Figure 3 Cost of pentamidine clinic per year.

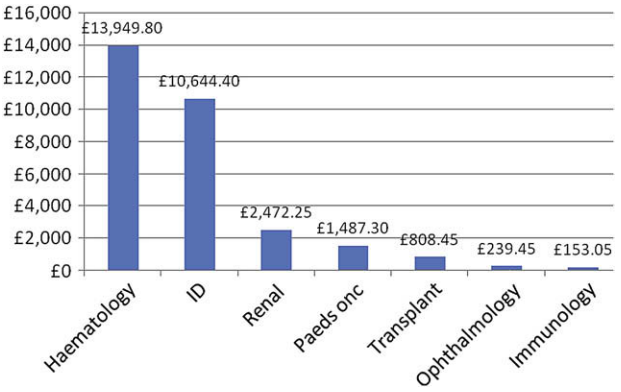

Figure 4 Cost of pentamidine clinic per referring team.

and those in other disciplines.[7] Inhaled pentamidine is a convenient and usefully intermittent therapy, which the majority of individuals find acceptable.[8]

In our study we were concerned to identify a relatively large minority of individuals in whom the justification for pentamidine prophylaxis was not adequately documented or substantiated and this has led to a review of our procedures and a formalisation of our criteria for admission to the pentamidine clinic.

The default rate on attendance for the clinic was worst in the HIV positive population and reflected the rather haphazard and chaotic lifestyles of some of these patients rather than any apparent difficulty in accessing the service for other reasons.

The minimum cost of a single monthly appointment at the pentamidine clinic was £47.00, which compares with £2.81 for a one-month course of cotrimaxazole, 960 mg taken three times weekly. Thus pentamidine is an expensive alternative to the gold standard therapy. Highlighting this has been a trigger to initiating a review of reasons why patients are on pentamidine rather than alternative drugs. The identification of the proportions of patients coming from different disciplines has been of value in establishing payment pathways to support the service.

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References

1. Wallace JM, Hansen NI, Lavange L, Glassroth J, Browdy BL, Rosen MJ, et al. Respiratory disease trends in the Pulmonary Complications of HIV Infection Study Cohort. *Am J Respir Crit Care Med* 1997;**155**:72–80.
2. Palella Jr FJ, Delaney KM, Moorman AC, Loveless MO, Fuhrer J, Satten GA, et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. HIV Outpatient Study Investigators. *N Engl J Med* 1998;**338**:853–60.
3. Rodriguez M, Fishman JA. Prevention of infection due to *Pneumocystis* spp. in human immunodeficiency virus-negative immunocompromised patients. *Clin Microbiol Rev* 2004;**17**(4):770–82.
4. Ioannidis JPA, Cappelleri JC, Skolnik PR, Lau J, Sacks HS. A meta-analysis of the relative efficacy and toxicity of *Pneumocystis carinii* prophylactic regimens. *Arch Intern Med* 1996;**156**:177–88.
5. Hughes WT, Kuhn S, Chaudhary S, Feldman S, Verzosa M, Aur RJA, et al. Successful chemoprophylaxis for *Pneumocystis carinii* pneumonitis. *N Engl J Med* 1977;**297**:1419–26.
6. Masur H. Prevention and treatment of *Pneumocystis carinii* pneumonia. *N Engl J Med* 1992;**327**:1853–60.
7. Walzer PD, Perl DP, Krogstad DJ, Rawson PG, Schultz MG. *Pneumocystis carinii* pneumonia in the United States: epidemiologic, diagnostic, and clinical features. *Ann Intern Med* 1974;**80**:83–93.
8. Sands F, Kron M, Brown R. Pentamidine: a review. *Rev Infect Dis* 1985;**7**:625–34.

C. Ward
D. O'Donovan
A.M.L. Lever*
Addenbrooke's Hospital, Hills Road, Cambridge CB2 0QQ, UK
*Corresponding author. Tel: +44 01223 330191; fax: +44 01223 336846.
E-mail address: amll1@mole.bio.cam.ac.uk (A.M.L. Lever)

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Simkania negevensis infection among Brazilian children hospitalized with community-acquired pneumonia

Dear Editor,

Simkania negevensis is an obligate intracellular microbe, related to the chlamydiae.[1] It has been found in domestic water supplies of infected children and this finding raises the possibility of transmission to children from the water of their homes.[2] *S. negevensis* respiratory infections have been described from Europe, Japan and North America.[3] In New York it was not a significant pathogen.[4] We analyzed if *S. negevensis* acute infection occurred in Brazil.

