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Smoking prevention and cessation programme in Cystic Fibrosis: integrating an environmental health approach

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Abstract

Background—There have been several studies assessing the epidemiology and effects of tobacco smoke in the cystic fibrosis (CF) population, but few address the efforts of smoking cessation interventions. Our objective is to present one tobacco prevention and cessation programme targeting patients with CF in the Mediterranean region of Murcia (Spain).

Methods—All registered patients in the Regional CF unit (n=105) in 2008 were included in a cross-sectional and prospective uncontrolled study of tobacco use and exposure in CF patients using a baseline and one-year follow-up. Target population includes both patients and other family members living at home. The study included an initial telephone questionnaire, measurement of lung function, urinary cotinine levels, and several telephone counselling calls and/or personalized smoking cessation services.

Results—Of the 97 contacted patients, 59.8% (n=58) were exposed to environmental tobacco smoke (ETS), 12.4% (n=12) had smoked at one time, and 14.3% (n=8) of patients over the age of 15 actively smoked. The mean age was 31.13 (range: 19-45). Of the non-smokers (n=89), 56.2% reported ETS and 26.9% live with at least one smoker at home. 49.2% had urinary cotinine levels >10 ng/ml. The correlation found between patients' cotinine levels and their reported tobacco exposure was (0.77, p<0.0001). Active smoking by mothers during pregnancy was associated with significantly lower lung function in young CF patients (-0,385, p= 0.04). At the one-year follow-up, 13 individuals made attempts to stop smoking, 6 of which are now ex-smokers (12.5% of all smokers).

Conclusions—Smoking during pregnancy adversely affects lung function in individuals with CF. Tobacco he targeted tobacco prevention and cessation programmes are an effective and vital component for CF disease management. The trained professionals in prevention and smoking cessation services could provide patients with adequate follow-up, integrating an environmental health approach into CF patients' healthcare

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Keywords

Environmental tobacco smoke; cystic fibrosis; smoking prevention

INTRODUCTION

Tobacco use and passive exposure to cigarette smoke are the first and third leading causes of preventable deaths in the developed world, respectively.[1]

Cystic fibrosis (CF) is the most common inherited disease among Caucasians. A life-shortening autosomal recessive disease that affects the body's exocrine glands, its most prominent manifestations include compromised function of the lungs, intestines, pancreas and liver. However, lung inflammation and chronic respiratory infections alone account for nearly 95% of the morbidity and mortality in patients with CF.[2] Consequently, the negative health effects of tobacco exposure in young patients with CF are even greater than in healthy children.[3]

There have been several papers assessing the incidence, prevalence and negative effects of active and passive smoking in the CF population. However, there are few reports on smoking cessation interventions. [4] The objective of this paper is to present the tobacco prevention and cessation programme targeting patients with CF in the Mediterranean region of Murcia, Spain. We report the findings of a tobacco exposure questionnaire given to these patients, their correlations with urinary cotinine levels, the lung function and the result of the smoking cessation interventions one year after start.

METHODS

We conducted a cross-sectional and prospective uncontrolled study of tobacco use and exposure in CF patients using a baseline and one-year follow-up in the Mediterranean region of Murcia, Spain. The study included all registered patients in the Regional CF Unit of the University Hospital Virgen of the Arrixaca from 1998 to 2008. As a result of the centralized attention of patients with CF in Murcia, almost all patients diagnosed with CF in the region (>95%) are included in this registry. Of the 120 registered patients, 15 patients who had died were excluded. Only 6 patients were diagnosed through neonatal screening, which was started in Murcia in 2007; the rest were diagnosed by symptomatology. A letter from the CF unit was initially mailed to all 105 patients informing them of the programme's inception. The letter reinforced the importance of living tobacco-free in families of patients with CF and communicated that a trained professional would be in contact in the coming weeks.

