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Outcome of *Burkholderia (Pseudomonas) cepacia* colonisation in children with cystic fibrosis following a hospital outbreak

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Abstract

***Background* – While there are reports on the outcome in adults and teenagers with cystic fibrosis of colonisation with *Burkholderia (Pseudomonas) cepacia*, there is little information in children.**

***Methods* – In December 1991 only one of 115 children with cystic fibrosis attending a paediatric centre was colonised with *B cepacia*. Over the next 12 months there was a rapid increase with 23 (20%) becoming colonised; eighteen (79%) of these became colonised in hospital at a time that overlapped with the admission of a *B cepacia* positive child. Three different bacteriocin types were isolated, with one type (S22/PO) being present in 17 (74%) patients. The outcome for children who became colonised with *B cepacia* was compared with that in 33 children who continued to be colonised with *Pseudomonas aeruginosa* alone.**

***Results* – Children colonised with *B cepacia* were older and more poorly nourished than those colonised with *P aeruginosa*, but did not have poorer pulmonary function. After colonisation, the forced expiratory volume in one second (FEV_1) deteriorated between consecutive annual tests, with the average deterioration being greater in those with higher initial levels. Five children with *B cepacia* died from respiratory failure although none showed a fulminant deterioration. Introduction of segregation measures within hospital led to a dramatic decrease in the number of newly colonised patients.**

***Conclusions* – This study provides further evidence for person-to-person spread of *B cepacia* and confirms the effectiveness of simple isolation measures in interrupting spread. Colonisation with *B cepacia* and *P aeruginosa* in children is associated with a more rapid decline in lung function and a significantly increased mortality compared with cases colonised with *P aeruginosa* alone.**

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Keywords: cystic fibrosis, children, *Burkholderia (Pseudomonas) cepacia*, lung function, cross infection.

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In the early 1980s cystic fibrosis centres in North America noted the emergence of *Pseudomonas cepacia* (*Burkholderia cepacia*) as an important pathogen in patients with cystic fibrosis. Subsequent reports have described the outcome of patients with cystic fibrosis following colonisation with *B cepacia*.[1–6]

Early studies suggested that *B cepacia* may be acquired nosocomially,[2 6] but more recent evidence has tended to confirm that *B cepacia* may be transmitted from patient to patient.[7] Patients colonised with *B cepacia* also contaminate the immediate environment with a possible increase in the risks of indirect acquisition.[8] However, there is still debate as to whether acquisition occurs mainly via direct person to person spread[7 9–11] or from environmental sources within or outside the hospital. Notwithstanding, concerns about nosocomial spread have led many centres to segregate patients colonised with *B cepacia*.[5 9 10]

Reports on the outcome after *B cepacia* colonisation have related predominantly to adults and adolescents with cystic fibrosis. We report the outcome of a hospital outbreak of *B cepacia* in children with cystic fibrosis already colonised with *P aeruginosa* attending a paediatric cystic fibrosis centre. Apart from providing further evidence of person to person spread, this outbreak allowed the outcome in the children who became colonised with *B cepacia* in addition to *P aeruginosa* to be compared with those remaining colonised with *P aeruginosa* only.

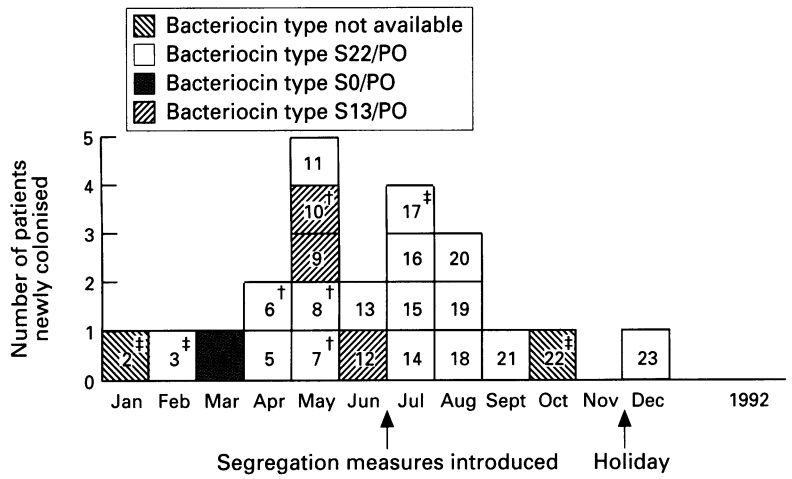
Methods

PATIENTS

In 1992 115 children of mean age 7·6 years (range 0·6–15·8) attended the Cystic Fibrosis Unit at the Royal Hospital for Sick Children, Glasgow. Children with cystic fibrosis needing inpatient care were admitted to one ward where they had complete freedom to play together and socialise. Older children with cystic fibrosis attended the hospital school together.