Children aged under five years hospitalized with community-acquired pneumonia (CAP) were enrolled in

Table 1 Patients with community-acquired pneumonia and acute *S. negevensis* infection, in Salvador, North—East Brazil.

Age (month)/ gender	IgM titer		Complaints on admission	Physical findings on admission	Chest X-ray findings on admission	Family and personal past history	Additional infectious agents	Follow up evaluation
	1st sample	2nd sample						
58[a]/female	5 (negative)	20	fever, cough, dyspnoea for 7 days	RR[b] 60, temp 37.5 °C, well nourished, chest retractions and indrawing, crackles	Alveolar infiltrate in left and right bases	day care centre attendance, no previous respiratory disease; 4 people at home, 1 child (the patient) who sleeps with 2 adults; mould at home	none	18 days after discharge, only with cough, RR 27
24/female	40	160	fever, cough, dyspnoea for 3 days	RR 68, temp 39.8 °C, well nourished, chest retractions and indrawing, crackles, wheezing, lethargy	alveolar infiltrate in the left base	no day care centre attendance, 4 previous respiratory discomfort episodes during the last 12 months, without hospitalization; 1 brother with asthma; 4 people at home, 2 children <5 years of age, the patient sleeps alone; no mould at home	respiratory syncytial virus	30 days after discharge, difficult breathing for the last 2 days, RR 48, wheezing
52/female	160	160	cough for 15 days, fever and dyspnoea for 3 days	RR 36, temp 38.9 °C, malnutrition,[c] chest retractions and indrawing, crackles, prolonged expiration	alveolar infiltrate in the right base; pleural effusion in the right hemithorax	day care centre attendance, 12 previous respiratory discomfort episodes during the last 12 months, persistent cough, no hospitalization; 1 brother with asthma; 5 people at home, 1 child (the patient), everybody sleeps in the same room; no mould at home	respiratory syncytial virus and influenza B	18 days after discharge, no complaints, receiving prednisone, RR 23

All three patients lived in Salvador, received penicillin G IV followed by amoxicillin PO and there were no smokers in the children's homes.

[a] Total white blood cell count 29,200/mm^3, 12% immature neutrophils, 75% mature neutrophils.

[b] Respiratory Rate (breaths/min).

[c] Weight 10.8 kg, Height 95 cm.

Salvador, Brazil, because of respiratory complaints. CAP was diagnosed when pulmonary infiltrate was described by a paediatric radiologist, blind to clinical information, on the admission chest X-ray (CXR). The investigation for other aetiologies has been published.[5] An in-house microimmunofluorescence test was used to measure IgG and IgM antibodies to *Chlamydia pneumoniae* and to *S. negevensis*, using purified, formalized elementary bodies of strains K6 in *C. pneumoniae* tests and ATCC strain Z (ATCC, Catalog no. VR-1471) in *S. negevensis* tests[6] in paired serum samples, at the National Public Health Institute, Oulu, Finland. Informed consent was requested before enrolment and the study was approved by the Ethics Committee of the Federal University of Bahia.

Acute pneumonia was radiologically confirmed in 184 patients. Three (1.6%) patients were diagnosed to have acute *S. negevensis* infection. Their characteristics are presented in Table 1. One patient had acute *C. pneumoniae* infection based on a 4-fold IgG titer rise between paired sera. There were no cases with simultaneous acute *S. negevensis* and *C. pneumoniae* infection.

The prevalence of acute *S. negevensis* infection was low (1.6%). Two studies reported 10% and 5% prevalence for acute *S. negevensis* infection among 174 Finnish[6] and 101 Italian[7] children with CAP, respectively. In the Finnish study, the prevalence of acute *S. negevensis* involvement was highest (15%) at the age group from 10 years onwards, and 67% of the 18 cases were aged older than 4 years.[6] In the Italian study, the mean age was 4.7 years (range 0.3—16 years).[7] Yamaguchi et al.[3] reported that the seropositivity rate of *S. negevensis* increased sharply in the middle age. The acquisition of *S. negevensis* infection is probably more frequent among older patients.

Our case with *S. negevensis* as the only etiological agent among 15 excluded pathogens (influenza A and B viruses, respiratory syncytial virus, parainfluenza virus types 1, 2, and 3, adenovirus, rhinovirus, enterovirus, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, *Chlamydia trachomatis*, *C. pneumoniae*, *Mycoplasma pneumoniae*) studied[5] had severe clinical presentation, the involvement of both lungs (Fig. 1), and acute inflammatory response(Table 1). Therefore, our results corroborate that *S. negevensis* may be a real cause of CAP among children.

