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Pseudomonas aeruginosa in the Home Environment of Newly Infected Cystic Fibrosis Patients

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ABSTRACT:

The source of acquisition of *Pseudomonas aeruginosa* in cystic fibrosis (CF) patients remains unknown. Patient-to-patient-transmission has been well documented, but the role of the environment as a source of initial infection is as yet unclear. Here we studied the origin of the first *P. aeruginosa* isolate in CF patients by comparing the *P. aeruginosa* genotype(s) from newly infected patients with genotypes of *P. aeruginosa* isolates from the home environment and from other patients from the same CF centre.

Fifty newly infected patients were studied. *P. aeruginosa* could be cultured from 5.9% of the environmental samples, corresponding to 18 patients. For 9 of these the genotype of the environmental *P. aeruginosa* isolate was identical to the patient's isolate. In total, 72% of the environmental *P. aeruginosa* isolates were encountered in the bathroom. Patient-to-patient transmission within the CF centre could not be ruled out for 3 patients.

In summary, we found a low prevalence of *P. aeruginosa* in the home environment of the newly infected CF patients. The bathroom should be targeted in any preventive cleaning procedures. An environmental source of the new infection could not be ruled out in 9 patients.

Word count: 190

KEYWORDS: Cystic fibrosis, environment, genotyping, *Pseudomonas aeruginosa*

INTRODUCTION

Pseudomonas aeruginosa is the major pulmonary pathogen in patients with cystic fibrosis (CF). Dehydration of mucus during exacerbations and a defective host defence make CF airways prone to chronic infection with *P. aeruginosa* (1, 2). By adulthood, over 80% of patients are infected with this pathogen, which adversely affects lung function and survival (3, 4).

To date, the source of initial infection of CF patients with *P. aeruginosa* remains unknown. Possible sources include the environment, person-to-person spread (especially from another person with CF), or through contact with contaminated objects. Patient-to-patient transmission has been well documented. It generally results from prolonged social contact such as that between siblings (5), between close friends or between people staying at holiday camps (6, 7) and CF rehabilitation centres (7, 8).

Epidemiological studies have not confirmed the acquisition of *P. aeruginosa* from the environment. As *P. aeruginosa* is widely present in soil, plants and water (9, 10), the environment could be the initial source of infection. The lack of knowledge on the exact role of the environment in the acquisition of *P. aeruginosa* by the CF patient is of concern to parents and physicians and sometimes leads to questionable preventive measures, such as not drinking tap water unless it has been boiled or not visiting swimming pools (11, 12).

In an attempt to elucidate the source of new strain acquisitions we carried out a Belgian multicentre study. For all patients who were not initially infected with *P. aeruginosa* but for whom a recent new infection with *P. aeruginosa* could be documented, cultures of wet surfaces were taken at their homes as soon as possible after detection of the infection. DNA fingerprints of the *P. aeruginosa* isolates from the newly infected patients were compared with fingerprints of *P. aeruginosa* strains recovered from the home environment. Using the Belgian inventory of the DNA fingerprints of most patient *P. aeruginosa* isolates (7), we

compared the DNA fingerprints of the *P. aeruginosa* isolates from the newly infected patients with the fingerprints of *P. aeruginosa* isolates from chronically infected CF patients attending the same CF centre as the newly infected patients.

METHODS

Study population and sampling

Between January 2003 and December 2005 five Belgian CF centres sent sputum, nasopharyngeal aspirate or an isolate of newly *P. aeruginosa*-infected patients to the microbiology laboratory of Ghent University Hospital (GUH) for culture, confirmation of the identification and genotyping of the *P. aeruginosa* isolates. Approval of the Ethics Committee of the GUH was obtained. In each centre, *P. aeruginosa*-negative CF patients were seen at 3-monthly intervals in segregated consultation rooms or on different consultation days. All newly infected patients were treated for 3 months with oral ciprofloxacin (15 mg/kg bid) and inhaled colistin (2.10^6 U bid) according to the international guidelines (13). As soon as possible after the diagnosis of a first *P. aeruginosa* infection, the CF centre nurse visited the home of the patient and collected environmental samples. Samples were taken from the nebulizer, the taps and sink in the kitchen, the taps and drains of the bath, the shower and washbasin in the bathroom and the toilet(s). Samples from other wet surfaces, e.g. swimming pools and aquaria, were taken at the discretion of the nurses. A sterile swab (Nuova Aptaca) was used to sample wet surfaces. Five ml of surface water was collected from toilets, swimming pools and aquaria by means of a sterile syringe. The medication container of the nebulizer was rinsed with 5 ml sterile saline, which was poured into a sterile sample container.

