

**MINUTES OF THE FOURTH ANNUAL MEETING OF THE INTERNATIONAL  
BURKHOLDERIA CEPACIA WORKING GROUP**

**April 9 -11, 1999, Banff Conference Centre, Banff Alberta, Canada**

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**OVERVIEW:**

The International *B. cepacia* Working Group comprises a group of researchers with an interest in this organism, its biology and the clinical infections it may cause. Meetings of IBCWG provide a forum for current research, future collaboration and research initiatives to be discussed. This meeting was organized by Eshwar Mahenthalingam with the assistance of co-chairs David Speert, Jane Burns and John LiPuma. The attendance was outstanding with 50 participants representing research groups from 7 different countries. Researchers investigating aspects of plant pathogenesis and biocontrol also took part in the meeting for the first time and delegates from the US Environmental Protection agency and Health Canada were also present. The two-day conference was divided into six main sections:

- (1) Progress/introductory reports
- (2) Research reports from new participants
- (3) Data presentations from lung transplantation groups
- (4) Group discussion topics
- (5) Presentations from other participants (Sunday Evening Session)
- (6) General Discussion and Concluding Remarks

A full program including abstracts was assembled for the meeting and these may be obtained from the IBCWG web site (<http://allserv.rug.ac.be/~tcoenye/>). A brief overview of the highlights of the conference are given in the minutes below:

**.1 PROGRESS/INTRODUCTORY REPORTS:**

Eshwar Mahenthalingam and John LiPuma welcomed the group. It was noted that this was the largest and most diverse meeting to date. Considerable research progress has been made since the last annual meeting and in addition an IBCWG website has been constructed by Tom Coenye (<http://allserv.rug.ac.be/~tcoenye/>).

**1.2 Meeting Numbering**

John LiPuma observed that this was in fact the 7<sup>th</sup> meeting of the IBCWG. It was subsequently agreed by the group to designate the Banff meeting as the 4<sup>th</sup> Annual meeting of the IBCWG. Subsequent annual meetings would thus be numbered in this way.

### 1.3 John LiPuma (US *B. cepacia* Referral Laboratory)

John reported that while clinical labs are still misidentifying *B. cepacia*, the rate of misidentification has dropped since last year. Within the misidentified group of strains, *B. gladioli* and *B. cepacia* are most commonly confused. There is still the odd *P. aeruginosa* called *B. cepacia* and *vice versa*. Just prior to the meeting over 1118 isolates had been recovered from 661 individuals from 116 centres. Dr. LiPuma lab has typed the majority of strains using biochemical and molecular approaches, including RAPD and PFGE. Preliminary genomovar data was also available for 443 *B. cepacia* complex colonized patients: 258 (58%) were genomovars I/III/IV; 159 (36%) were *B. multivorans*; 15 (3.5%) were *B. vietnamiensis* and 11 (2.5%) were group 13 a novel group in the process of genomovar assignment. Dr. LiPuma feels that they are now ready to proceed with strain typing and correlating of that data to clinical data collected by the CF registry.

### 1.4 Deb Henry (Canadian *B.cepacia* strain Repository)

Deb reported that the occurrence of mis-identified *B. cepacia* in Canada has been greatly reduced by education of labs and the development of better selective agars. Deb has been working to identify a series of simple biochemical tests to quickly and accurately determine if an isolate is *cepacia* and if so, to which genomovar it belongs. Biochemical data is being correlated to conventional genomovar testing performed by Peter Vandamme and Tom Coenye and is also being compared with the *recA* testing performed by Eshwar Mahenthalingam and Jocelyn Bischof.

