in already well described genera. Based on the criteria of biochemical tests, electron-microscopy, and DNA homology, the 22 strains could not be placed in an existing genus. Therefore, a new genus *Mobiluncus* (mobilis capable of movement, uncus a hook, *Mobiluncus* a mobile curved rod) was proposed. This has a guanine: cystosine ratio of 49–52%.

To our knowledge this is the first isolation outside the genital tract of *Mobiluncus* sp in this country. There is one report of a confirmed isolation outside the genital tract from the Netherlands,[3] and a series of four patients from Belgium[4] where it was deduced retrospectively that the isolates belonged to the *Mobiluncus* genus.

Anaerobic breast abscess due to *Bacteroides* species, often in association with other anaerobes, has been well documented.[5 6] Leach *et al*[5] concluded that anaerobic breast abscesses occur in non-puerperal women with inverted nipples and postulated that the source of the organism was either the vagina or the oropharynx rather than the bowel. In the only confirmed report of *Mobiluncus* isolated from a breast abscess the patient was not pregnant and had inverted nipples.[3] Our patient was also not pregnant and had a prosthetic breast implant following surgery for duct ectasia. The implant would have provided a focus for organisms to settle, similar to the focus offered by duct ectasia or chronic breast disease in patients with inverted nipples, the organism gaining access to the breast either by bacteraemia or by direct transfer from the genital tract. There was no history of vaginal "discharge" at presentation, nor was it specifically looked for, consequently no high vaginal swab was taken at the time.

It seems likely, therefore, that the same mechanism may operate for both *Bacteroides* and *Mobiluncus* spp in causing breast abscess, although at present, there have been too few isolates of *Mobiluncus* spp to be certain.

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References

[1] *Bacterial vaginosis*. Pardh P-A, Taylor-Robinson D, eds. Stockholm: Amlqvist and Wiksell International, 1984.

[2] Spiegel CA, Roberts M. Mobiluncus gen nov, Mobiluncus curtisii subsp curtisii sp now, Mobiluncus curtisii subsp holmesii subsp now, and Mobiluncus mulieris sp nov, Curved rods from the human vagina. *International Journal of Systematic Bacteriology* 1984;**34**: 177–84.

[3] Sturm AW, Sikkenk PJH. Anaerobic curved rods in breast abscess. *Lancet* 1984;ii:1216.

[4] Glupczynski Y, Labbe M, Crokaert F, *et al*. Isolation of Mobiluncus in four cases of extragenital infections in adult women. *Eur J Clin Microbiol* 1984;**3**:433–5.

[5] Leach RD, Eykyn AJ, Phillips I, Corrin B. Anaerobic subareolar breast abscess. *Lancet* 1979;i:35–7.

[6] Hale JE, Perinpanayagam RM, Smith G. Bacteroides: an unusual cause of breast abscess. *Lancet* 1976;ii:70.

Detection and importance of β lactamase producing "non-pathogens" in patients with chronic obstructive airways disease

Since β lactamase activity was first described in 1940 by Abraham and Chain[1] attention has been directed to its detection and clinical importance in body secretions. In 1945 Gots[2] described a rapid method for determining whether organisms produce penicillinase, using a penicillin agar medium inoculated with an organism sensitive to penicillin.

In recent years it has been reported that β lactamase producing "non-pathogenic bacteria" have contributed to the failure of β lactamase treatment in patients with respiratory infections.[3 4] The commonest β lactamase producing organisms described are *Staphylococcus aureus, Haemophilus influenzae, Haemophilus parainfluenzae, Branhamella catarrhalis* and *Bacteroides* spp.[3]

This report sets out to show whether such production of β lactamase is clinically important in patients with acute exacerbations of chronic obstructive airways disease (COAD), who are often treated with ampicillin.

Between September 1984 and March 1985 a random selection of sputum from patients with exacerbations of COAD was made. The specimens were cultured for respiratory pathogens and subsequently were examined for β lactamase produced from "non-pathogens" present in the upper or lower respiratory tract.