B CEPACIA OUTBREAK AND SUBSEQUENT SEGREGATION POLICIES

At that time and for 18 months previously 56 children of mean age 8·9 years (range 2·2–15·8) colonised with *P aeruginosa* had had their chest physiotherapy separately from children in whom *P aeruginosa* had not been isolated. Those with *P aeruginosa* (other than infants and toddlers) attended the physiotherapy department twice daily; in those without *P aeruginosa* physiotherapy was performed on their beds in the ward. Each child had an individual



Consecutive cases of B cepacia colonisation during the period of the study indicating periods of overlapping contact in hospital and bacteriocin types isolated. † Deceased; ‡ no inpatient contact with B cepacia colonised patients.

peak flow meter and nebuliser for use throughout the admission.

From January to December 1992 there was a rapid increase in the number of children colonised with *B cepacia* (figure) which occurred exclusively in children already colonised with *P aeruginosa*. The nature and rapidity of the increase strongly suggested spread from child to child. Accordingly, in June 1992 (figure) segregation measures were introduced and thereafter children known to be colonised with *B cepacia* were admitted to a separate ward. While all children with cystic fibrosis continued to attend a single outpatient clinic, those with *B cepacia* were moved to a different waiting area and given appointment times at the end of the clinic. At the introduction of these measures a letter was sent to all parents of children attending the centre explaining the reasons for segregation and suggesting that it would be sensible for children with *B cepacia* to avoid close physical contact with other children with cystic fibrosis outside hospital.

MICROBIOLOGY

Specimens for bacterial culture were either sputum samples or cough swabs in those unable to produce sputum.

Before the *B cepacia* outbreak the microbiology laboratory had been routinely culturing and isolating *B cepacia* for a number of years. During this period only three children with cystic fibrosis had ever been found to be colonised. At the time of the outbreak no changes had occurred in the handling of specimens or methods of culture.

All specimens were cultured fresh for *B cepacia*. Specimens were plated on McConkey agar (Becton Dickinson, Cumbernauld, UK), *B cepacia* selective media (Mast Diagnostics, UK) and blood agar plates (Oxoid Ltd, Oxford, UK) and incubated at 37°C in 5% CO_2. After 24 hours incubation all plates were examined. Normal flora was recorded as "light", "moderate", or "heavy". Presumptive pathogenic organisms were identified by colony morphology and oxidase tests, plated onto blood agar purity plates, and incubated for a further 24 hours at 37°C in 5% CO_2. Diagnostic sensitivity agar plates (Becton Dickinson) and antibiotic impregnated discs (Oxoid Ltd) were used in a modified Stokes method for antibiotic sensitivity testing. Antibiotics tested included tobramycin (10 μg), ceftazidime (30 μg), aztreonam (30 μg), ciprofloxacin (1 μg), colistin sulphate (10 μg), and azlocillin (75 μg). *B cepacia* isolates were identified by 20 NE API strips (Bio Merieux).

Bacteriocin typing of all *B cepacia* organisms was performed by standard methods.[12]

COMPARISON OF CHILDREN COLONISED WITH *B CEPACIA* AND THOSE COLONISED WITH *P AERUGINOSA*

The group of children colonised with *P aeruginosa* who became colonised with *B cepacia* ("*B cepacia* group") was compared with the remaining children with *P aeruginosa* who did not become colonised with *B cepacia* ("*P aeruginosa* group") using data from approximately annual assessments of nutrition (height and weight centiles), respiratory status (peak expiratory flow rate (PEFR), forced expiratory volume in one second (FEV_1), residual volume to total lung capacity ratio (RV/TLC)), and Swachman score.[13]

For the *B cepacia* group the results of two consecutive annual reviews were chosen corresponding as closely as possible to a period six months before and six months after colonisation with *B cepacia* (average period between results 15·5 months; all data from September 1988 to September 1993). In the *P aeruginosa* group we also used results of reviews approximately 12 months apart (average period between results 18·0 months; results obtained between March 1990 and September 1993).

