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have a key homologous recombination role in VlsE variation within *B. burgdorferi* [4]. RecA mutant strains of *B. burgdorferi* were still able to undergo mosaic VlsE lipoprotein variation, so RecA would appear not to have a role in the recombination events utilized for antigenic variation in *Borrelia*, in contrast to other organisms, such as *Neisseria gonorrhoeae* [5].

Furthermore, possession of non-functional RecA has been shown not to have any deleterious effects on the infectiousness of *B. burgdorferi*. This is in contrast to the lethality of RecA mutations hypothesized by others [6]. However, this strain showed a loss in ability to cause joint infections [4]. These findings suggest that loss of RecA may not affect the viability of the closely related relapsing fever spirochaetes, but could account for differences in clinical consequences. Indeed, clinical differences are seen between these infections, with *B. recurrentis* resulting in significant jaundice, petechiae, epistaxis and major organ involvement (central nervous system and cardiac), and *B. duttonii* being associated with pregnancy complications and high perinatal mortality [7]. The obvious difference between these two spirochaetes is their ability to be either tick-borne or louse-borne, resulting in either local endemic or epidemic disease, respectively. It is unlikely that RecA is not necessary among louse-borne pathogens, as RecA remains functional in *Rickettsia prowazekii*, another louse-borne pathogen whose genome is also subject to degradation as compared with non-louse-borne counterparts [8]. Whether the truncation of RecA within *B. recurrentis* plays a role in the differing clinical presentations or vectorial capabilities of these spirochaetes remains to be determined. What is apparent is that samples from louse-borne relapsing fever patients showed the same 'signature' SNPs, differentiating them from *B. duttonii*, including the premature stop codon, thus suggesting a clonal ancestry.

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Transparency Declaration

The authors declare no conflict of interests.

References

1. Lescot M, Audic S, Robert C *et al.* The genome of *Borrelia recurrentis*, the agent of deadly louse-borne relapsing fever, is a degraded subset of tick-borne *Borrelia duttonii*. *PLoS Genet* 2008; 4: e1000185.
2. Cutler SJ, Bonilla EM, Singh R. Insights into the population structure of East African relapsing fever *Borrelia*. *Emerg Infect Dis* 2010; [Epub ahead of print]. In review.
3. Scott JC, Wright DJM, Cutler SJ. Typing African relapsing fever spirochetes. *Emerg Infect Dis* 2005; 11: 1722–1729.
4. Liveris D, Mulay V, Sandigursky S, Schwartz I. *Borrelia burgdorferi* vlsE antigenic variation is not mediated by RecA. *Infect Immun* 2008; 76: 4009–4018.
5. Helm RA, Seifert HS. Pilin antigenic variation occurs independently of the Recbcd pathway in *Neisseria gonorrhoeae*. *J Bacteriol* 2009; 191: 5613–5621.
6. Putteet-Driver AD, Zhong J, Barbour AG. Transgenic expression of *recA* of the spirochetes *Borrelia burgdorferi* and *Borrelia hermsii* in *Escherichia coli* revealed differences in DNA repair and recombination phenotypes. *J Bacteriol* 2004; 186: 2266–2274.
7. Cutler SJ, Abdissa A, Trape J-F. New concepts for the old challenge of African relapsing fever borreliosis. *Clin Microbiol Infect* 2009; 15: 400–406.
8. Dunkin SM, Wood DO. Isolation and characterization of the *Rickettsia prowazekii recA* gene. *J Bacteriol* 1994; 176: 1777–1781.