A modified Gots's[2] technique was used using mannitol salt agar containing 1·6 μg/ml of penicillin—that is, four times the minimal inhibitory concentration of penicillin to *Staphylococcus aureus* NCTC 6571. This was seeded with a four hour broth culture of *S aureus* NCTC 6571. Size 4 wells were punched out and half filled with sputolysed sputum (Stat-Pack Dithiothreitol solution, Calbiochem-Behring). The following morning they were examined for growth of *S aureus* on the surface of the agar. In those which showed growth the sputum cultures were re-examined, each isolate being tested for production of β lactamase, using Mast intralactam strips. Of the 105 sputa tested, only six showed evidence of β lactamase activity. There were no *Haemophilus influenzae* producing β lactamase in the group, and examination of the culture of the sputa showed no organisms producing β lactamase.

Of the six positive sputa showing β lactamase activity, culture yielded upper respiratory tract flora in three cases and *Streptococcus pnuemoniae* in three cases, two of which responded to amoxycillin, the third patient died of carcinomatosis of the lung the day the specimen was taken.

The increased incidence of production of β lactamase by *Haemophilus spp*[5] has created a dilemma in the choice of initial antibiotic treatment in patients with exacerbations of COAD. In an area with a relatively low prevalence of β lactamase producing *Haemophilus influenzae* (less than 1% in this hospital) it seems that ampicillin is appropriate first line treatment in these patients, in view of the low incidence of β lactamase in their sputum.

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References

[1] Abraham EP, Chain E. An enzyme from bacteria able to destroy penicillin. *Nature* 1940; **146**:837.

[2] Gots J. The detection of penicillinase-producing properties of micro-organisms. *Science* 1945; **102**:309.

[3] Brook I, Gober A. Rapid method for detecting β lactamase producing bacteria in clinical specimens. *J Clin Pathol* 1984;**37**:1392–4.

[4] Maddocks JL. Indirect pathogenicity. *J Antimicrob Chemother* 1980;**6**:307–9.

[5] Philpott AJ, Philpott-Howard J, Williams JD. Increase in antibiotic resistance in haemophilus influenzae in the UK since 1977. *Br Med J* 1983;**284**:1597–9.

Biphenotypic leukaemia

We previously reported a case of biphenotypic leukaemia (T acute lymphoblastic leukaemia and acute mycloblastic leukaemia)

Immunological phenotype of blast cells

Marker	*Diagnosis*	*Relapse*
White blood cell count ($\times 10^9$/l)	167	67
Blasts (%)	98	95
Precursor cells:		
TdT	69*	90
I2	40	19
J5/CALLA	<1	<1
Myeloid cells:		
My7	14	10
My9	13	22
My4	11	1
T cells:		
3A1/RFT2	30	28
OKT11	25	73
OKT4	<1	2
OKT8	<1	1
OKT6	<1	<1

*Results shown as (%) positive cells

diagnosed by morphological, cytochemical, immunological, and ultrastructural methods.[1] Following remission induction with daunorubicin, vincristine, prednisolone, and L-asparaginase she received maintenance treatment with methotrexate, 6 mercaptopurine, cytosine arabinoside, vincristine, and prednisolone for 12 months before this was stopped due to neutropenia.

She remained well until August 1985 (28 months after diagnosis) when she relapsed. Full blood count at this time showed haemoglobin 10·4 g/dl, white cell count 67·0 $\times$ 10^9/l (differential 95% blasts, 4% neutrophils, 1% lymphocytes), and platelet count 55·0 $\times$ 10^9/l. Examination of May-Grunwald-Giemsa stained smears showed two distinct blast cell populations with about 70% having lymphoblastic and 30% myeloblastic features. The Table shows details of the immunological studies at diagnosis and relapse, and, apart from an increased expression of OKT11 at relapse, they are remarkably similar. She was treated with vincristine, prednisolone, daunorubicin, and L-asparaginase, but her disease was totally refractory and she died 14 days after relapse. Necropsy examination was not performed.