DATA ANALYSIS

The data from children colonised with *B cepacia* and those colonised with *P aeruginosa* were compared using either χ^2 tests of association or Fisher's exact test (as appropriate) for the categorical variables, and either two sample *t* intervals or the non-parametric equivalent for the continuous variables. Group comparisons were undertaken because appropriate matching of cases from the two groups could not be achieved for the baseline parameters of age, sex, pulmonary function, and nutritional status. Analysis of covariance (ANCOVA) was used to examine possible effects of the initial status of the patient on the change in each pulmonary function test result. The recorded deterioration in each measurement was regressed on its initial value, separately for each of the two groups of patients, and a confidence interval for each slope parameter was derived to determine whether the initial value had an influence on deterioration. A model with parallel regression lines was next tested within the general model provided by wholly distinct regression in the two groups. Finally, a model with coincident regression lines, corresponding to the hypothesis of no group effect, was tested within the model with parallel lines (if appropriate).

Results

TRANSMISSION OF *B CEPACIA*

The increase in children colonised with *B cepacia* throughout 1992 is shown in the figure. Of the three children who had *B cepacia* cultured between 1986 and 1992, only one had the organism present consistently at the start of the outbreak.

During January 1992 one child (subject 2, a two year old with mild disease and no hospital admissions from diagnosis at five weeks of age) had *B cepacia* isolated from the sputum. As *B cepacia* quickly cleared from the sputum and has not been isolated again, bacteriocin typing of this strain was not available. A second child (subject 3) with a respiratory exacerbation in January 1992 had a sputum sample on admission which grew three different strains of *P aeruginosa*. *B cepacia* was isolated from a second sputum sample three days before discharge in February 1992. Thereafter the number of children with *B cepacia* increased (figure) so that, by December 1992, 23 (20%) were colonised.

In the *B cepacia* group of 23 patients 19 had more than 10 separate isolates of the organism. Of these, 17 showed persistent colonisation and two intermittent colonisation. Four of the *B cepacia* group had fewer than 10 isolates, and bacteriocin typing on these showed three to be S22/PO; typing was not performed in the remaining patient. The *B cepacia* isolates from three of the 23 children were sensitive to aminoglycosides (tobramycin). Of these, two were chronically colonised (bacteriocin types S22/PO and S0/PO) and one had only a single positive culture (bacteriocin type not available).

B cepacia was sensitive to aztreonam in 22 of the 23 patients and, of these, 19 also showed sensitivity to azlocillin and two to ceftazidime.

In June 1992, following the increase in *B cepacia*, segregation measures were introduced. Over the next few months a decline in the number of newly colonised children occurred (figure). One further case occurred in November 1992 after a holiday in Florida for children with cystic fibrosis, both with and without *B cepacia*. Thereafter the downward trend continued, with only two new cases in the next 12 months, and the rate of *B cepacia* acquisition has remained very low.

Eighteen (78%) of the 23 who became colonised with *B cepacia* were inpatients at a time that overlapped with admission of at least one other child with the organism (figure). The periods of overlap varied between one and 14 days. The time interval between possible exposure and first isolation of *B cepacia* in sputum varied from 61 to 123 days.

During the same period 24 other children with cystic fibrosis were inpatients at the same time as a child with *B cepacia*, but they did not subsequently become colonised with the organism. Again the period of overlap was 1–14 days. However, the children who did not become colonised were significantly younger (5·8 years *v* 10·4 years; $p<0·005$). Seven were *P aeruginosa* negative and therefore received their chest physiotherapy separately from the *P aeruginosa* (and *B cepacia*) group.

BACTERIOCIN TYPES

Bacteriocin types were available for 21 of the 23 *B cepacia* organisms: one child (subject 2) had only one positive sputum sample and another child (subject 22) was transferred to another unit around the time of colonisation (figure). All children had bacteriocin type S22/PO except subject 4 (S0/PO), and subjects 9, 10, and 12 (all S13/PO). None of these bacteriocin types has been associated with fulminant illness due to *B cepacia*.[7]

GROUP COMPARATIVE DATA

Sex distribution

While there were more girls in each group, there was no significant difference in sex distribution ($\chi^2=0·14$, df=1, p=NS; table).

Age distribution

The *B cepacia* group was significantly older (10·4 *v* 7·24 years; $\chi^2=8·5$, df=2, $p<0·05$; table). Because of the small numbers the patients were divided into three age groups (0–5, 6–10, and 11–14 years); 36% of the children colonised with *P aeruginosa* were in the youngest age group compared with only 18% of the *B cepacia* group.

