Acinetobacter baumannii in Human Body Louse

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While we were isolating Bartonella quintana from body lice, 40 Acinetobacter baumannii strains were also isolated and genotyped. One clone was unique and the other was ampicillin susceptible. A. baumannii DNA was later detected in 21% of 622 lice collected worldwide. These findings show an A. baumannii epidemic in human body lice.

The body louse has been demonstrated to be the vector of three human pathogens: Rickettsia prowazekii, the agent of epidemic typhus; Bartonella quintana, the agent of trench fever; and Borrelia recurrentis, the agent of louseborne recurrent fever (1). While trying to isolate Bartonella quintana from body lice of homeless persons in Marseille, we isolated six Acinetobacter spp. (2) subsequently identified as A. baumannii. They were susceptible to ampicillin, whereas Acinetobacter are almost always resistant to ampicillin in France (3). We further isolated other A. baumannii from body lice in Marseille and now have 40 isolates; 21 are susceptible to ampicillin. To investigate the possibility of a clonal diffusion in lice, the recA gene sequence of isolates was determined and compared to that of the collection and strains. To test if the body louse–A. baumannii association is observed worldwide, we investigated the presence of A. baumannii DNA in a large collection of body lice.

The Study

The 40 body lice–associated A. baumannii were obtained during studies of homeless shelters in Marseille (4). The procedure for isolation of these strains has been described previously (2). Provisional identification of isolates was based on Gram stain and results of oxidase test and API 20NE identification strip (Biomerieux, Marcy l’Etoile, France). We also tested the 19 strains of A. baumannii available at the CIP (Institut Pasteur, Paris, France) and 3 clinical strains isolated in our laboratory during the same period (Table 1). Bacteria were routinely grown at 37°C with 5% CO₂ on Columbia sheep blood agar (Biomerieux). The recA gene amplification was performed with specific primers rA1 (5′-CCTGAATCTTCTCSGTAAAAC-3′) and rA2 (5′-GTGTTCGGGCTGCCCACATTAC-3′), as described previously (5). All sequences were manually edited, and all ambiguous parts were amplified and sequenced again. Amplifying the recA gene allowed unambiguous determination of the sequence of a 336-bp fragment for all isolates. Variation in nucleotides occurred at 10 positions and determined eight genotypes (Table 1), which have been deposited in the GenBank database with the following accession no.: 1, AY274826; 2, AY274827; 3, AY274828; 4, AY274829; 5, AY274830; 6, AY274831; 7, AY274832; 8, AY274833. The translated protein sequences were all identical, except for genotype 2, in which a valine was replaced by an isoleucine at position 68. recA types 1 and 2 were isolated from body louse–associated A. baumannii; for the 21 collection strains, seven genotypes were observed. Genotype 2 was unique to body louse–associated A. baumannii. Genotype 1 was associated with susceptibility to ampicillin in body louse–associated A. baumannii and was common to seven collection strains. However, all collection strains, whatever the genotype, were resistant to ampicillin, even strains of the Unité des Rickettsies that have the same geographic origin as the body louse A. baumannii.

We then tested a large collection of body lice for A. baumannii DNA. We tested by polymerase chain reaction (PCR) a collection of 622 body lice sampled in France, Burundi, Rwanda, Peru, Algeria, Portugal, and the Netherlands (6). Fifty laboratory lice were used as controls. Detection was performed by amplifying the recA gene with A. baumannii–specific primers ACI381F (5′-CACAATGGCATTGCAAGCAATTG-3′) and ACI382R (5′-CCAATTTTCATACGAATCTGG-3′) specifically designed for this study. These primers were previously shown not to produce amplicons from A. calcoaceticus, Acinetobacter genospecies 3, Acinetobacter genospecies 13, A. haemolyticus, A. johnsonii, or A. lwoffi. As control for PCR amplification, we used 18Saigd-18Sbi primer pair, which allows amplification of an 18S RNA gene fragment of arthropods. Consensus forward primer 18Saigd (5′-TCTGGTGATCCTGCCCAGTA-3′) was

