increased sputum production. He received meropenem, ceftazidime and amikacin. He persisted with fever and hypercapnia so noninvasive ventilation was initiated. Later, multiresistant *Burkholderia contaminans* was isolated from sputum and blood cultures. He persisted with fever and the CT scan showed deterioration so it was assumed as cepacia syndrome. Antibiotic synergy tests were performed. Aggressive treatment with 5 IV antibiotics: Meropenem, piperacillin tazobactam, levofloxacin, minocycline and chloramphenicol (40 days), oral corticosteroids (5 days) and cyclosporine (14 days), resulted in clinical and biochemical resolution of his cepacia syndrome (negative blood cultures, no fever, C-reactive protein and arterial blood gases normalized).
Serum drug dosage was performed for cyclosporine, and therapeutics levels were insured.
In spite of this treatment, the patient finally died one month later due to respiratory failure.
Cepacia syndrome is characterized by rapidly progressing radiological and clinical signs of necrotizing pneumonia, pyrexia and an almost universally fatal outcome. In our patient the combination of antibiotics, corticosteroids and cyclosporine was a beneficial treatment, and may be could be considered as a therapeutic option.

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Cross-infection with *Pseudomonas aeruginosa*, *Burkholderia cepacia* complex and *Achromobacter* spp. between people with cystic fibrosis in Russia

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Objectives: Patients with cystic fibrosis (CF) attending a clinic are at high risk of cross-infection with different bacteria that can be harmful to them. We aim to assess the incidence of cross-infection with *P. aeruginosa*, *B. cepacia* complex (Bcc) and Achromobacter spp. between people with CF in Russia.
Methods: Over a 7-year period 175 strains of *P. aeruginosa* from 90 patients, 171 Bcc strains from 105 patient and 46 Achromobacter spp. strains from 36 patients were isolated and genotyped by RAPD-PCR and MLST to determine whether cross-infection was occurring.
Results: The genotyping of *P. aeruginosa* strains showed that the vast majority of patients (81%) retained their own unique strain. Only five strains appeared in more than one unrelated patients. These results suggested that the environment was the most important source of *P. aeruginosa* for CF patients in Russia. The genotyping of Bcc strains showed that 95,2% of patients were infected by *B. cenocepacia*. Among them 67,6% were colonized with epidemic genotype ST 709 and 12,4% - with genotype ST 208. The 87,2% of Achromobacter spp. strains belonged A. ruhlandii. The genotyping revealed that 28,6% of strains belonged to A. ruhlandii ST36. Cross-contamination with the Bcc ST709, ST208 and A. ruhlandii ST36 is supposed to occur during hospitalizations and clinic visits in Moscow and Samara respectively.
Conclusion: The incidence of cross-infection with *P. aeruginosa* among unrelated CF patients in Russia is very low. On the contrary, the incidence of cross-contamination with Bcc and A. ruhlandii is high. Unique genotype of *B.* cenocepacia ST709 now account for the majority of *B. cenocepacia* chronic infections within CF patient in Russia. As a result of our study it is was recommended that CF patient with Bcc and A. ruhlandii infection should be segregated.

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Habitat and diversity of *Achromobacter* in households of chronically colonized cystic fibrosis patients

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Objectives: *Achromobacter* spp. are emerging Cystic Fibrosis Respiratory Tract (CFRT) pathogens likely to originate from the environment. *Achromobacter xylosoxidans*, the major species identified in CFRT, has been identified in hospital, houses and natural environments but its habitat and reservoir remain partially described. We described *Achromobacter* distribution and diversity in houses of CF patients chronically colonized by *A. xylosoxidans* with the aim to precise the role of environmental reservoirs in the patients' persistent colonization.
Methods: We performed 273 samples in 3 houses of CF patients, including water and surface samples. Non selective medium cultured communities were studied by using a specific PCR for *Achromobacter*. For PCR-positive communities, colonies obtained on specific medium were identified by MALDI-TOF® mass spectrometry and multilocus sequence typing (MLST). Environmental strains were compared to patients' strains by MLST and Pulsed-Field Gel Electrophoresis.
Results: *Achromobacter* spp. were isolated from 5.5% of the samples, mainly from moist or water-related samples. High species and genotypes diversity was found both among and within houses and samples. *A. xylosoxidans* was not the major species and *Achromobacter animicus* and *Achromobacter mucicolens* were described for the first time from environmental sources. The bacterial genotype colonizing the patient was never found in his domestic environment.
Conclusion: Patients' houses hosted a high diversity of *Achromobacter* species and genotypes but genotypes chronically colonizing the patients were never found in their environment dismissing the idea of an environmental reservoir that could be incriminated in persistent CFRT colonization. These results suggested that after initial colonization from an environmental source and subsequent specialization to the CFRT, the survival of adapted *A. xylosoxidans* strains in the environment is probably impaired.