Microbiology

Sputum and environmental samples were inoculated onto McConkey agar (BBL Becton Dickinson, Cockeysville, MD, USA.). After two days of incubation at 37 °C, morphologically different lactose-negative colonies were picked, subcultured on 5% sheep blood agar (BBL) and tested for oxidase. Only oxidase-positive colonies were further identified, using tDNA-PCR (14).

Genotyping

For each patient, all *P. aeruginosa* isolates exhibiting different colonial morphology on McConkey were first genotyped using alkaline cell lysis for DNA extraction and the random amplified polymorphic DNA fingerprinting technique with Ready-to-Go beads (Amersham Biosciences AB, Uppsala, Sweden) and primer ERIC2 (AAGTAAGTGACTGGGGTGAGCG) at an annealing temperature of 35 °C, as described previously (8). This approach is designated ERIC2-PCR in this study. This enabled us to reduce the number of isolates that were subsequently genotyped by the more laborious fluorescent amplified fragment length polymorphism analysis technique (fAFLP) (7), since only single representatives of each ERIC2-PCR type were further genotyped by this procedure.

RESULTS

Table 1 represents the epidemiological and sampling data of the patients included. Genotyping of the *P. aeruginosa* was performed for 50 patients (26 male, 2 sex not recorded, median age 7 years and 11 months, range 9 months – 31 years and 4 months), including one sibling pair. A total of 427 environmental samples were obtained and genotyped with a median number of 8 samples per patient (mean: 8.5 samples, range: 4 – 23 samples). Nurses were asked to collect a minimum of four samples per home, with at least one sample from the

kitchen, bathroom, toilet and the patient's nebulizer. Complete sampling was carried out for 39/50 patients. Overall, one-third and one-fifth of the samples originated from the bathroom and the kitchen respectively (Table 2), for which 70.8% originated from the drains. The environmental samples were taken within one month of the positive sputum culture for *P. aeruginosa* in 37 of the 50 patients and within one week for 29 of these 37 patients (Table 1). Environmental samples were also taken and processed for 3 patients for whom isolation of *P. aeruginosa* by the referring centre could not be confirmed by the reference laboratory. For one of these patients an identical *P. aeruginosa* genotype was encountered in the sink drain 6 months before the first confirmed isolation of *P. aeruginosa* in the patient. A total of 25 environmental samples from 17 houses harboured *P. aeruginosa*. This corresponded to 18 patients, including one sibling pair. The median number of samples taken from houses with *P. aeruginosa* (9 samples per house) was not statistically different from the median number taken from all houses (8 samples per house) and the locations from which the samples were taken (i.e. bathroom, kitchen or drain, tap) did not differ globally either. The vast majority of *P. aeruginosa*-positive samples were recovered from the bathroom (i.e. 18 out of 25, or 72%). For twelve of the 25 patients for which *P. aeruginosa* could be recovered from the house, the house isolate was different from the patient isolate. These 12 samples came from 9 patients (including one sibling pair) at 8 different houses. Thirteen of 25 environmental samples harboured a *P. aeruginosa* with an identical genotype compared to that of the patient. The 13 samples came from 9 patients at 9 different houses. *P. aeruginosa* was found in only one of the 75 samples taken from the 48 nebulizers of the 50 patients. The latter *P. aeruginosa* isolate had an identical genotype to that of the patient. Another 6 nebulizers from 6 different patients harboured other bacteria, with one nebulizer contaminated with *S. maltophilia.* This *S. maltophilia* genotype was different from the genotypes of the two positive samples for *S. maltophilia* found in the home environment of this patient. However,

this patient had no positive sputum culture for *S. maltophilia*. The other bacteria cultured from the nebulizers are not usually considered to be of pathological significance for CF patients. It should be noted that in our study the nebulizer samples were only processed to recover gram-negative organisms. *P. aeruginosa* could not be recovered from any of the 3 private swimming pools of our CF patients. The sibling pair in the study (patients nos. 18 and 21) were apparently simultaneously newly infected by the same strain of *P. aeruginosa*. The source of infection could not be identified since the environmental *P. aeruginosa* recovered had a different genotype. Consulting the previously published national Belgian database of *P. aeruginosa* genotypes (7), the *P. aeruginosa* genotypes of the newly infected patients were also compared with the genotypes of *P. aeruginosa* cultured from chronically infected CF patients followed at the same centre. In 3 patients, followed at 2 different centres, the genotypes of the newly acquired *P. aeruginosa* strain was identical to the genotype of already chronically infected patients from their own centres.