A total of 866 isolates recovered from 448 patients have been examined. *B. cepacia* genomovar III strains were the most prevalent genomovar recovered (82.6% of patients). *B. multivorans* was present in 8.2% of patients and the remaining genomovars (I, IV and *B. vietnamiensis*) accounted for 4.7% of patients. Examples of single strain types which were recovered from multiple patients were common in genomovar III, indicative of acquisition by patient-to-patient spread. The most common epidemic genomovar III strains were as follows, strain type 02 (74.9% of patients), strain type 04 (14.1%), strain type 01 (4.6%); all these latter strains encode the BCESM, but only strain type 02 encodes the cable pilus subunit gene. Approximately 25% of CF patients in Alberta from whom *B. cepacia* complex bacteria were recovered carried a single clone of *B. cepacia* genomovar IV, strain type 16. Instances of multiple patients sharing the same clone of *B. multivorans* were rare. Patients sharing a single clone of *B. cepacia* genomovar I or *B. vietnamiensis* has not been observed among Canadian CF patients.

The epidemiology observed at the Vancouver CF clinics was presented. Essentially, prior to 1985 there was no significant *B. cepacia* problem, only 4 patients were colonized and only 2 patients shared a strain. However, by 1995 there were 39 colonized patients, 29 of these were colonized with one of four *B. cepacia* genomovar III strain types. Stringent infection control measures were put into effect after 1995, and as a result the number of new infections has greatly decreased as have occurrences of patient to patient spread. The Canadian laboratory will continue strain typing through molecular and biochemical methods and hope to move into correlating clinical outcome data to the typing

information.

### **1.5 John Moore, Belfast City Hospital.**

John proposed the international standardization of strain typing for *B. cepacia*, the idea being that researchers around the world could use pulsed field gel electrophoresis (PFGE) as a standard method of strain typing and that if we all agreed on a standard method that data could easily be shared and compared between labs. The proposal was to draft a consensus document for standard method, circulate that method to all IBCWG members with PFGE interests, publish the document in a methods journal, and finally post the standard PFGE data on a web site where it could be easily accessed. Dr. Moore continued to propose the establishment of a PFGE database and a reference lab to administer the submitted data. This issue was raised later in the meeting and the general feeling was that we should work towards a consensus protocol and work up a proposal for a trial study on reproducibility between laboratories.

## **2.1 RESEARCH REPORTS FROM NEW PARTICIPANTS**

### **2.2 Carlos Gonzalez, Texas A&M University**

Dr. Gonzalez reminded us that *B. cepacia* has always been a plant pathogen and that its “mode of attack” in plants may help elucidate pathology in human disease. *B. cepacia* is an opportunistic pathogen, it exploits wounds in plants to gain entry to the living tissues of the plant where it can initiate rot. The rot consists of two different events, maceration and watersoaking, where maceration is complete break down of tissue and watersoaking being plant cell electrolyte leakage. Maceration seems to occur due to the activity of a hydrolase encoded by the *pehA* gene. Watersoaking has been linked to the presence of a cytotoxic factor, wts, that can be isolated from some culture supernatants. It will be helpful to learn if these molecules play a part in the human infection. Dr. Gonzalez is currently examining the experimental panel of *B. cepacia* complex strains for hydrolase and maceration activities.

### **2.3 Jennifer Parke, Oregon State University**

Dr. Parke spoke about the use of *B. cepacia* as a biopesticide, why it’s important and how it is being controlled. Most of the food we grow is susceptible to fungal infections; we have no chemical treatment for many of these fungi. The chemical fungicides we do have available have been linked to the dramatic increase in cases of cancer, immunological, neurological, reproductive and developmental problems. AMMD, a biopesticide strain of *B. cepacia*, has protective action against forms of root rot there is no chemical treatment for; interestingly, it also shows plant growth promotion even in the absence of disease. To protect the immunocompromised and persons affected with CF against potential exposure to *B. cepacia* as a biopesticide, warning labels have been added, personal protective equipment is required and, all spray applications have been banned.

Dr. LiPuma raised the question - what is a strain “type” in terms of registration? For example the currently registered biocontrol strains are type “Wisconsin”. This a phenotypic designation given for the type of biocontrol properties the strains possess and

is not linked to a genetic type in any way.

