* Thomson RB, Vanzo SJ, Henry NK, Guenther KL, Washington JA. Contamination of cultures processed with the isolator lysis-centrifugation blood culture tube. *J Clin Microbiol* 1984;19:97-9.

¹⁰ Cashman JS, Boshard R, Matsen JM. Viability of organisms held in the isolator blood culture system for 15 h and their rapid detection by acridine orange staining. *J Clin Microbiol* 1983;18:709-12.

¹¹ Bille J, Edson RS, Roberts GD. Clinical evaluation of the lysis-centrifugation blood culture system for the detection of fungemia and comparison with a conventional biphasic broth blood culture system. *J Clin Microbiol* 1984;19:126-8.

¹² Whimby E, Wong B, Kiehn TE, Armstrong D. Clinical correlations of serial quantitative blood cultures determined by lysis-centrifugation in patients with persistent septicemia. *J Clin Microbiol* 1984;19:766-71.

¹³ Stockman L, Roberts GD, Ilstrup DM. Effect of storage of the Du Pont lysis-centrifugation system on recovery of bacteria and fungi in a prospective clinical trial. *J Clin Microbiol* 1984;19:283-5.

¹⁴ Bille J, Stockman L, Roberts GD, Horstmeier CD, Ilstrup DM. Evaluation of a lysis-centrifugation system for recovery of yeasts and filamentous fungi from blood. *J Clin Microbiol* 1983;18:469-71.

¹⁵ Fleming WH, Lewis BH, Dorn GL. Effectiveness of lysis-centrifugation blood culture technique in recovering fungi from blood. *Lab Med* 1984;15:104-8.

Chlamydia psittaci infection in a man and his birds

A 48 year old motor mechanic became ill on 16 October 1984 with headache, loss of appetite, fever, and other symptoms similar to those of influenza but no cough. Complement fixation antibodies to common respiratory viruses or *Chlamydia psittaci* were not detected in a sample of serum taken one week later. A second serum sample, however, collected on 6 November had a chlamydial complement fixation titre of 256. When the sera were examined by indirect immunofluorescence, using the L₂ strain of *C trachomatis* and the EAE strain of *C psittaci* similar titres were noted. Chlamydial IgM, however, was not detected by immunofluorescence in either serum sample (Table). These results were compatible with recent *C psittaci* infection. The patient was treated with tetracycline and made a complete recovery.

The patient owned over 100 birds, including Australian parakeets, ducks, chickens, and geese. In view of the serological data the recent clinical history of the aviary birds in particular was investigated. Although signs of illness had not been

Chlamydial antibody titre in patient's serum

Date serum obtained (1984)	CF titre	IFA titre		IFA IgM
		L ₂	EAE	
23 October	< 8	< 16	< 16	Negative
6 November	256	1024	1024	Negative

CF—complement fixation; IFA—indirect immunofluorescence antibody; EAE—Enzoatic abortion of ewes strain of *C psittaci*; L₂ = L₂ strain of *C trachomatis*.

observed before the patient's illness, two birds had died for no apparent reason on 29 October. Faecal droppings obtained the next day from all the birds that were housed in three aviaries were examined for the presence of *C psittaci*. Suspensions of the faecal material were inoculated into mouse L929 cells, and after incubation at 37°C for 48 hours the monolayers were examined for chlamydial inclusions using a *C psittaci* antiserum labelled with fluorescein. Aviary C was found to contain the most birds excreting *C psittaci* (7/15). Three of the 19 birds in aviary A but none of the 12 in aviary B were positive for *C psittaci*.

On 25 November a yellow rosella from aviary C died immediately after medication for worms had been administered. Post-mortem examination showed many nematodes in the intestine. The liver was enlarged and copper coloured and the spleen was enlarged and mottled, both these signs being the principal characteristic lesions associated with psittacosis.¹ *C psittaci* was isolated from spleen, liver, and cloacal samples according to the method used by Chalmers *et al.*² Shortly after this all birds in aviary C were given aureomycin. The antibiotic was added to the feed at a concentration of 4 g/kg and to the water at a concentration of 50–150 mg/l. The birds were on medication for five days then off for two days throughout the 45 day course. The faecal droppings were then re-examined, but no chlamydias were isolated from any sample.