| Table 1. Types of recA gene sequences* |
|--------------------------------------|
| **Acinetobacter baumannii** strains | recA type | Ampicillin susceptibility |
| (n = 62)                            |           |                          |
| Lice associated (n = 21)             | 1         | Yes                      |
| Lice associated (n = 19)             | 2         | No                       |
| CIP 70.34, CIP 70.32, UR 121120, CIP | 1         | No                       |
| 70.8, CIP 70.9, CIP 70.33, UR 73415 |           |                          |
| CIP 54.147, CIP 70.28, CIP 103572,  | 3         | No                       |
| UR 37033, CIP 53.77, CIP 70.22, CIP  |           |                          |
| 105742                              |           |                          |
| CIP 70.24, CIP 68.38                | 4         | No                       |
| CIP 70.10, CIP 70.21                | 5         | No                       |
| CIP 54.97                           | 6         | No                       |
| CIP 53.79                           | 7         | No                       |
| CIP 64.1                            | 8         | No                       |

*CIP, strains from the Collection de l’Institut Pasteur (Paris, France); UR, clinical strains from Unité des Rickettsies.

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body lice were positive for the one described by DeSalle et al. (7). A total of 130 (21%) strains were isolated from Marseille, we sequenced 50 recA amplicons obtained from the lice of different geographic origins (Table 2). Genotypes 1 and 2 were the only ones detected in France; genotype 2 was found in France only. In other parts of the world, genotypes 1, 3, and 4 were observed, with a predominance of genotype 1, similar to the findings in France. Type 4 genotype was the second most common genotype but was absent in European lice. It seems that body louse–associated A. baumannii are oligoclonal, and their distribution is different from that of collection strains. Even if genotype 1 is the most common in all cases, genotypes 2 and 4 are overrepresented in body louse–associated strains. However, contrary to culture after body lice decontamination, we cannot rule out that A. baumannii infection occurred through external contamination.

**Conclusions**

The genotype of the 40 A. baumannii from Marseille from the body lice of homeless persons are limited to two clones; one is exclusively associated with strains caused by body lice, and the other is associated with ampicillin susceptibility in body louse–associated strains. This finding shows an A. baumannii epidemic in body lice. A. baumannii is mainly implicated in cases of hospital-acquired infections but has also been reported as a cause of severe community-acquired infections, including pneumonia, endocarditis, and meningitis, mostly in persons who are alcoholics (8). While ingesting only blood from humans, the louse has a sterile midgut, and the presence of bacteria is likely caused by the louse’s ingesting contaminated blood (2). Moreover, previous studies have shown that A. baumannii is not a common skin-associated Acinetobacter in Europe, unlike in tropical areas, since it is found on the skin of <1.5% of healthy persons (9). Our results indicate that association of A. baumannii with body lice is likely caused by undiagnosed transient A. baumannii bacteremia in patients harboring body lice; however, because the frequency of skin association of A. baumannii in the homeless subpopulation is unknown, contamination from body lice cannot be ruled out. Relatively low-virulence flora, such as Staphylococcus epidermidis or diphtheroids, may be destroyed by leukocytes, antibody, and complement in the blood meal, whereas the more virulent bacteria, such as A. baumannii, could survive because they resist the defense mechanism of the blood meal and those of the body lice. However, we never isolated S. aureus from lice, which is a virulent bacterium and known to be a common skin commensal agent. From preliminary work, we have observed that body lice may be infected by several bacterial species (L. Houamdi, unpublished data) and that the occurrence of body louse–transmitted disease occurs because causative bacteria (B. quintana, R. prowazekii, and B. recurrentis) induce relapsing bacteremia rather than specifically adapting to body lice (10). Finally, if our hypothesis of A. baumannii bacteremia in patients harboring body lice is true, their clinical manifestations in homeless persons remain to be determined.

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