Questions raised that we have yet the means to answer include: Do natural sources of *B. cepacia* pose threats to public health? Is there any way to distinguish between a clinically dangerous strain and a commercially useful one? What if there are no significant differences? How are we altering the natural balance of microbes by using one in high concentration over vast areas of land? These and other biocontrol issues were discussed at length during the remainder of the conference.

#### **2.4 Doug Gurian-Sherman, United States Environmental Protection Agency, and Dr. William Yan, Health Canada Pest Management Regulatory Agency**

Dr. Sherman and Dr. Yan reviewed the procedures the US and Canada have for the approval of microbial strains as biocontrol agents. Three *B. cepacia* strains are registered in the US. Further registration of new strains has ceased until review of the risk assessment procedures has been carried out. For registration a strain must be deposited in a recognized culture collection, it may not have been isolated from a clinical source, and it must pass a human safety risk assessment. The risk assessment takes into account the expected route of exposure, the LD<sub>50</sub> in animal studies, gross pathology in animal studies, growth at 37 degrees, natural history of the microbe. Recently, molecular biology has made it possible to use the discovery of genomic virulence factors as exclusion criteria.

A question raised was: once a strain was approved, would it be continually monitored and subjected to new testing criteria as they are developed? The answer was a qualified yes - in that if a concern is raised the process could be set in motion.

An example of a company monitoring their own products was given where after finding, by recent genetic analysis, that a strain proposed for biocontrol carried epidemic lineage markers. The strain was removed from all production without any clinical or lab data indicating it as harmful, only the knowledge that it shared genes with epidemic strains and therefore had the potential to be a threat to health.

The current status of *B.cepacia* biopesticide usage within the US is as follows:

- (i) Residential uses are banned
- (ii) Foliar spray and turf applications are banned
- (iii) No new registrations will be granted until a review of the risk assessment procedures has been carried out.

The US and Canada are in the process of developing a common registration process for pesticides and biopesticides. Discussion of the use of opportunistic pathogens as biocontrol agents will form a key part of this process.

Questions were raised as to whether all government agencies were communicating regarding the usage of *B. cepacia*. For example bioremedial uses of *B. cepacia* would be

governed under the Department of Environment in the US. Dr. Sherman indicated that communication between the regulatory agencies was beginning to occur in the US.

**2.5 Barb Conway, University of Iowa**

Dr. Conway outlined her research at Dr. Peter Greenberg's laboratory which examines the molecular basis for quorum sensing in *B. cepacia*. The pathway and genetic organization of the quorum sensing apparatus was presented.

**2.6 Keith Poole, Queen's University**

Dr. Poole presented data from his laboratory on molecular aspects of drug resistance in *B. cepacia*. The strains he was working with were from genomovars III and VI and he is in the process of cloning and characterizing genes encoding the multidrug efflux pumps and energy coupling protein, TonB.

**2.7 Shawn Aaron, Ottawa General Hospital**

Dr. Aaron presented a scheme for synergy testing of antibiotics effective against multidrug resistant *B. cepacia*.

**2.8 Otakar Nye, FN Motol, Prague**

Dr. Nye presented data from a CF treatment centre in Prague. The resistance of *B. cepacia* isolates recovered from patients was followed over the last few years. In the Prague clinic approximately 20% of patients have *B. cepacia*. An increase in the rate of multidrug resistant isolates was reported and these strains have been genetically investigated by ribotyping. Most of these strains appeared to be unique genetic types.

**3.1 PRESENTATIONS FROM LUNG TRANSPLANTATION GROUPS**

**3.2 Peter Gilligan, University of North Carolina**

Dr. Gilligan presented an overview of the experience of the North Carolina CF transplant centre with *B. cepacia*. A total of 96 patients have been transplanted and 13 of these were positive for *B. cepacia*. Six strains were genomovar III and 4 strains were *B. multivorans*. Five year survival rate for *B. cepacia* positive patients was 33% (versus 54% if negative). One year survival rate for *B. cepacia* positive patients was 60% (versus 84% if negative). In 3 of the 13 *B. cepacia* transplanted patients, fatal sepsis occurred; all three patients had genomovar III strains.