DISCUSSION

P. aeruginosa is the most important pathogen in patients with CF. The origin of *P. aeruginosa* infection in patients with CF has not yet been clearly established. The role of the environment as a source of *P. aeruginosa* acquisition in CF patients is still difficult to ascertain and remains a matter of debate (11, 12). Patients with CF rarely appear to share genotypes, unless they are siblings or close friends, suggesting that patient-to-patient transmission is rare and requires intense and long-standing contact. *P. aeruginosa* is a typical water organism and has been isolated from a number of environmental reservoirs (9, 10). The wide distribution of *P. aeruginosa* genotypes established in young CF children suggests acquisition from environmental reservoirs (15). *P. aeruginosa* has been recovered from environmental sources in both in- and out-patient health care settings: e.g. sinks, tap water in a paediatric ward (16), toys, baths, hand soaps (17), pulmonary function equipment, hospital drains (18), whirlpools

(19), dental equipment (20), hands of health care workers and CF patients (16-18). The possibility of environment-to-patient transmission of these strains has been studied: Doring *et al*. (16) described the possibility of environment-to-patient transmission of *P. aeruginosa* in a hospital setting. He suggested that the ability of CF sputum to facilitate survival of *P. aeruginosa* may be an important issue for strain transmission. During a cross-infection outbreak Jones *et al.* (21) looked for the presence of epidemic strains in the environment of a CF centre and concluded that aerosol dissemination may be the most important factor in patient-to-patient spread of epidemic strains of *P. aeruginosa*.

In contrast to the extensive studies on the presence of *P. aeruginosa* in the hospital environment (16-20, 22-24) and on patient-to-patient transmission, there are only a few reports on the prevalence of *P. aeruginosa* in the home environment of both CF and non-CF patients (Table 3) (22, 25-28). These studies provide conflicting results. None of them studied the possibility of environment-to-patient transmission and neither did they compare genotypes of *P. aeruginosa* strains recovered from patients or from their home environment.

To the best of our knowledge, this is the first study to look for the presence of *P. aeruginosa* in the home environment of newly *P. aeruginosa*-infected CF patients and to compare genotypes of the environmental strains with the genotypes found in the patients.

In our study, *P. aeruginosa* could be cultured from 5.9% of the home environmental samples in 34% of the houses. Our figures are comparable to figures found in non-CF houses, although comparison is difficult because of different methodologies. We report a lower environmental contamination with *P. aeruginosa* in CF houses compared to the other studies. The different findings possibly might be explained by i) different sampling sites, i.e. we did not study vegetables, which were an important source of *P. aeruginosa* in the study of Mortensen *et al.* (27), ii) a different sampling methodology, i.e. 100 ml aliquots of tap water were collected in the study of Barben *et al.* (26) vs 5 ml aliquots in this report and cultures of

standing water before the taps were used in the mornings in the studies of Barben *et al.* (26) and Regnath *et al.* (25), iii) different cleaning procedures carried out by the patients, and iv) probably most importantly, the different colonization status of the CF patients, i.e. non-colonized patients in our study, and *P. aeruginosa*-colonized patients in the other studies. However, the colonization status is not always explicitly described.

Although previous studies occasionally detected strains with the same (geno)type in the health care environment and in patients (16, 17, 24, 29), it remains unclear whether patients were the initial source of environmental contamination or whether the strains from the contaminated environmental source infected the patients. In our study, for 9 of the patients the genotype of the environmental *P. aeruginosa* isolate was identical to that of the *P. aeruginosa* isolated from the patient's sputum or nasopharyngeal aspirate. Therefore, an environmental strain could have been a possible source of infection for only 9/50 (18%) of the newly *P. aeruginosa*-infected CF patients. Because regular prospective home environmental cultures before the first positive *P. aeruginosa* infection in the patient are lacking, in this study too we cannot definitely conclude that the environmental strains were the source of infection or conversely that contamination of the environment was caused by the patient. However, our study indicates that there is no overwhelming *P. aeruginosa* contamination of the home environment of newly infected CF patients. As the vast majority of environmental *P. aeruginosa* isolates were found in the bathroom of the patients, it would be advisable to pay special attention to the cleaning and disinfecting procedures for bathrooms. Compared to data from other studies (30, 31), contamination of the nebulizers with *P. aeruginosa* in our study was low. These findings suggest that the specific disinfecting procedures of the nebulizer equipment, as advised by the medical committee of the Belgian CF patient association (32), are effective.