Biphenotypic leukaemia may either result from a single mutation in a pluripotential progenitor cell with maintained capacity for bilineage differentiation, or, alternatively, may be two unrelated transformations. The fact that the relapse occurred with similar proportions of T cell and myeloid blasts (as found at diagnosis) strongly suggests that in this case they had arisen from a single malignant progenitor. Recently blasts in a proportion of cases of acute myeloid leukaemia have been shown to express membrane sheep red blood cell (E) receptors, or the OKT11 determinant.[2,3] Our case, however seems to differ from these, as the lymphoblasts were positive with more than one T cell antibody (OKT17, 3A1, RFT2) and also because we were able to show mutual exclusion of myeloperoxidase and T cell specific antigens, confirming true biphenotypic leukaemia.

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References

[1] Morgan MAM, Scott CS, Tavares de Castro J, *et al.* Biphenotypic leukaemia: a case of mixed T lymphoblastic and myeloblastic leukaemia. *J Clin Pathol* 1985;**38**:575–81.
[2] Mirro J, Antoun GR, Zipf TF, Melvin S, Stass S. The E rosette associated antigen of T cells can be identified on blasts from patients with acute myeloblastic leukaemia. *Blood* 1985; **65**:363–7.
[3] Neame PB, Soamboonsrup P. E-rosette formation in acute nonlymphocytic leukaemia. *Blood* 1985;**66**:247.

Fibrinogen mediated activation of platelet aggregation

We previously reported fibrinogen mediated enhancement of platelet aggregation in platelet rich plasma in vitro.[1] We have now evaluated another human fibrinogen preparation that was a gift from IMCO (Stockholm, Sweden). This fibrinogen preparation (97–100% of clottable protein) did not enhance or induce platelet aggregation when assessed, using the techniques previously described.[1] Others, however, have shown that IMCO fibrinogen enhances, depending on concentration, both aggregation and serotonin release in gel filtered platelet preparations.[2] Similar findings, using another fibrinogen preparation and platelets resuspended in buffer solutions,[3] have also been reported.

These findings suggest that reports of platelet and fibrinogen interactions vary according to the fibrinogen preparation used and the platelet function index assessed This is illustrated by the interlaboratory differences when reporting the characteristics of fibrinogen binding to platelets.[4,5] Another problem is the presence of contaminants[5] in fibrinogen preparations, as well as the possible structural modification of the fibrinogen molecule either during purification procedures or in vivo as part of a disease process (such as diabetes mellitus).[6] Such structural modifications may in turn affect function, as has been suggested in diabetes mellitus.[7]

We have also become aware of an earlier report[8] showing that albumin influences fibrinogen and platelet interactions. Washed platelets resuspended in buffer were shown to adhere to tubes coated with fibrinogen, and this process was accompanied by a mild platelet release reaction.[8] This fibrinogen and platelet interaction was attenuated by prior addition of albumin to the platelet suspension.[8]

As previously stated,[1] whether these in vitro findings correlate with pathological findings cannot be conclusively answered at present. Nevertheless, evidence is accumulating that fibrinogen has an important role in the pathogenesis of vascular disorders, even when other risk factors, such as familial hypercholesterolaemia are present.[9]

References

[1] Mikhailidis DP, Barradas MA, Maris A, Jeremy JY, Dandona P. Fibrinogen-mediated activation of platelet aggregation and thromboxane A_2 release: pathological implications in vascular disease. *J Clin Pathol* 1985;**38**:1166–71.
[2] Tomikawa M, Iwamoto M, Soderman S, Bllomback B. Effect of fibrinogen on ADP-induced platelet aggregation. *Throm Res* 1980;**19**:841–55.
[3] Phillips HM, Mansouri A, Perry CA. The ability of fibrinogens, F I and F II, to support ADP-induced platelet aggregation. *Thromb Haemost* 1983;**50**:527–9.