**3.3 Presentation from the Toronto CF Treatment centres**

***B. cepacia* epidemiology in Toronto:**

Dr. Tullis presented an overview of *B. cepacia* infection among CF patients in Toronto. In 1979 the first outbreaks of the ET12/cable pilus strain occurred. In 1992, strict infection control procedures were introduced and the rates of infection have decreased. The following patterns of clinical disease progression have been seen in *B. cepacia* infected patients (predominantly *B. cepacia* genomovar III ET12 strain lineage):

1. Chronic infection without apparent impact
2. Usual progressive disease, steady FEV<sub>1</sub> decline from 20 to 50 years of age
3. Relentless rapid deterioration over months/years, recurrent exacerbations, hospitalization and IV/inhaled antibiotics, fast FEV<sub>1</sub> decline over 10 years.
4. *B. cepacia* Syndrome - progressive necrotizing pneumonia over days/weeks, high fevers, elevated WBC, septicemia, high mortality. This may not always occur immediately following colonization, for example one patient followed profile 1 for 20 years prior to dying of *B. cepacia* Syndrome.

John Govan asked - whats the trigger for *B. cepacia* syndrome? stress? unrelated illness? There were no obvious links but apparent links will be investigated.

John LiPuma - are outcomes evenly distributed among 4 groups? No, and a given patient may switch from one type of decline to another.

#### **Toronto Lung Transplantation experience:**

Dr. Chaparro presented data from the lung transplant clinic Toronto. Since March 1998 there have been 80 double lung transplants and 75 of these were for patients with CF. 39 CF patients were *B. cepacia* positive, 25 have died (versus 7 deaths among the 36 *B. cepacia* negative patients). Clinic has been able to overcome potential infection control problems associated with transplanting *B. cepacia* positive patients; however, they are struggling to improve the survival rates for *B. cepacia* infected patients. Significant mortality appears to occur within the first 90 days of transplantation and ways to improve survival at this point are being examined.

#### **Examination of *B. cepacia* infected lung tissues:**

Dr. Forstner presented data on the immunolocalization of *B. cepacia* in infected lung sections obtained from patient who died after transplantation. Two antibodies were used a) R418 was a polyclonal antibody recognizing all *B. cepacia* strains, b) Anti-22 kDa was a monoclonal recognizing the cable pilin protein of ET12 strains. Interestingly *B. cepacia* was found to be associated with the epithelial airway cells of the lower airways either between septa or within epithelial layer. *B. cepacia* was not seen in lumen of the airway like *P. aeruginosa*. Strain lacking the cable pilus (2 of 8 samples) were also found to be associated with cartilage within the lung tissues. Distribution of *B. cepacia* was different from *P. aeruginosa* being more invasive in acute infections.

#### **4.1 GROUP DISCUSSION TOPICS**

#### **4.2 TOPIC 1 - IDENTIFICATION AND MOLECULAR EPIDEMIOLOGY OF *B. cepacia* (Chair - John LiPuma)**

The following points were raised and discussed by the group:

#### **4.3 What is the current definition of the *B. cepacia* complex?**

The group agreed to keep the term “*B. cepacia* complex” to describe this group of closely related species. There was a real concern that to continue changing the naming of species may lead to the CF community regarding certain strains as “safer” before the epidemiology was fully understood. By keeping the designation as *B. cepacia* complex this would provide greater consistency.

It was noted that:

- (a) genomovar IV will be proposed as new binomial species shortly.
- (b) Group 13 organisms will receive a genomovar designation of genomovar VI of the *B. cepacia* complex.