Nutrition

While there was no significant difference between groups in terms of the height distribution based on centiles ($\chi^2=2·1$, df=5; p=NS combining neighbouring categories as necessary for a valid test; table), there were significant differences in the weight distribution ($\chi^2=9·6$; df=3; $p<0·05$, again combining categories as necessary; table). In particular, 61% of the *B cepacia* group had weights on or below the third centile compared with only 21% of the *P aeruginosa* group. In the period following colonisation there was no significant change between the two groups for height or weight ($\chi^2=2·5$, df=2, and 0·90, df=2 respectively, p=NS). Thus, although more were poorly nourished at colonisation, the linear growth and nutrition of the *B cepacia* group did not deteriorate disproportionately in the medium term.

Pulmonary function

The table shows the mean results for FEV_1, PEFR, and the RV/TLC ratio at their baseline assessments. Some children were too young to undergo pulmonary function testing and, accordingly, the numbers in each group differ. For each variable there was no significant difference between the two groups (95% confidence interval (CI) for the difference in the means (−27·1 to 1·8); (−18·3 to 8·1); and (−22 to 71), p=NS). Between groups the mean changes in PEFR and RV/TLC over the study period did not differ. However, the average deterioration in FEV_1 for the *B cepacia* group was significantly greater than that of the *P aeruginosa* group ($p<0·05$). Somewhat surprisingly, the mean FEV_1 for the *P aeruginosa* group had improved 12 months after the baseline result, but not significantly.

An analysis of covariance demonstrated that for both PEFR and RV/TLC the child's initial pulmonary function influenced the change in pulmonary function; for PEFR in both groups those with better initial values fell more, while for RV/TLC both groups declined in a similar direction but those with higher (worse) RV/TLC values fell more. There was no evidence of a group effect on the change in either measurement. For the *B cepacia* group this was also true of FEV_1 in that those with higher initial values deteriorated more on average. However, for the *P aeruginosa* group the initial FEV_1 had no effect on the average change in FEV_1 throughout the study period if one outlier, whose result distorts the analysis, is removed. The combination of *B cepacia* and *P aeruginosa* was therefore associated with a faster deterioration in lung function than infection with *P aeruginosa* alone, and this was most evident in children with better initial lung function. The linear modelling was extended to investigate possible age effects and age group interactions but no significant effects were found.

Swachman scores

The median baseline Swachman scores[13] for the *B cepacia* group was significantly lower (95% CI −5 to −25, $p<0.05$; table). The median scores for children in both groups who had scores available showed no difference between groups in the median reduction in scores over the period of the study (95% CI −5 to −15, p=NS).

Mortality

During the study period five children (two boys and three girls) in the *B cepacia* group died between five and 15 months after colonisation.

Baseline data of children at acquisition of B cepacia colonisation compared with those colonised with P aeruginosa alone

	P aeruginosa + *B cepacia*	*P aeruginosa alone*
n	23	33
Sex		
Boys	10	16
Girls	13	17
Age groups		
0–5 years	4	18
6–10 years	10	10
11–14 years	9	5
Height centile		
0–3	5	4
>3–10	6	7
>10–25	3	5
>25–50	3	6
>50–75	2	6
>75–90	3	2
>90–100	1	3
Weight centile		
0–3	14	7
>3–10	3	10
>10–25	1	1
>25–50	3	7
>50–75	0	6
>75–90	0	1
>90–100	2	1
Swachman scores (median, range)	65 (30–90)*	85 (50–95)**
Lung function (% predicted)	(n=18)	(n=20)
FEV_1	71·4	84·0
PEF	83·1	88·2
RV/TLC	279·1	254·3

* 14 scores available; ** 19 scores available.
FEV_1 = forced expiratory volume in one second; PEF = peak expiratory flow; RV = residual volume; TLC = total lung capacity.

Since the start of the study only one child not colonised with *B cepacia* has died due to a brainstem glioblastoma. The five children colonised with *B cepacia* who died all suffered a steady clinical deterioration. Prior to colonisation with *B cepacia* they were not in a particularly poor condition with respect to either nutrition or pulmonary function.

A further four with *B cepacia* have died since the end of the study.

Discussion

This report describes a substantial hospital outbreak of *B cepacia* infection in a children's cystic fibrosis centre, and the impact of colonisation on the subsequent outcome.