Molecular epidemiological investigation of multidrug-resistant *Acinetobacter baumannii* strains in four Mediterranean countries with a multilocus sequence typing scheme

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Abstract

Thirty-five multidrug-resistant *Acinetobacter baumannii* strains, representative of 28 outbreaks involving 484 patients from 20 hospitals in Greece, Italy, Lebanon and Turkey from 1999 to 2009, were analysed by multilocus sequence typing. Sequence type (ST)2, ST1, ST25, ST78 and ST20 caused 12, four, three, three and two outbreaks involving 227, 93, 62, 62 and 31 patients, respectively. The genes $bla_{oxa\text{-}58}$, $bla_{oxa\text{-}23}$ and $bla_{oxa\text{-}72}$ were found in 27, two and one carbapenem-resistant strain, respectively. In conclusion, *A. baumannii* outbreaks were caused by the spread of a few strains.

Keywords: *Acinetobacter baumannii*, carbapenemases, molecular epidemiology, multilocus sequence typing, pulsed-field gel electrophoresis

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Acinetobacter baumannii epidemics have been recently described in Europe, and are caused worldwide by a limited number of genotypic clusters of strains [1–14]. Major *A. baumannii* outbreak clones were initially named European clones I to III, but are now regarded as international [14]. Multilocus sequence typing (MLST) is the standard method for defining the clonal structure of bacterial species, and has defined clones 1–3 as clonal complexes (CCs) 1–3 [14–16]. The aims of the present study were: (i) using MLST, to define the genetic identity of *A. baumannii* strains associated with outbreaks in Mediterranean hospitals; (ii) to compare MLST data with those obtained using pulsed-field gel electrophoresis (PFGE) and trilocus sequence-based typing (3LST) [17]; and (iii) to identify the genes for carbapenem-hydrolysing β-lactamases involved in these outbreaks.

Thirty-five *A. baumannii* strains isolated during 28 outbreaks that occurred in 20 hospitals in Greece, Italy, Lebanon and Turkey from 1999 to 2009 were included in the study. Nearly all strains were representative of cross-trans-

TABLE 1. Epidemiological, phenotypic and genotypic data of the *Acinetobacter baumannii* isolates included in the study