The yellow rosella had been purchased by the patient on 6 October at a parrot show in Luton. It is believed from the ring coding that the bird had recently been imported, probably from Belgium. Ten days after purchasing the bird the patient began to feel unwell. One of his daughters, who had suffered a respiratory complaint all summer, regularly helped to feed all the birds with the exception of those housed in aviary C. The patient himself looked after this aviary and was also responsible for cleaning out all the aviaries. Antibody to *C psittaci* was not detected in the daughter's serum taken on 23 November, and the respiratory disorder subsequently resolved.

The patient's wife, his other daughter, and a friend who occasionally helped look after the birds all remained well.

Although the patient cleaned his aviaries thoroughly during the last week of September, the source of infection was most probably the yellow rosella, which he had recently acquired. Interestingly, chlamydial IgM antibody was not detected in either of the patient's serum samples. This may indicate that he had been reinfected.

The protection of the public against contracting chlamydiosis from infected imported birds has been the subject of considerable discussion. The main responsibility of the veterinary officer is to prevent the spread of chlamydia to poultry. To some extent this safeguard is provided by the Importation of Birds, Poultry, and Hatching Eggs Order (1979). Veterinarians who diagnose chlamydial infection in birds should warn the owners of the possibility of contracting the disease but are under no obligation to do so. It should be noted that birds may act as carriers and excrete chlamydia without showing clinical manifestations of disease.³ Signs of chlamydiosis usually develop in birds suffering a concurrent secondary infection.⁴ It may be that in the case described chlamydial infection developed in this yellow rosella as a result of stress induced by infestation by worms.

DR TG WREGHITT

Clinical Microbiology and
Public Health Laboratory
Addenbrooke's Hospital
Cambridge CB2 2QW

DR WSK CHALMERS
Animal Health Trust,
Lanwades Park,
Kennet, Newmarket
Suffolk CB8 7PN

DR EO CAULFIELD
Public Health Laboratory
Myrtle Road, Kingsdown
Bristol BS2 8EL

References

- Mohan R. Epidemiologic and laboratory observation of *Chlamydia psittaci* infection in birds. *J Am Vet Med Assoc*

1984;1984:1372-4.

² Chalmers WSK, Farmer H, Evans RT, Woolcock PR. Isolation in McCoy cells of Chlamydia psittaci obtained from the domestic duck (*Ana platyrhynchos*). *Avian Pathology* 1983;12:341-5.

³ Meyer KF. The host spectrum of psittacosis—lymphogranuloma venereum (PL) agents. *Am J Ophthalmol* 1967;63:1225-45.

⁴ Page LA, Avian (ornithosis). In: Hofsted MS, Calnek BW, Helmboldt CF, Reid WU, Yoder HW jnr, eds. *Diseases of poultry*. Iowa: Iowa State University Press, 1978.

Capnocytophaga ochracea: an unusual opportunistic pathogen

We read with interest the article by Hawkey *et al*¹ in which they reviewed *Capnocytophaga ochracea*, and we report a similar case that had interesting features. A 48 year old man, who had acute myeloid leukaemia, developed neutropenia and mouth ulcers after cytotoxic treatment. He subsequently became ill with fever and septicaemia. Two sets of blood cultures were taken and a course of piperacillin and cefuroxime was started, after which he quickly recovered.

A slender Gram negative bacillus was isolated from only the aerobic 0.1% glucose broth of one set at 48 hours. On subculture, after 72 hours' incubation in 5% carbon dioxide on chocolate agar, 1-3 mm, grey-golden, moist, flat colonies were grown, which had a spreading edge and some knob like projections; also on the blood agar incubated for 72 hours anaerobically, 0.5-1.0 mm, grey-golden, flat colonies were visible that had pitted the surface of the agar. No growth had occurred on the aerobic blood agar on primary subculture, but subsequent attempts at subculture showed that *C. ochracea* will grow in air, forming pinpoint colonies at 24 hours, which, on further incubation, develop a metallic sheen. This observation agrees with that of Kristiansen *et al*,² who doubted the degree of dependence on carbon dioxide of *C. ochracea*, but disagrees with the view of Hawkey *et al*,¹ who considered carbon dioxide to be an absolute requirement.