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Colistin resistance in *Achromobacter* sp. and *Pseudomonas aeruginosa* isolated from Danish cystic fibrosis patients is not related to plasmid-mediated expression of *mcr-1*

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Objectives: Colistin is an antibiotic of major importance for the treatment of pulmonary infections in cystic fibrosis (CF). Recent reports of the global spread of a new plasmid-mediated resistance mechanism to colistin, *mcr-1*, can have major implications for the treatment of CF patients. We investigated the occurrence of the *mcr-1* gene in colistin resistant *Pseudomonas aeruginosa* and *Achromobacter* species isolated from sputum samples from cystic fibrosis (CF) patients from Aarhus University Hospital, Denmark.
Methods: 38 *P. aeruginosa* and 28 *Achromobacter* isolates obtained between 2008 and 2016 had colistin minimum inhibitory concentrations of greater than 2 mg/L. One isolate was included per patient. The isolates were investigated for the presence of the *mcr-1* gene by polymerase chain reaction (PCR) as described by Liu et al.
Results: The *mcr-1* gene was not detected in 66 colistin resistant *P. aeruginosa* and *Achromobacter* isolates obtained from cystic fibrosis patients.
Conclusion: Although the *mcr-1* gene was not found in this study, it does not diminish the importance of screening for this gene in colistin resistant CF isolates to prevent a potential dissemination of colistin resistance within the CF population.

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Mechanism of β-lactam resistance in Achromobacter xylosoxidans

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Objectives: Bacteria of genus Achromobacter are emerging pathogens in cystic fibrosis with a troublesome propensity to develop pan-resistance to

antimicrobial agents. We induced resistance to β-lactams in two strains of Achromobacter xylosoxidans and assessed the accumulation of DNA mutations.
Methods: A clinical strain (J-11_22405) and the type strain (LMG_1863T) of A. xylosoxidans were cultured under increasing concentrations of piperacillin-tazobactam or meropenem untill high-level resistance occured. Isolates were subjected to whole-genome sequencing, and single-nucleotide polymorphisms (SNPs) and indels were searched for.
Results: Resistant isolates of J-11_22405 acculmulated 150–200 SNPs. All four isolates carried identical, non-synonymous mutations (Pro ->Thr) in a metallo-β-lactamase super family protein; in addition, one strain encoded a frame-shift mutation in a transcriptional regulatory protein upstream of a putative β-lactamase. The two piperacillin-resistant isolates of the type strain encoded the same (Arg ->His) substitution in the β-lactamase class D gene, the same Cys ->Gly substitution in a fosmidomycin resistance protein, and several substitutions in ftsI; however, one strain encoded a Val ->Ala substitution in the RND-type multidrug efflux pump.
Conclusion: Several putative targets that may be decisive for development of β-lactam resistance in A. xylosoxidans have been identified. The substitutions are being searched for in clinical isolates, and will be stepwise inserted in a laboratory strain.

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Cystic fibrosis respiratory microbiology in the western side of Romania

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Objectives: We aimed to audit the spectrum of pathogens among our CF patients.
Methods: An observational, cross-sectional transversal study including fifty seven patients evaluated the prevalence of CF-related microbes in the study group and their pulmonary status.
Results: The most frequent microbe found in our CF group was *Staphyloccocus aureus*, closely followed by *Pseudomonas aeruginosa*. Bacillus tuberculosis was a rare germ, despite the important frequency in our country. Regarding the distribution of infections in the group of ages, the infancy period was' dominated' by the presence of methicillin resistant *Staphylococcus aureus* (MSSA), while for the 6–12 years group, *P. aeruginosa* was found in an equal percentage of 14% with MSSA. *Pseudomonas* prevalence was found in 14.0% of adults and the combined infections were diagnosed in about a fifth of our patients.
Conclusions: We concluded that the percentage of respiratory infections with redoubtable microbes is still moderate, compered with previous reports in the country. This might be an effect of segregation and recent antibiotherapy regimes adopted. The presence of underweight among CF patients with severe mutations are risk factor for a worse outcome and measures should be instituted.