					MLST									Carbapenem resistance		
					Allele											
Strain	Hospital	Year	Patients[a]	PFGE type	*cpn60*	*fusA*	*gltA*	*pyrG*	*recA*	*rplB*	*rpoB*	ST	3LST group	IMP MIC	CHDL	MBL
700	F-Naples/IT	1999	81	A	1	1	1	1	5	1	1	1	2	0.5		
3891	Thessaloniki/GR	2000	3	B	1	1	1	1	5	1	1	1	2	16	OXA-58	
3886	Athens/GR	2005	4	B	1	1	1	1	5	1	1	1	2	16	OXA-58	
3887	Serres/GR	2006	5	C	1	1	1	1	5	1	1	1	2	32	OXA-58	VIM-4
2979	Agrigento/IT	2002	14	D	3	1	1	1	5	1	1	20	2	0.5		
3130	SG-Beirut/LB	2004	17	E	3	1	1	1	5	1	1	20	2	16	OXA-58	
2105	F-Naples/IT	2002	43	F	2	2	2	2	2	2	2	2	1	16	OXA-58	
2638	M-Naples/IT	2003	42	F	2	2	2	2	2	2	2	2	1	16	OXA-58	
3892	Thessaloniki/GR	2003	23	F	2	2	2	2	2	2	2	2	1	16	OXA-58	
3893	Larissa/GR	2004	48	F	2	2	2	2	2	2	2	2	1	16	OXA-58	
3894	Serres/GR	2006	19	F	2	2	2	2	2	2	2	2	1	32	OXA-58	VIM-1
4245	Pozzuoli/IT	2009	4	F	2	2	2	2	2	2	2	2	1	16	OXA-58	
2735	M-Naples/IT	2004	2	F1	2	2	2	2	2	2	2	2	1	64	OXA-58	
3858	Catania/IT	2004	27	F2	2	2	2	2	2	2	2	2	1	2		
3889	Athens/GR	2005	4	G	2	2	2	2	2	2	2	2	1	16	OXA-58	
4026	SJ-Beirut/LB	2007	5	H	2	2	2	2	2	2	2	2	1	16	OXA-58	
4030	SG-Beirut/LB	2006	6	I	2	2	2	2	2	2	2	2	1	16	OXA-58	
4009	Genoa/IT	2007	4	J	2	2	2	2	2	2	2	2	1	32	OXA-23	
3237	SG-Beirut/LB	2004	1	K	3	3	2	2	3	1	3	3	3	1		
4025	SG-Beirut/LB	2005	3	K	3	3	2	2	3	1	3	3	3	16	OXA-58	
3890	Thessaloniki/GR	2003	12	L	3	3	2	4	7	2	4	25	4	16	OXA-58	
3865	Kocaeli/TK	2005	47	M	3	3	2	4	7	2	4	25	4	64	OXA-23 OXA-58	
4190	M-Naples/IT	2009	3	N	3	3	2	4	7	2	4	25	4	64	OXA-72	
3909	M-Naples/IT	2007	55	O	25	3	6	2	28	1	29	78	6	16	OXA-58	
3696	CT-Naples	2007	4	O	25	3	6	2	28	1	29	78	6	16	OXA-58	
3933	CA-Naples/IT	2007	1	O	25	3	6	2	28	1	29	78	6	2		
4175	CA-Naples/IT	2009	2	O	25	3	6	2	28	1	29	78	6	16	OXA-58	
3868	Izmir/TK	2003	2	P	6	6	8	2	3	5	4	15	5	16	OXA-58	
3869	Istanbul/TK	2003	1	P	6	6	8	2	3	5	4	15	5	16	OXA-58	
3871	Istanbul/TK	2003	1	P1	6	6	8	2	3	5	30	84	5	16	OXA-58	
3872	Izmir/TK	2003	1	P1	6	6	8	2	3	5	30	84	5	16	OXA-58	
3875	Trabzon/TK	2003	1	P1	6	6	8	2	3	5	30	84	5	16	OXA-58	
2978	Agrigento/IT	2001	3	Q	28	3	2	1	4	4	4	82		1		
2977	Agrigento/IT	2001	1	Q	28	3	2	1	4	4	4	82		16	OXA-58	
3866	Kayseri/TK	2003	2	R	26	4	2	2	9	1	4	83		16	OXA-58	

CA-Naples, Cardarelli Hospital, Naples; CT-Naples, Cotugno Hospital, Naples; F-Naples, Federico II University Hospital, Naples; M-Naples, Monaldi Hospital, Naples; SG-Beirut, Saint Gorge Hospital, Beirut; SJ-Beirut, Saint Joseph Hospital, Beirut; GR, Greece; IT, Italy; LB, Lebanon; TK, Turkey; CHDL, class D carbapenem-hydrolysing oxacillinase; IMP, imipenem; MBL, class B metallo-β-lactamase; MLST, multilocus sequence typing; PFGE, pulsed-field gel electrophoresis; ST, sequence type; 3LST, trilocus sequence-based typing.
[a]Number of patients from whom an isolate with each particular PFGE type was detected. Strain 3237 from Beirut, strain 3933 from Naples, strain 2977 from Agrigento and strains 3869, 3871, 3872 and 3875 from Turkey were single isolates in different hospitals.

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mission episodes, and were isolated with identical PFGE types from more than two patients of the same or different institutions (Table 1). Antimicrobial susceptibilities were determined by a reference microdilution method [18]. Although *A. baumannii* strains were not *a priori* selected because of a multidrug resistance phenotype, all of the strains were resistant to more than two of five antimicrobial classes and were considered to be multidrug-resistant [1]; 29 of 35 strains exhibited an imipenem MIC ≥16 mg/L and were considered to be carbapenem-resistant (Table 1). PFGE analysis and interpretation of PFGE profiles were performed as reported previously [6]. Eighteen PFGE types (A–R) and three PFGE subtypes (F1, F2 and P) were identified (Table 1).