#### **4.4 How should diagnosis/identification be reported ?**

As *B. cepacia* complex (then either new species designation - *B. vietnamiensis* or *B. multivorans*, or as genomovar if testing for this is available)

eg. *B. cepacia* complex (*B. multivorans*)

eg. *B. cepacia* complex (genomovar III)

#### **4.5 Update on clinical microbiology definition of *B. cepacia* complex**

Additional testing should include the following and clinical diagnostic laboratories should be made aware of the useful information they can provide:

Oxidase

ONPG

Lysine decarboxylase

Sucrose

Lactose

Growth at 42 degrees

Nitrate

#### **4.6 Updates on selective media**

The group advocated the use of selective media in the identification of *B. cepacia*. The *B. cepacia* Selective Agar (BCSA) was now being manufactured by Remel or Hardy Diagnostics (<http://www.hardydiagnostics.com/>). It was noted that certain centres prefer

other selective media (OFPBL or MAST) because they can isolate yeast and fungi that may be present in a sample.

#### **4.7 Commercial systems**

No specific systems were recommended, however the use of specific biochemical testing outlined above should also be applied to assist identification. A general dislike of commercial systems was noted. For example the API strip results were in need of updating to recognise *Burkholderia* species.

#### **4.8 Use of referral laboratories**

This should continue to be recommended by all clinical labs. They can confirm ID, genotype strains and identify genomovar. These parameters are vitally important to understand the global epidemiology of the *B. cepacia* complex. PCR identification was considered not realistic by most clinical diagnostic laboratories and this should be continued to be done by the research referral laboratories.

#### **4.9 Strain Typing**

Genetic methods were recommended (ribotyping and RAPD) and PFGE was specifically identified as a method that the group should be adopting as “standard”. However, this does not exclude the use of other methods for local clustering of strains.

A consensus method for PFGE should be drawn up by the group. Conditions, enzymes and molecular size markers should be standardized. Standardization of equipment and analytical software may be difficult given the costs. Dr. Moore agreed to develop this initiative.

#### **4.10 Correlation of clinical and microbiological data**

Efforts should be made to correlate these data. This may be achieved via the patient data registry. This can in future provide real time analysis and updates on the epidemiology of *B. cepacia* infection.

#### **4.11 Outstanding epidemiological questions**

What is the source of genomovar III strains? Now with increased infection control practices epidemic strains were being reduced however significant numbers of unique genomovar III infections were still being observed. The source of these infections is not clear. The potential divergence of genomovar III *in vivo* was discussed. It was noted that this mechanism of evolution was unlikely in the short time frame in which *B. cepacia* has emerged as significant CF pathogen.

#### **4.12 TOPIC 2 - VIRULENCE, PATHOGENESIS AND MODELS OF *B. CEPACIA***

## **INFECTION** (Chair - David Speert)

A number of points were raised and discussed by this group:

### **4.13. Can one legitimately define virulence determinants in an opportunistic pathogen ?**

The following candidates were suggested:

Outer membrane components ?- LPS induces TNF

- secreted factors
- invasin production ?
- secretion mechanism such as siderophore production

Role of modifying factors ? - host-exuded factors

- other bacterial/viral infections
- the role of injury in triggering virulence ?

### **4.14 What animal models of infection do we have?**

It was agreed that we do not have a good animal that is relevant and specific to *B. cepacia* infection in patients with cystic fibrosis. There were a number of promising models:

**Rat agar bead model**                      There was a concern that this lacked specificity and may “level the field” for all genomovars

**Murine intraperitoneal infection model**      The relevance of this model was questioned and in addition the correlation to human pathogenicity in CF was not obvious e.g. genomovar III strains are generally rapidly cleared and *B. multivorans* strains persist.

**Neutropenic mice**                              The specificity and relevance of this model was also questioned. This system may assist with modelling immunocompromised host but may not be relevant to patients with CF

**Transgenic knockouts**                      The relevance of CF knockout mice as an infection model was also questioned. In addition to the poor modelling of infection such mice are very difficult to work with and expensive. Cytokine knockouts were suggested as future candidates. In addition a large number of different genes have been disrupted and a screen of transgenic mice with knockouts not necessarily related to immunity may identify promising hosts.

