacter* skin carriage among US Army soldiers deployed in Iraq. *Infect Control Hosp Epidemiol* 2007;**28**:720–2.
46. Dijkshoorn L, van Aken E, Shunburne L, van der Reijden TJ, Bernards AT, Nemeč A, et al. Prevalence of *Acinetobacter baumannii* and other *Acinetobacter* spp. in faecal samples from non-hospitalized patients. *Clin Microbiol Infect* 2005;**11**:329–31.
47. Seifert H, Dijkshoorn L, Gerner-Smidt P, Pelzer N, Tjernberg I, Banechoutte M. Distribution of *Acinetobacter* species on human skin: comparison of phenotypic and genotypic identification methods. *J Clin Microbiol* 1997;**35**:2819–25.
48. Vanechoutte M, Devriese LA, Dijkshoorn L, Lamote B, Deprez P, Verschraegen G, et al. *Acinetobacter baumannii*-infected vascular catheters collected from horses in an equine clinic. *J Clin Microbiol* 2000;**38**:4280–1.
49. Francey T, Gaschen F, Nicolet J, Burnens AP. The role of *Acinetobacter baumannii* as a nosocomial pathogen for dogs and cats in an intensive care unit. *J Vet Intern Med* 2000;**14**:177–83.
50. Brachelente C, Wiener D, Malik Y, Huessy D. A case of necrotizing fasciitis with septic shock in a cat caused by *Acinetobacter baumannii*. *Vet Dermatol* 2007;**18**:432–8.
51. Endimiani A, Hujer KM, Hujer AM, Bertschy I, Rossano A, Koch C, et al. *Acinetobacter baumannii* isolates from pets and horses in Switzerland: molecular characterization and clinical data. *J Antimicrob Chemother* 2011;**66**:2248–54.
52. Hamouda A, Findlay J, Al Hassan L, Amyes SG. Epidemiology of *Acinetobacter baumannii* of animal origin. *Int J Antimicrob Agents* 2011;**38**:314–8.
53. Zordan S, Prenger-Berninghoff E, Weiss R, van der Reijden T, van den Broeck P, Baljer G, et al. Multidrug-resistant *Acinetobacter baumannii* in veterinary clinics, Germany. *Emerg Infect Dis* 2011;**17**:1751–4.
54. La Scola B, Raoult D. *Acinetobacter baumannii* in human body louse. *Emerg Infect Dis* 2004;**10**:1671–3.
55. Kempf M, Alemseged A, Diatta G, Trape JF, Angelakis E, Mediannikov O, et al. Detection of *Acinetobacter baumannii* in human head and body lice from Ethiopia and identification of new genotypes. *Int J Infect Dis* 2012;**16**:e680–3.
56. Bouvresse S, Socolovshi C, Berdjane Z, Durand R, Izri A, Raoult D, et al. No evidence of *Bartonella quintana* but detection of *Acinetobacter baumannii* in head lice from elementary schoolchildren in Paris. *Comp Immunol Microbiol Infect Dis* 2011;**34**:475–7.
57. Chim H, Hock Tan B, Song C. Five-year review of infections in a burn intensive care unit: high incidence of *Acinetobacter baumannii* in a tropical climate. *Burns* 2007;**33**:1008–14.
58. Sebeny PJ, Riddle MS, Petersen K. *Acinetobacter baumannii* skin and soft-tissue infection associated with war trauma. *Clin Infect Dis* 2008;**47**:439–43.
59. Petersen K, Cannegieter SC, van der Reijden TJ, van Srijen B, You DM, Babel BS, et al. Diversity and clinical impact of *Acinetobacter baumannii* colonization and infection at a military medical center. *J Clin Microbiol* 2011;**49**:159–66.
60. Ake J, Scott P, Wortmann G, Huang XZ, Barber M, Wang Z, et al. Gram-negative multidrug-resistant organism colonization in a US military healthcare facility in Iraq. *Infect Control Hosp Epidemiol* 2011;**32**:545–52.
61. Yun HC, Murray CK, Roop SA, Hospenthal DR, Gouridine E, Dooley DP. Bacteria recovered from patients admitted to a deployed U.S. military hospital in Baghdad, Iraq. *Mil Med* 2006;**171**:821–5.
62. Scott P, Deye G, Srinivasan A, Murray C, Moran K, Hulten E, et al. An outbreak of multidrug-resistant *Acinetobacter baumannii*-calcoaceticus complex infection in the US military health care system associated with military operations in Iraq. *Clin Infect Dis* 2007;**44**:1577–84.
63. Chu YW, Leung CM, Houang AT, Ng KC, Leung CB, Leung HY, et al. Skin carriage of *Acinetobacter* in Hong Kong. *J Clin Microbiol* 1999;**37**:2962–7.