MLST analysis was performed as previously described [14] (http://www.pasteur.fr/mlst). Only ten different sequence types (STs) were found. Strains with PFGE profiles A–C, D and E, F–J and L–N were assigned to ST1, ST20, ST2 and ST25, respectively; strains with PFGE profiles K, O, P, P1, Q and R were assigned to ST3, ST78, ST15, ST84, ST82 and ST83, respectively. Interestingly, more than six band differences were found among PFGE profiles A, B and C, among PFGE profiles F, G, H, I and J, and among PFGE profiles L, M and N, respectively, showing that international STs ST1, ST2 and ST25 represent heterogenous genotypic entities. ST2 predominated, being identified in 12 strains from 11 hospitals in Greece, Italy and Lebanon (Table 1 and Fig. 1). Together, these 12 strains represented 12 clusters that involved 227 (46.2% of the total) infected patients. The other most frequently assigned STs were ST1, ST20, ST25 and ST78. They were identified in four, two, three and four strains, respectively, representing 93 (18.9%), 31 (6.3%), 62 (12.6%) and 62 (12.6%) patients. The results are in accordance with previous reports showing that many hospital *A. baumannii* strains circulating in Europe and elsewhere belonged to international clones CC1 and CC2, respectively [1,3,4,7,12,14]. Also, as in the Czech Republic [7], a shift towards ST2 (international clone II) was observed in Greece, Italy and Lebanon. Notably, strains assigned to ST2 and PFGE profile F were observed to progressively overtake, numerically, those assigned to ST1. Indeed, between 1999 and 2006, ST1 represented four strains (93 patients, 23% of the total over this period), whereas between 2002 and 2009, ST2 represented 12 strains (227 patients, 56% of the total over this period; Table 1 and Fig. 1). Moreover, strains assigned to ST2 with PFGE profile F were isolated in three Italian hospitals and