**Susceptible species**                              It was noted that a systematic survey of other small animal species has not been carried out with regard to susceptibility to *B. cepacia* infection. A species with an innate susceptibility to *B. cepacia* may exist.

**4.15 In vitro models** It was noted that in vitro models such as epithelial cell (CF and non-CF) invasion, phagocytic cell interaction and infection of amoeba may be used as models in the laboratory.

#### **4.16 Host factors**

**The role of specific CFTR mutations** It was noted that no specific correlation had been made. Does the mutation affect susceptibility? This was unknown

**Primary or secondary role** In plants, *B. cepacia* infection is a secondary event which occurs opportunistically after a wounding event. Is this relevant to human infection ?  
Is the CF lung primed for *B. cepacia* infection by preceding bacterial/viral infection ?

**Human-Plant connection** Some discussion was continued around this point. *B. cepacia* is an opportunistic pathogen/wound pathogen. Does cell leakage induce bacterial growth? Can plant models be used to model human disease. Coordinated studies among members of the group may assist in answering these questions.

#### **4.17 Molecular tools**

The following progress was noted:

**Transposon mutagenesis** This is being more widely applied to *B. cepacia* - Dr. Sokol's laboratory has had great success with the *B. cepacia* genomovar III strain K56-2.

**Site-directed mutagenesis** Systems for allelic replacement and gene disruption have been developed and applied.

**Vector systems** Cosmid and plasmid vectors are available.  
*B. cepacia* phages are being characterized.

**Major problems** Selective markers were not abundant with trimethoprim and tetracycline resistance being most widely used.  
The complex genome, multireplicon structure and intrinsic antibiotic resistance were all considered problematic for working with these organisms.

#### **4.18 TOPIC 3 - TRANSPLANTATION, THERAPY AND CLINICAL PRACTICES IN**

## RELATION TO *B. cepacia* INFECTION (Chair - Debbie Toder)

The following goals and points of discussion were raised relating to general clinical practice with *B. cepacia* infected patients:

### 4.19 Increase rate of strain referral to the research referral labs.

Although many US centres are already participating, this can be increased substantially.

### 4.20 There should be an effort to correlate clinical outcomes with genomovar status

### 4.21 Identification

Identification at the *Burkholderia* species level was still not great (*B. gladioli* and *R. picketti* were still being misidentified as *B. cepacia* complex)

### 4.22 Early diagnosis

More emphasis on ways to determine *B. cepacia* infection prior to culture  
Antibodies to the core LPS may be observed prior to culture, but this may not mean an infection is actually present  
PCR methods are beginning to be applied and have shown some success with sputum testing

### 4.23 Selective media

Questions were raised about the sensitivity of these media - what has to be bacterial load in the lung before positive cultures can be recovered ?

The following points pertinent to transplantation were raised (discussion chaired by George Mallory):

### 4.24 Premise:

*B. cepacia* complex-infected CF patients will continue to experience progressive lung disease and become candidates for lung transplantation

### 4.25

There should be a continuing education and exhortation of transplant programs as to the appropriate microbiological culture techniques for CF microbial flora and they should submit *B. cepacia* complex organisms to the referral laboratories for genomovar testing and identification.

### 4.26

There should be a systematic compilation of worldwide experience (retrospectively) with lung transplantation in CF patients infected with *B. cepacia* complex. This study should include culture results, genomovar ID (if known) and clinical outcomes.

### 4.27

Continuing analysis of CF patient experiences at selected centres (Toronto, University of North Carolina and Birmingham,UK) with lung transplantation in *B. cepacia* complex

infected patients should be encouraged.