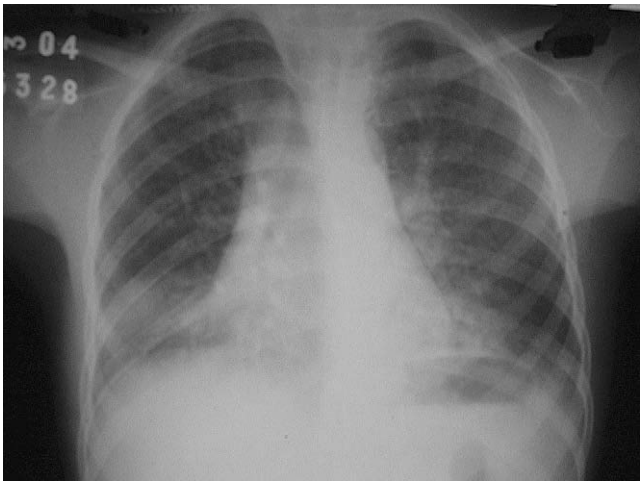

Figure 1 Presence of bilateral alveolar pulmonary infiltrate on the chest X-ray of the 58-month-old child with *S. negevensis* as the only established aetiology of community-acquired pneumonia.

All patients received penicillin and full recovery was achieved (Table 1). Interestingly, *S. negevensis* is totally resistant to penicillin and macrolides are the choice options.[1] In a Canadian study, 64% of 22 infants hospitalized with acute bronchiolitis had *S. negevensis* acute infection and all of them recovered without specific antibiotic treatment.[8] Apparently, the health assistance in the hospital ensured the improvement of the cases, but one may raise the question if an appropriate treatment could shorten the duration of the disease. Our study being, according to our knowledge, the first report of *S. negevensis* infection in the Central and South Americas adds a new continent to the geographical distribution of *S. negevensis* infection in the world.

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References

1. Friedman MG, Dvoskin B, Kahane S. Infections with *Chlamydia*-like microorganism *Simkania negevensis*, a possible emerging pathogen. *Microbes Infect* 2003;**5**:1013—21.
2. Kahane S, Greenberg D, Newman N, Dvoskin B, Friedman MG. Domestic water supplies as a possible source of infection with *Simkania*. *J Infect* 2007;**54**:75—81.
3. Yamaguchi Y, Yamazaki T, Inoue M, Mashida C, Kawagoe K, Ogawa M, et al. Prevalence of antibodies against *Simkania negevensis* in a healthy Japanese population determined by the microimmunofluorescence test. *FEMS Immunol Med Microbiol* 2005;**43**:21—7. doi:10.1016/j.femsim.2004.06.023.
4. Kumar S, Kohlhoff SA, Gelling M, Roblin PM, Kutlin A, Kahane S, et al. Infection with *Simkania negevensis* in Brooklyn, New York. *Pediatr Infect Dis J* 2005;**24**:989—92. doi:10.1097/01.inf.0000183755.24578.0b.
5. Nascimento-Carvalho CM, Ribeiro CT, Cardoso MRA, Barral A, Araújo-Neto CA, Oliveira JR, et al. The role of respiratory viral infections among children hospitalized for community-acquired pneumonia in a developing country. *Pediatr Infect Dis J* 2008; **27**:939—41. doi:10.1097/INF.0b013e3181723751.
6. Heiskanen-Kosma T, Paldanius M, Korppi M. *Simkania negevensis* may be a true cause of community acquired pneumonia in children. *Scand J Infect Dis* 2008;**40**:127—30. doi:10.1080/00365"-540701558680.
7. Fasoli L, Paldanius M, Don M, Valent F, Vetrugno L, Korppi M, et al. *Simkania negevensis* in community-acquired pneumonia in Italian children. *Scand J Infect Dis* 2008;**40**:269—72. doi:10.1080/00365540701642146.
8. Greenberg D, Banerji A, Friedman MG, Chiu C-H, Kahane S. High rate of *Simkania negevensis* among Canadian inuit infants

hospitalized with lower respiratory tract infections. *Scand J Infect Dis* 2003;**35**:506—8. doi:10.1080/00365540310014648.