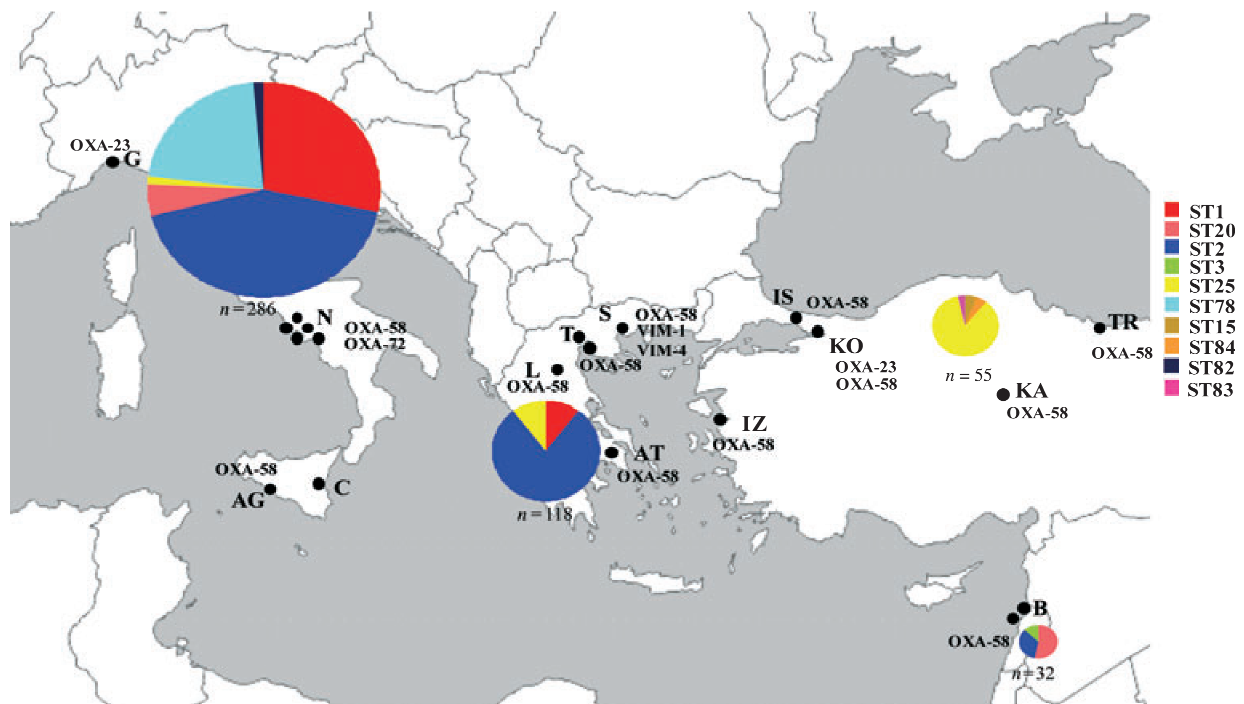

FIG. 1. Geographical distribution and genotypic characterization of *Acinetobacter baumannii* strains included in the study. Black dots indicate the location of hospitals in which *A. baumannii* strains were isolated. Cities are indicated by the following letters: G, Genoa; N, Naples; AG, Agrigento; C, Catania; L, Larissa; T, Thessaloniki; AT, Athens; S, Serres; IS, Istanbul; IZ, Izmir; KO, Kocaeli; TR, Trabzon; B, Beirut. Coloured pie charts indicate the prevalence of *A. baumannii* isolates assigned to different sequence types (STs) in each country. The size of circles is related to the number of patients per countries as indicated. Carbapenem-hydrolysing enzymes isolated in each city are also indicated.

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three Greek hospitals (Table 1), thus suggesting that this clone might have encountered favourable conditions for spread within the same city or between different countries, as described for other *A. baumannii* epidemics [1,2,5,6]. Our data also demonstrate the diffusion of strains assigned to ST25 in Greece, Italy and Turkey, of those assigned to ST78 in Italy, and of those assigned to ST15 and ST84 in Turkey (Table 1 and Fig. 1).

eBURST analysis [19] of the ten STs as compared with the 82 profiles of the database showed that ST20 and ST1 are single-locus variants and belong to CC1 [14]. ST84 formed a novel CC with ST15, as these two STs differ by a single allelic mismatch. ST82 was placed in CC10 [14] as a single-locus variant of ST10. All other STs were singletons, i.e. differed by at least two genes from all other profiles. *A. baumannii* CCs can be regarded as clones [14,19,20]. Typing results generated by 3LST [17] were concordant with MLST data (Table 1). No novel 3LST group was assigned to strains 2977, 2978 and 3866, because they were microepidemic strains [8]. Overall, the present data show that *A. baumannii* strains circulating between 1999 and 2009 represent a highly structured population. The MLST scheme adopted herein [14] differs from the MLST scheme used in previous publications [13,15,16]; correspondence can be established using genome sequences or reference strains.

In accordance with previous findings [1,2,9,10,12], a class D carbapenem-hydrolysing oxacillinase was found in all 29 carbapenem-resistant strains by PCR and DNA sequence experiments, performed as reported previously [6]. The bla_{OXA-58} gene was identified in 27 strains assigned to 18 distinct PFGE profiles and STs. The bla_{OXA-23} gene was identified in an ST2 strain from Genoa, Italy, and in an ST25 strain from Kocaeli, Turkey; the bla_{OXA-72} gene was found in an ST25 strain from Naples, Italy. The metallo-β-lactamase-encoding genes bla_{VIM-1} and bla_{VIM-4} were detected in ST2 and ST1 strains isolated in Greece (Table 1 and Fig. 1). No acquired class D carbapenem-hydrolysing oxacillinasess or metallo-β-lactamasess were identified in the six carbapenem-susceptible strains (Table 1). In accordance with our data, the genes bla_{OXA-23}, bla_{OXA-24} and bla_{OXA-58} were found in a few distinct clusters defined by other typing methods, some of which correspond to international clones I, II and III [7,10,12,13].