Table 1 shows the results of our identification tests.

We agree with Forlenza³ that these results may vary according to the techniques and media used; indeed, our peptone water sugar results to galactose, raffinose, and glycerol differ from those of Hawkey *et al*,¹ and the explanation may be that, unlike us, they used broth base serum water sugars.

Disc diffusion tests for sensitivity to

Table 1 Results of biochemical tests

Biochemical reactions:		Acid production from sugars:	
Catalase	—	Glucose	+
Oxidase	—	Sucrose	+
Aesculin hydrolysis	+	Galactose	—
Arginine dihydrolase	—	Lactose	+
Ornithine decarboxylase	—	Raffinose	—
Lysine decarboxylase	—	Maltose	+
Reduction of nitrate	+	Starch	+
chlamydiosis urease		Dextrin	+
		Glycerol	+
		Mannitol	—
		Salicin	—
		Sorbitol	—
		Trehalose	—

Table 2 Results of disc diffusion tests

Resistant to:	Sensitive to:		
Metronidazole	Penicillin G	Fusidic Acid	Cefoxitin
Sulphonamide	Ampicillin	Vancomycin	Cefamandole
Trimethoprim	Piperacillin	Rifampicin	Ceftazidime
	Azlocillin	Chloramphenicol	Ceftoxime
	Methicillin	Cephazolin	Cefsulodin
	Erythromycin	Cephalexin	Moxalactam
	Tetracycline	Cefuroxime	Ciprofloxacin

Table 3 Sensitivity tests in different gaseous environments

Aminoglycoside	Oxygen	Carbon dioxide
Gentamicin	Intermediate	Resistant
Tobramycin	Resistant	Resistant
Netilmicin	Intermediate	Resistant
Amikacin	Intermediate	Resistant

antibiotics were performed on the isolate by the Stokes method using appropriate sensitive controls: Oxford *Staphylococcus* (NCTC 6571); *Haemophilus influenzae* (NCTC 8143); *Clostridium perfringens*; and *Pseudomonas aeruginosa* (NCTC 10662). The plates were incubated in air and 5% carbon dioxide for 24 hours. The inoculum was standardised, and the medium was sensitivity test agar (Oxoid) supplemented with 5% lysed blood and Vitox (Oxoid). The discs were of the routine strengths advised for the clinical laboratory. Table 2 summarises the antibiotic profile obtained.

In contrast to Hawkey *et al*,¹ we found *C. ochracea* to be sensitive to the range of cephalosporins, and this may be an important therapeutic point as these cidal antibiotics are often used to treat neutropenic patients as part of a regimen giving broad spectrum cover. This organism needs effective treatment, particularly as it is said to produce a toxic inhibitor of neutrophil activity, which may further reduce the impaired immunity.⁴

The only antibiotic results affected by carbon dioxide were those of the aminoglycosides whose zones of inhibition were

smaller in carbon dioxide than oxygen, as expected, but this influenced interpretation (Table 3).

On the basis of these results and considering the dose related toxicity, it would not be advisable to treat infection with *C. ochracea* with aminoglycosides alone but it would be interesting to find out if the aminoglycosides have synergy with the penicillins or cephalosporins, as this combination is often used to treat immunosuppressed patients.

In conclusion, our case again illustrates how *C. ochracea* can cause septicaemia in the neutropenic patient, the source of which may be mouth ulcers. To isolate the organism the microbiologist may have to culture the specimen in carbon dioxide for more than 48 hours on primary isolation. Further reports of the susceptibility to antibiotics of *C. ochracea* would be of interest.

We thank Mr H Malnick of the National Collection of Tissue Cultures, Colindale, for confirming the identity of our isolate, and Professor Barrett for permission to report on his patient.