- 4.28** Despite disappointing results reported in some CF patients infected with *B. cepacia* complex who undergo lung transplantation, it would be premature and unwise for governmental agencies or private insurers to abrogate the clinical decision by transplant physicians and patients/families as to the indications for transplantation in individual patients.
- 4.29** Clinical Epidemiology: With continuing input of the IBCWG and others, there should be a detailed update in the worldwide incidence and prevalence of *B. cepacia* complex infection in patients with CF with as much information about genomovars as is available. There must be a commitment to openness and sharing of data (as has been the tradition within the IBCWG) and careful attention to minimizing sensationalism or any tendency towards discriminatory treatment of individual patients or treatment centres.
- 4.30** **Synergy testing.** The IBCWG should support and advise Dr. Aaron's proposed multi-centre study of multidrug resistant *B. cepacia* complex bacteria recovered from patient with CF.

## **5.1 PRESENTATIONS FROM OTHER PARTICIPANTS**

Several presentations were made on the evening of Sunday 11 April 1999. The abstracts for these presentations are contained on the IBCWG website (<http://allserv.rug.ac.be/~tcoenye/>).

## **6.1 GENERAL DISCUSSION AND CONCLUDING REMARKS**

- 6.2** The challenge for the IBCWG is to deal with the many outstanding questions concerning *B. cepacia* complex biology, pathogenesis and epidemiology using a multi-disciplinary approach. This will be facilitated by the ongoing collaboration and communication among the diverse members of the IBCWG and other investigators wishing to take part in the initiative.
- 6.3** The clinical challenge will be to apply what we have already learned. For example, in Vancouver and other centres where the strain epidemiology is well understood the epidemic spread of problematic genomovar III strains has been halted by good infection control practices. Such outcomes may not be easy to achieve across all treatment centres.
- 6.4** Should the IBCWG speak more as one voice ? This issue was raised by John Govan who is frequently approached to handle tricky issues concerning *B. cepacia* complex infection. The views of the group as a whole may solicit greater power behind an opinion; however, concern was raised that this may have legal ramifications and lead to problems of confidentiality. Informal communication among IBCWG participants to solicit opinions was already happening and should be encouraged to deal with such matters.

- 6.5** There is a need for consensus. Bob Beall noted that there was an urgent need for consensus now on many issues pertaining to the *B. cepacia* complex. Many of these could not wait until the science had all the answers. He encouraged that guidelines and data should be shared among the group.
- 6.6** **Status of the experimental panel of *B. cepacia* complex strains.** A short note has been submitted to the Journal of Clinical Microbiology describing the strains within the panel. It is hoped that this will be accepted for publication soon. All the strains within the panel will be available from the Belgium Culture Collection in Ghent. An update on the progress of this manuscript will be provided at the North American CF Meeting in Seattle in October. As the make-up of strains within the *B. cepacia* complex changes it is hoped that additions to the panel will be made.
- 6.7** **IBCWG Publications List.** It was agreed that a list of publications which have resulted from the activities of the group should be drawn up and posted on the website.
- 6.8** **4<sup>th</sup> Annual Meeting Abstracts to placed on the website.** Its was agreed that these may be placed on the website and participants who have not submitted abstracts should be encouraged to provide them
- 6.9** **Fifth Annual IBCWG Meeting.** The following was decided about the next annual meeting:
- Where ?** - within the US  
**When ?** - March/April 2000  
**Organizers ?** - Jane Burns and John LiPuma (further planning of the meeting will take place at the IBCWG gathering at the North American CF Meeting)
- 6.10** **Sunday Evening Presentations.** Abstracts for all of these presentations can be found in the meeting program which has been posted on the IBCWG Website (<http://allserv.rug.ac.be/~tcoenye/>).
- 6.11** **Acknowledge sponsors.** The sponsors of the meeting (Zeneca Pharma Canada Inc., the Canadian and US Cystic Fibrosis Foundations, the UK CF Trust and the Canadian Medical Research Council) were acknowledged and the formal meeting was adjourned.

Minutes prepared by Eshwar Mahenthiralingam with assistance from Jocelyn Bischof, Jane Burns and John LiPuma. Equiries regarding the 5<sup>th</sup> Annual International *B. cepacia* Working Group meeting should be directed to Jane Burns or John LiPuma.