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Emerging pathogens in cystic fibrosis pediatric patients (CF): cross-sectional reports during the last 30 years

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Our laboratory has been studying bacterial colonization of respiratory secretions in pediatric CF patients since 1987. During these 30 years significant changes have been observed.
Objective: To analyze emerging pathogen changes between 1987 and 2016.
Material and methods: A cross sectional study was designed. Every sample (sputum, oropharyngeal swab and BAL) taken in 1987, 1996, 2006 and 2016 were included. Samples were plated on 5% blood agar, chocolate agar, CLED agar, Chapman Medium, BCSA and Cetrimide Agar.
Results: 411 pt, aged 0.1–21.7yo were included

Table:
Emerging pathogens Prevalence

	1987	1996	2006	2016	Logistic regression OR [95% CI]	p
Patients	77	92	151	162		
Samples (median per pt)	115 (1)	238 (2)	447 (3)	723 (4)		
MRSA (%)	5.2	22.8	31,1	34.6	OR = 1.054/year [1.031; 1.076]	(p < 0.001) (1987–2016)
SaSCV*(%)	0	0	0	11.7		(p < 0,001) (2006–2016)
BCC*,**(%)	2.6	1.1	11.9	9,3	OR = 1.050/year [1.014; 1.093]	(p < 0,011) (1987–2016)
Achromobacter spp. (%)	0	0	6.6	8	OR = 1.099/year [1.041; 1.17]	(p < 0,002) (1987–2016)
S. maltophilia (%)	0	0	4	13.6	OR = 1.21/year [1.118; 1.365]	(p < 0,001) (1987–2016)
Mycobacterium spp. (%)	0	0	0	1.9		(p = 0.9) (1987–2016)

*SaSCV (*S. aureus* Small Colony Variant)/BCC (*B. cepacia* complex)
**After an outbreak of BCC in 2005, its prevalence was decreasing until 3.7% in 2015.

Conclusions: During the last 30 years, MRSA colonisation has significantly increased and *S. aureus* SCV started being identified. BCC colonisation peaked in 2005 due to an outbreak, and a new increase was found actually. Prevalence of *Achromobacter* and S.maltophilia has also increased significantly and *Mycobacterium* spp. was also recently identified in our CF population.
These emerging pathogens should be recognised and considered important by the microbiological CF laboratories.

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Chryseobacterium respiratory tract infections in pediatric patients with cystic fibrosis

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Chryseobacterium is a group of Gram-negative, nonfermenting, catalase-positive, oxidase-positive and indole-positive aerobic bacilli. According to the published literature its isolation seems to be increasing in patients with cystic fibrosis (CF).
Objectives: To assess the frequency of *Chryseobacterium* spp. isolates and to analyze clinical outcome and resistance patterns in a 7-year period.
Methods: A retrospective microbiological analysis of respiratory samples obtained from CF pediatric patients at a center in Argentina between 2009 and 2016 was made by using VITEK 2 compact. Age of infection acquisition and antimicrobial susceptibility profile were assessed. Outcomes such as Z score W/A, H/A, BMI, FEV_1 and co-infection (defined as sputum culture positive for more than one microorganism) were measured 12 and 6 months before and after isolation.
Results: The prevalence of *Chryseobacterium* infection was 14% in our cohort. 34 strains were isolated from 25 patients (32 were *C. indologenes* and 2 *C. gleum)*. Mean age of acquisition was 4,56 years old. The infection was not associated to deterioration in clinical outcomes (Table 1). All patients were co-infected with methicillin-sensitive *Staphylococcus aureus*. *Chryseobacterium* strains showed antimicrobial resistance with activity only for trimethoprim sulfamethoxazole and quinolones.
Conclusions: The reported prevalence may be significant considering the data in the published literature. Despite the fact that the clinical impact of