In only 3 of the 50 newly infected patients (6%) could there have been patient-to-patient transmission within the same CF centre. As far as traceable, this transmission remains unexplained, since there was no close relationship between the patients, they were seen in segregated consultations, they were never hospitalized at the same time and they never attended the same CF rehabilitation centre or CF summer camp. None of these genotypes belonged to a cluster of genotypes established previously for Belgian colonized cystic fibrosis patients (7). These results confirm the low patient-to-patient transmission percentages after the introduction of segregation measures in the CF centres (33).

Because our study results suggest that neither the home environment nor other patients are important sources of *P. aeruginosa* infection in CF patients, the question remains regarding the actual source of a first infection. Apart from wet surfaces, the potential sources of *P. aeruginosa* in the home environment mentioned in literature are washing cloths, sponges for cleaning (28) and vegetables (27, 34). As *P. aeruginosa* is widely spread in soil and water, acquisition from the environment outside the house is probable. More studies are needed to elucidate the role of these potential infecting sources of *P. aeruginosa* in CF patients.

In conclusion, this is, to our knowledge, the first study to look for the presence of *P. aeruginosa* in the home environment of CF patients newly infected with *P. aeruginosa* and compare genotypes of environmental *P. aeruginosa* with the genotypes of the strains recovered from these patients. We report low percentages of possible home environment-to-patient transmission (18%) and of patient-to-patient transmission within the same CF centre (6%). In addition, our study clearly shows that there is no overwhelming contamination level with *P. aeruginosa* in the home environment of this population of newly *P. aeruginosa*-infected patients. Contamination seems to occur mainly in the bathroom of the patient, suggesting that this place should be targeted for more rigorous cleaning procedures. More

studies are needed to elucidate the role of other potential infecting sources of *P. aeruginosa* in CF patients.

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Patient	Centre	Gender	CFTR genotype	Age (y m)*	Time interval sampling†	Number of environmental samples	Sampling remarks	Genotypes patient and environment	
								Identical	Different
1	1	M	p.[delPhe508]+[delPhe508]	19 01	< 1 w+	3	No bathroom		
2	1	M	p.[delPhe508]+[Glu656X]	07 04	1 m+	5			
3	1	M	p.[delPhe508]+c.[1703delT]	25 05	< 1 w+	4	No kitchen		
4	1	F	p.[delPhe508]+[Arg1070Trp]	01 00	< 1 w+	3	No kitchen		
5	1	M	p.[delPhe508]+[delPhe508]	15 03	6 m-	5		Kitchen washbasin drain	
6	1	F	p.[delPhe508]+[delPhe508]	20 07	< 1 w+	5	No kitchen		
7	1	F	p.[delPhe508]+[delPhe508]	23 08	6 m+	4	No kitchen		
8	2	F	p.[delPhe508]+c.[1585G>A]	04 00	< 1 w+	11			
9	2	M	c.[3528delC]+[3140-26A>G]	02 07	< 1 w+	11			
10	2	M	p.[delPhe508]+c.[1585G>A]	00 09	2 m+	10			
11	2	F	p.[delPhe508]+[delPhe508]	01 00	< 1 w+	10		Bathroom bath drain = Bathroom bath tap	
12	2	F	p.[delPhe508]+[delPhe508]	28 00	< 1 w+	9			Bathroom bath drain <> Bathroom washbasin drain
13	2	M	p.[delPhe508]+[delPhe508]	23 09	2 w+	10	No toilet		
14	2	M	p.[delPhe508]+[delPhe508]	02 04	< 1 w+	9	No aerosol		
15	2	F	p.[delPhe508]+[delPhe508]	04 05	3 w+	10			
16	2	M	p.[delPhe508]+[Ser1251Asn]	17 03	1 m+	10			Kitchen sink drain
17	2	M	p.[delPhe508]+[delPhe508]	03 05	< 1 w+	10		Bathroom shower drain	
18	2	F	p.[delPhe508]+[delPhe508]	04 07	< 1 w+	8‡			Bathroom shower drain. See patient 21
19	2	M	p.[delPhe508]+[Ala455Glu]	04 04	< 1 w+	10			
20	2	F	p.[delPhe508]+[delPhe508]	03 07	2 w+	7		Bathroom washbasin drain = Kitcchen sink drain	
21	2	M	p.[delPhe508]+[delPhe508]	02 10	< 1 w+	8‡			Bathroom shower drain. See patient 18
22	2	F	p.[delPhe508]+[Arg553X]	02 11	< 1 w+	10			
23	2	F	p.[delPhe508]+[delPhe508]	00 11	< 1 w+	10	No bathroom	Kitchen sink drain	
24	2	M	p.[delPhe508]+[Iledel507]	06 01	2 w+	8			
25	3	F	p.[delPhe508]+[delPhe508]	05 00	< 1 w+	6			
26	3	M	p.[delPhe508]+[delPhe508]	15 02	4 m-	6			
27	3	F	p.[delPhe508]+[delPhe508]	08 02	< 1 w+	9			
28	3	M	p.[delPhe508]+[Ser1251Asn]	07 11	7 w+	5			
29	3	F	p.[delPhe508]+[Arg553X]	10 04	3 m+	10			Bathroom bath drain
30	3	F	p.[delPhe508]+[delPhe508]	06 01	< 1 w+ and 5 m+	16+9			Bathroom shower drain <> 2 x Bathroom bath drain§