In conclusion, *A. baumannii* outbreaks in four Mediterranean countries were caused by the spread of strains belonging to few genotypes, in particular ST2 and, to a lesser extent, ST1, ST25 and ST78, probably favoured by the bla_{OXA-58}, bla_{OXA-23} and bla_{OXA-72} genes. The MLST scheme utilized herein represents a useful standardized typing method for identifying important *A. baumannii* clones and tracking their geographical expansion.

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Transparency Declaration

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References

1. Peleg AY, Seifert H, Paterson DL. *Acinetobacter baumannii*: emergence of a successful pathogen. *Clin Microbiol Rev* 2008; 21: 538–582.
2. Zarrilli R, Giannouli M, Tomasone F, Triassi M, Tsakris A. Carbapenem resistance in *Acinetobacter baumannii*: the molecular epidemic features of an emerging problem in health care facilities. *J Infect Develop Ctries* 2009; 3: 335–341.
3. Dijkshoorn L, Aucken H, Gerner-Smidt P *et al.* Comparison of outbreak and nonoutbreak *Acinetobacter baumannii* strains by genotypic and phenotypic methods. *J Clin Microbiol* 1996; 34: 1519–1525.
4. Van Dessel H, Dijkshoorn L, van der Reijden T *et al.* Identification of a new geographically widespread multiresistant *Acinetobacter baumannii* clone from European hospitals. *Res Microbiol* 2004; 155: 105–112.
5. Coelho JM, Turton JF, Kaufmann ME *et al.* Occurrence of carbapenem-resistant *Acinetobacter baumannii* clones at multiple hospitals in London and Southeast England. *J Clin Microbiol* 2006; 44: 3623–3627.
6. Zarrilli R, Casillo R, Di Popolo A *et al.* Molecular epidemiology of a clonal outbreak of multidrug-resistant *Acinetobacter baumannii* in a university hospital in Italy. *Clin Microbiol Infect* 2007; 13: 481–489.
7. Nemec A, Krizova L, Maixnerova M *et al.* Emergence of carbapenem resistance in *Acinetobacter baumannii* in the Czech Republic is associated with the spread of multidrug-resistant strains of European clone II. *J Antimicrob Chemother* 2008; 62: 484–489.
8. Giannouli M, Tomasone F, Agodi A *et al.* Molecular epidemiology of carbapenem-resistant *Acinetobacter baumannii* strains in intensive care units of multiple Mediterranean hospitals. *J Antimicrob Chemother* 2009; 63: 828–830.
9. Mendes RE, Spanu T, Deshpande L *et al.* Clonal dissemination of two clusters of *Acinetobacter baumannii* producing OXA-23 or OXA-58 in Rome, Italy. *Clin Microbiol Infect* 2009; 15: 588–592.