Cristiana M. Nascimento-Carvalho*
Department of Paediatrics, Federal University of Bahia School of Medicine, Rua Prof. Aristides Novis, No.105/1201B, 40210-630 Salvador, CEP 40110-100, Brazil
*Corresponding author. Tel.: +55 71 32357869; fax: +55 71 33320725.
E-mail address: nascimentocarvalho@hotmail.com

Maria-Regina A. Cardoso
Department of Epidemiology, Faculty of Public Health, São Paulo University, São Paulo, CEP 01246-904, Brazil

Mika Paldanius
National Public Health Institute, Oulu, 90220 Finland

Aldina Barral
Department of Pathology, Federal University of Bahia School of Medicine and Centro de Pesquisa Gonçalo Muniz, Fundação Oswaldo Cruz, Salvador, CEP 40110-100, Brazil

César A. Araújo-Neto
Department of Image Diagnosis, Federal University of Bahia School of Medicine, Salvador, CEP 40110-100 Brazil

Annika Saukkoriipi
National Public Health Institute, Oulu, 90220 Finland

Raija Vainionpää
Department of Virology University of Turku, Turku, 20520 Finland

Maija Leinonen
National Public Health Institute, Oulu, 90220 Finland

Olli Ruuskanen
Department of Pediatrics, University of Turku, Turku, 20520 Finland

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Water-borne *Sphingomonas paucimobilis* epidemic in an intensive care unit

Sphingomonas paucimobilis, previously known as *Pseudomonas paucimobilis*, is a gram-negative, oxidase positive bacterium. *Sphingomonas* typically produces yellow-pigmented colonies on blood agar. The bacterium, commonly observed in land and water habitats, can occasionally lead to hospital infections.[1,2] *S. paucimobilis* epidemics have particularly been reported in hospital departments where immune-suppressed patients are monitored, such as the hematology and oncology clinics, and intensive care units (ICUs).[3—5]

In the last week of April 2007, *S. paucimobilis* was isolated from two of the three patients at the Kocaeli University Medical School Hospital, Cardiovascular ICU within three days interval. From the environmental samples that were collected to investigate the origin of the strains, *S.paucimobilis* with the same antibiotic susceptibility was isolated. The present study aimed to investigate the genotypic relationship of *S. paucimobilis* strains isolated from clinical and environmental samples with the PFGE method, and to prevent the spread of the strain among patients.

The cardiovascular ICU of the Kocaeli University Medical School Hospital is a six-bed, open and invasive ICU. The unit was established two years ago and it has been used only for patients requiring intensive care following cardiovascular surgery. The ICU run two shifts per day, with 1 doctor, 2 nurses and 1 nursing assistant working on each shift. Until the day of the particular epidemic, no cases of *S. paucimobilis* infection had been observed in the unit.

The first patient infected with *Sphingomonas* was a 62-year-old male. He was on mechanical ventilation following arterial embolectomy. He had an Acute Physiological Assessment and Chronic Health Evaluation (APACHE) II score of 16 in the first 24 h, and a Glasgow coma score of 3. An increase in the tracheal secretion was observed on the third day of admittance, accompanied by fever and high white blood cell count (WBC: 14.6×10^9/L). Pneumonic infiltrations were identified on chest x-ray and meropenem (3×1 g/day, intravenously) was started empirically with the diagnosis of pneumonia. On the following day, 10^5 cfu/ml of *S. paucimobilis* was isolated from the tracheal aspirate specimen and trimethoprim-sulfamethoxazole ($2 \times 80/400$ mg/day, intravenously) was added to the therapy. Urine, wound, and blood cultures revealed no pathogenic microorganism. On the third day of therapy, the patient's fever resolved, and his WBC count decreased to 9.6×10^9/L. The pneumonia symptoms receded and therapy was completed in 14 days. The patient had a Glasgow coma score of 3 throughout the period of stay in the unit. Two days after completion of the therapy, the patient died due to cardiopulmonary arrest.

The second patient was a 22-year-old woman who had undergone mitral valve replacement surgery. She had an APACHE II score of 14 in the first 24 h, and a Glasgow coma score of 12. Average post-operative body temperature was 39 °C and 10^5 cfu/ml of *Acinetobacter baumannii* was isolated from tracheal aspirate culture. The patient had a WBC count of 30×10^9/L, was diagnosed as pneumonia and cefoperazone/sulbactam (2×2 g/day, intravenously) was initiated. *S. paucimobilis* growth was detected in the two blood samples drawn on the same day. Trimethoprim-sulfamethoxazole ($2 \times 80/400$ mg/day, intravenously) was added to the therapy. On the third day of therapy, fever resolved, a WBC count of 9×10^9/L was detected. The therapy was completed on the 14th day and the patient was discharged.

After detection of *S. paucimobilis* growth in clinical samples, within the same week, a total of 15 smear samples were collected from soap and detergent, povidone iodine