31	3	M	p.[delPhe508]+[delPhe508]	31 04	< 1 w+	5			
32	3	M	p.[delPhe508]+[Ser1251Asn]	01 04	6 w+	7			
33	3	F	p.[delPhe508]+[delPhe508]	11 00	< 1 w+	10			
34	3	F	p. [delPhe508]+[Cys276X]	09 05	3 w+	11	No nebulizer		
35	3	M	p.[delPhe508]+[Gly542X]	03 07	< 1 w+	7			
36	3	F	p.[delPhe508]+[delPhe508]	14 01	< 1 w+ and 5 w+	5			
37	3	M	p.delPhe508]+[delPhe508] [Ser1251Asn]	16 00	2 w+	10			
38	3	F	p.[delPhe508]+[delPhe508]	11 11	< 1 w+	12			
39	3	M	p.[delPhe508]+[delPhe508]	07 06	< 1 w+	6			
40	3	M	p.[delPhe508]+[delPhe508]	08 05	7 m- and 7 w+	10+4			
41	3	M	p.[delPhe508]+[delPhe508]	13 04	4 m+	13		Toilet first floor = Toilet second floor	
42	3	M	p.[delPhe508]+[delPhe508]	20 04	< 1 w+	5	No kitchen	Nebulizer	
43	3	F	c.[1585-1G>A]+[?]	02 00	< 1 w+	8		Bathroom bath drain = Bathroom washbasin	
44	3	M	p.[delPhe508]+[Asn1303Lys]	05 01	14 m -	7			Bathroom toilet
45	4	M	NA	13 01	2.5 m+	10			
46	4	NA	NA	08 08	< 1 w+	8			Bathroom washbasin tap
47	4	NA	NA	09 06	< 1 w+	7	No toilet		
48	5	M	p.[delPhe508]+[Asn1303Lys]	12 10	< 1 w+	12			
49	5	F	p.[delPhe508]+[delPhe508]	09 07	< 1 w+	16			Bathroom wasbasin drain = Kitchen sink tap
50	5	F	p.[delPhe508]+[delPhe508]	02 09	< 1 w+	9		Bathroom bath drain	

Table 1 : Patient and Sampling Characteristics.

* y : year, m : month
† w : week, m : month, + : environmental sampling later than patient sampling, - : environmental sampling prior to patient sampling.
‡ Six samples shared for patients 18 and 21. For each patient an additional two samples were obtained from the nebulizer.
§ Bathroom bath drain samples taken with an interval of 4 months.
NA : not available

Sampling Site	Number of Samples (Percentage)	Number of Positive Samples		Percentage of Positive Samples
		Identical *	Different †	
Bathroom	**146 (34.2)**	**8**	**10**	**12.4**
Bath drain	39	3	4	17.9
Bath showerhead	2	0	0	0
Bath tap	7	1	0	14.3
Shower drain	17	1	2	17.6
Shower tap	3	0	0	0
Showerhead	8	0	0	0
Toilet	5	0	1	20.0
Toothbrush	1	0	0	0
Washbasin drain	49	3	2	10.2
Washbasin tap	15	0	1	6.7
Kitchen	**87 (20.4)**	**2**	**2**	**4.9**
Sink drain	60	2	1	5.0
Sink tap	22	0	1	4.5
Other	5	0	0	0
Toilet room	**68 (15.9)**	**2**	**0**	**2.9**
Toilet	56	2	0	3.6
Washbasin drain	11	0	0	0
Washbasin tap	1	0	0	0
Nebulizer	**75 (17.6)**	**1**	**0**	**13.3**
Diverse	**51 (11.9)**	**0**	**0**	**0**
Animal	1	0	0	0
Aquarium	7	0	0	0
Bedroom	3	0	0	0
Plant	3	0	0	0
Swimming pool	3	0	0	0
Tumble dryer	2	0	0	0
Washing machine	3	0	0	0
Washroom	6	0	0	0
Other inside house	19	0	0	0
Other outside house	4	0	0	0
TOTAL	**427**	**13**	**12**	**5.9**