The data provide further evidence that patient to patient transmission of *B cepacia* occurs. Firstly, the incidence of *B cepacia* infection rose rapidly with most of the children who became colonised being in contact during their hospital stay with at least one other child colonised with the organism. The potential duration of exposure to *B cepacia* was not long, between one and 14 days, and is similar to the six days previously reported between two children attending a summer camp.[10] Secondly, more than 74% of children became colonised with a single bacteriocin type, S22/PO. Two children were colonised with this type at the start of the outbreak and may have been the original source. Patient 1 had been colonised with bacteriocin type S22/PO for two years before the outbreak. Although this child was not admitted to the ward for four months before the outbreak, there had been social contact with other children with cystic fibrosis. The same type was isolated from patient 3 who was first found to be colonised after a brief attendance at another cystic fibrosis centre. The isolation of three bacteriocin types suggests that the outbreak did not arise solely from direct patient to patient cross infection. The index cases of the other two bacteriocin types may represent acquisition from the environment. Our data suggest that this is a less important source than patient to patient spread. Other centres have also reported mutiple types, usually with one predominating.[7 14]

The nature of the contact necessary for transmission of *B cepacia* is not clear – for example, by droplet spread or via contaminated fomites or by aerosol spread. A number of reports have linked attendance at cystic fibrosis camps to acquisition of *B cepacia*[15–17] when there appears to be an opportunity for prolonged exposure of non-colonised children to colonised ones, especially through communal activities such as physiotherapy which might promote spread either via aerosols or contaminated fomites.[7] None of the children had recently attended cystic fibrosis camps but, at the time of our outbreak, all the children colonised with *Pseudomonas* species in hospital were having communal physiotherapy in an enclosed space. In stark contrast, the children with cystic fibrosis who were not colonised with *P aeruginosa* at the same time and who received their physiotherapy separately did not become colonised

with *B cepacia*. It is of interest that Humphreys *et al*[18] noted that *B cepacia* could be recovered from room air during occupation by five or six patients with cystic fibrosis colonised with *B cepacia*. The number of bacteria isolated was greater when the patients were coughing. The isolation of *B cepacia* from the air of rooms occupied by colonised patients suggests that dissemination can occur by aerosol spread. In using communal physiotherapy in an enclosed space we may inadvertently have created an environment conducive to the spread of *B cepacia*. The children who became colonised with *B cepacia* were significantly older and it is possible that some spread arose from social contact,[7] more likely in older children, especially in a hospital setting. Interestingly, three children colonised with *B cepacia* had siblings with cystic fibrosis, but only one sibling became colonised, in contrast to previous studies where pairs of siblings generally showed mutual colonisation.[4 9]

The other possibility that the infection arose and spread from a common environmental source is unlikely. Firstly, at the time of the outbreak children were not sharing respiratory equipment so that spread via contaminated equipment is improbable. Also, surveillance cultures of respiratory function equipment have always failed to recover *B cepacia*. Secondly, types identified on subsequent environmental screening of sites such as sinks were of different bacteriocin types than those isolated from colonised children. Finally, simple cross infection measures involving segregation of children colonised with *B cepacia*, with no additional environmental measures, effectively interrupted the progress of the outbreak.

Not all of the children in the *P aeruginosa* group exposed to *B cepacia* became colonised. Those who did were older, less well nourished, and had lower Swachman scores, but did not have worse lung function. During the study there was no evidence of deterioration in nutrition, linear growth, or clinical score in either group. To date it has not been clear whether *B cepacia* colonisation is responsible for the deterioration in lung function in patients with cystic fibrosis or whether it is simply a marker of disease severity. At the time of colonisation the lung function between the two groups did not differ significantly. While there was no evidence of a decline in lung function within each group, the group colonised with both *B cepacia* and *P aeruginosa* showed a significant decline in FEV_1 compared with the group colonised with *P aeruginosa* only. Further analysis of the FEV_1 results in the *B cepacia* group showed that those with the highest FEV_1 values (that is, >50% predicted) deteriorated the most. Colonisation with *B cepacia* was also associated with a significant mortality from respiratory failure.[7] Thus, the dominant impact of *B cepacia* in children with cystic fibrosis is on respiratory function, and may be greater in those with better levels of respiratory function. This contrasts with the adults and adolescents described by Taylor *et al*[19] in whom those with normal or mild lung disease at the outset of infection remained clinically stable, whereas few of those with severe disease remained stable.

In conclusion, cross infection with *B cepacia* arising from person-to-person spread is an important problem in children with cystic fibrosis, and has a major impact on the respiratory system which is reflected in a faster decline in FEV_1 and a higher mortality from respiratory failure. Simple measures to limit cross infection dramatically reduce the incidence of new infections, emphasising that measures to limit the spread of this organism should be introduced in all paediatric cystic fibrosis units.

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