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10. Higgins PG, Dammhayn C, Hackel M, Seifert H. Global spread of carbapenem-resistant *Acinetobacter baumannii*. *J Antimicrob Chemother* 2009; 65: 233–238.
11. Giannouli M, Cuccurullo S, Crivaro V *et al.* Molecular epidemiology of multi-drug resistant *Acinetobacter baumannii* in a tertiary care hospital in Naples, Italy, shows the emergence of a novel epidemic clone. *J Clin Microbiol* 2010; 48: 1223–1230.
12. Mugnier P, Poirel L, Naas T, Nordmann P. Worldwide dissemination of the bla_{OXA-23} carbapenemase gene of *Acinetobacter baumannii*. *Emerg Infect Dis* 2010; 16: 35–40.
13. Fu Y, Zhou J, Zhou H *et al.* Wide dissemination of OXA-23-producing carbapenem-resistant *Acinetobacter baumannii* clonal complex 22 in multiple cities of China. *J Antimicrob Chemother* 2010; 65: 644–650.
14. Diancourt L, Passet V, Nemec A, Dijkshoorn L, Brisse S. The population structure of *Acinetobacter baumannii*: expanding multiresistant clones from an ancestral susceptible genetic pool. *PLoS ONE* 2010; 5: e10034.
15. Bartual SG, Seifert H, Hippler C, Luzon MA, Wisplinghoff H, Rodrìguez-Valera F. Development of a multilocus sequence typing scheme for characterization of clinical isolates of *Acinetobacter baumannii*. *J Clin Microbiol* 2005; 43: 4382–4390.
16. Wisplinghoff H, Hippler C, Bartual SG *et al.* Molecular epidemiology of clinical *Acinetobacter baumannii* and *Acinetobacter* genomic species 13TU isolates using a multilocus sequencing typing scheme. *Clin Microbiol Infect* 2008; 14: 708–715.
17. Turton JF, Gabriel SN, Valderrey C, Kaufmann ME, Pitt TL. Use of sequence-based typing and multiplex PCR to identify clonal lineages of outbreak strains of *Acinetobacter baumannii*. *Clin Microbiol Infect* 2007; 13: 807–815.
18. Clinical and Laboratory Standards Institute. *Performance standards for antimicrobial susceptibility testing; eighteenth informational supplement.* Approved Standard M100-S18. Baltimore, MD: CLSI, 2008.
19. Feil EJ, Li BC, Aanensen DM, Hanage WP, Spratt BG. eBURST: inferring patterns of evolutionary descent among clusters of related bacterial genotypes from multilocus sequence typing data. *J Bacteriol* 2004; 186: 1518–1530.
20. Spratt BG, Feil EJ, Smith NH. Population genetics of bacterial pathogens. In: Sussman M, ed. *Molecular medical microbiology*. London: Academic Press, 2001; 445–484.

Evaluation of eight cases of confirmed *Bordetella bronchiseptica* infection and colonization over a 15-year period

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Abstract

We describe eight human cases of *Bordetella bronchiseptica* infection and colonization over a 15-year period. Amongst the eight patients, seven had significant underlying disease. Cat exposure was documented in three cases. Symptoms ranged from asymptomatic carriage to severe pneumonia. We could not identify a homogeneous pattern of clinical disease among symptomatic patients. Although *B. bronchiseptica* infection remains a rare clinical condition among humans, it should be considered as potentially pathogenic when found in airways of immunocompromised patients.

Keywords: *Bordetella bronchiseptica*, case series, clinical study, epidemiology, humans, outcome, Switzerland

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Bordetella spp. are aerobic coccobacilli known to be present in the upper respiratory tract of many animals [1]. *B. bronchiseptica* infections are uncommon in humans [2]. The literature on this subject is subsequently poor. Two comprehensive reviews have been published in the last two decades. The first included 25 cases from 1911 to 1990 [1]. However, the presence of *B. bronchiseptica* was microbiologically confirmed in only ten patients. The other study, published in 1995, included 52 patients but no details were provided about the microbiological identification of *B. bronchiseptica* [3]. Another study published in 2005 focused on the pathogenesis but gave little information on human infections [4]. Recently, several case reports addressed the problem in cystic fibrosis patients [5], HIV patients [2,6] or children with lung transplants [7]. Facing this lack of recent information, we decided to review all cases detected at our institution from 1993 to 2008.

The present study was undertaken at the Geneva University Hospitals, a 2220-bed tertiary care centre. We performed a retrospective case review approved by the institutional ethics review board and based on computerized laboratory log files, examining the pattern of disease caused by *B. bronchiseptica*, addressing underlying conditions and exposures, and examining antimicrobial treatment in relation to patient outcome. To avoid misclassification bias, we included only patients with verified *B. bronchiseptica* isolates because automated identification systems can lead to misclassification as *Acinetobacter* spp. or other nonfermentative Gram-negative rods. Most cases were first identified by API 20NE gallery or VITEK2 (bioMerieux SA, Marcy-l'Etoile, France) phenotypic identification systems, and then supported by a positive oxidase reaction. All available stored

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