Table 2 : Sampling Details
Legend :
* : identical = identical genotype of the environmental isolate compared to the genotype from the patient genotype
† : different = different genotype of the environmental isolate compared to the genotype from the patient genotype

Study	Study population	Sampling method	Sampling sites	Culture medium	Positive/Total Samples (percentage)
Regnath 2004*	102 houses of 118 CF patients	Sterile swab	Bathroom washbasin drain	Cetrimid agar & Acetamide Broth	35/101 (34.7)
		Water sample	Bathroom washbasin tap	Cetrimid agar & Acetamide Broth	1/102 (1)
		Sterile swab	Bathroom shower drain	Cetrimid agar & Acetamide Broth	40/101 (39.6)
		Sterile swab	Bathroom showerhead	Cetrimid agar & Acetamide Broth	6/100 (6)
		Direct agar contact plate method	Bathroom bath	Rodac plate	5/102 (4.9)
		Sterile swab	Toilet washbasin drain	Cetrimid agar & Acetamide Broth	3/66 (4.5)
		Water sample	Toilet drain	Cetrimid agar & Acetamide Broth	27/102 (26.5)
		Sterile swab	Kitchen sink drain	Cetrimid agar & Acetamide Broth	35/100 (35)
		Water sample	Kitchen sink tap	Cetrimid agar & Acetamide Broth	7/102 (6.9)
Barben 2005†	50 houses of 50 CF patients		Bathroom washbasin tap		
		100 ml water	Standing water winter	Cetrimid agar	0/51 (0)
		100 ml water	Running water winter	Cetrimid agar	0/51 (0)
		100 ml water	Standing water summer	Cetrimid agar	2/50 (4)
		100 ml water	Tunning water summer	Cetrimid agar	0/50 (0)
Mortensen 1995‡	14 houses of 14 CF patients	Sterile swab	Total drains §	McConkey agar	10/106 (9.4)
Mortensen 1995	13 houses in same area as CF houses	Sterile swab	Total drains§	McConkey agar	10/106 (9.4)
Whitby 1972 **	33 houses of 33 hospital laboratory staff members	Sterile swab	Bath, sink and washbasin drains	Modified King's medium B	7/114 (6.1)
		Sterile swab	Bath, sink and washbasin drains	Modified King's medium B	1/21 (4.8)
Ojima 2002	86 Japanese houses	Direct agar contact plate method	Kitchen sink tap	NAC agar II	6/83 (7.2)††
		Direct agar contact plate method	Kitchen sink drain	NAC agar II	23/85 (27.1) ††
		Direct agar contact plate method	Kitchen sink tap	NAC agar II	11/85 (12.9) ††
		Direct agar contact plate method	Bathroom bath	NAC agar II	11/86 (1.2) ††

Direct agar contact plate method	Bathroom showerhead	NAC agar II	0/5 (0) ††
Direct agar contact plate method	Toilet room sink	NAC agar II	0/79 (0) ††
Direct agar contact plate method	Washroom sink	NAC agar II	1/43 (2.3) ††

Table 3 : Studies on the prevalence of *P. aeruginosa* in CF and non-CF houses.

* *P. aeruginosa* colonisation status of patients known for 88 patients, of which 28 were colonised. Age 7 months to 41 years (mean 11 years), *P. aeruginosa* was detected in 71.6% of the CF-houses.
† Ages and colonisation status for *P. aeruginosa* of patients unknown.
‡ Patients colonized with *Burkholderia cepacia*. Ages and colonisation status for *P. aeruginosa* of patients unknown.
§ total drains = kitchen sink drains + shower heads + refrigerator drain pan + bathtub drains
II Nalidixic Acid Cetrimide Agar (Nissui Pharmaceuticals, Tokyo, Japan)
†† Number of houses with *P. aeruginosa* in the specified location/total number of houses examined for this location (Percentages of households in which *P. aeruginosa* is detected).
** *P. aeruginosa* detected in 4/33 houses.