Families were contacted by telephone to schedule interviews. Up to 9 attempts were made to establish telephone contact with the patients. The interviews were conducted between June-July 2008, and they lasted between 20 to 30 minutes. The administered questionnaire specifically asked about the patient's and family's tobacco use and exposure during different critical periods in the patient's life. Family members included anyone who lives with the patient (parents, siblings, spouse, or children of the patient). We have used the following classification of smoking [5]: 1. Non-smoker: a) not exposed to tobacco smoke, and b) passive smoker, exposure to smoke from his/her friends and/or parents and/or work and/or bars and/or others. 2. Occasional smoker: does not smoke daily. 3. Smoker: smokes at least 1 or more cigarettes/day. 4. Ex-smoker: does not smoke at the time of the study, and has not smoked for at least 6 months.

The questionnaire also inquired about the number of cigarettes smoked per week, age of smoking initiation and the pack/years smoked by all of the patients, parents and relatives. Moreover, we registered the patients' passive tobacco exposure during the pre-gestational period (the year before the mother's pregnancy and periconceptional period), the pregnancy, and the postnatal period (up until the diagnosis). The questionnaire also specifically asked about the many different sources of possible tobacco exposure in 2008: at home, work, in the car, places of leisure (no visits, 1 visit/week, 2-3 visits/week, >3 visits/week), restaurants/bars (no visits, 1 visit/week, 2-3 visits/week, >3 visits/week), and family visits (no visits, 1 visit/week, 2-3 visits/week, >3 visits/week). With all of this data, we created a variable called "Global passive exposure," which collects any type of passive exposure of special interest, especially in non-smoking patients. Other collected variables included the family's monthly net income and the education level of the patients and their parents.

A nurse from the Paediatric Environmental Health Specialty Unit (Pehsu) with training and knowledge in smoking cessation and prevention, telephone counselling and risk communication carried out the smoking cessation counselling via telephone to all smokers and non-smokers. This nurse took an interactive training course with an appropriate balance between theoretical (40 hours) and practical learning (100 hours).

The intervention included proactive telephone counselling that used active recruitment and targeted both patients and other family members living at home. The intervention had 4 principal messages: a) the short- and long-term effects of smoking in CF, with an emphasis on the health benefits of quitting; b) strategies for eliminating the patient's tobacco use and/or exposure; c) appropriate motivation based on which stage in the process of abandoning addictions the individual is at according to Prochaska and DiClemente (i.e. pre-contemplation, contemplation, preparation, action, and relapse); and, d) an offer of face-to-face tobacco cessation services and telephone support tobacco cessation resources. During the one-year follow-up, sequenced calls from this trained cessation counsellor were made every 6 months (no smoker at home) and every 4 months (any smoker at home). When smokers agreeing to make a quit attempt within two weeks are include in the cessation sessions. Up to seven telephone assistance or face-to-face sessions were available along 12 weeks. The process of effective tobacco-use cessation counseling can be broken into 5 steps, called the 5 A's: ask, advise, assess, assist, and arrange follow-up. The intervention included motivational interviewing, individual behavioural counselling and nicotine replacement therapies.[6] The smokers could "recycle" at least once if they failed to quit or to maintain cessation.

In the same way, the CF and Paediatric Pulmonology units also reinforce tobacco cessation during medical visits with brief messages and written leaflets. At the beginning of the program, the forced expiratory volume in one second (FEV1), the forced vital capacity (FVC) and FEV1/FVC were measured (all expressed as a percentage of the predicted value). Data obtained before 5 years of age or after lung transplantation were excluded. Cotinine urine levels were also solicited from patients with CF who came to the hospital for other purposes. Cotinine levels should be below 10 ng/ml in patients not exposed to tobacco smoke, and above that level, concentrations increase with increasing grade of exposure. Cotinine has been studied as a valuable variable in intervals (< 10 ng/ml; 10-50 ng/ml; 51-200 ng/ml; 201-400 ng/ml; >400 ng/ml). The patient's pseudomonas colonization (yes/no) was considered. To assess the effect of variation in the gene that causes CF, participants were divided into three groups: those with two copies of the most common mutation (F508del homozygotes), those with one and those with no copies of that mutation.

SPSS 15.0 was used to analyze data and form contingency tables of the frequencies required for this study. In that way, correlation studies with Spearman's rho were done on exposure

variables in the questionnaire and the urine cotinine levels of the patients and other family members in the home.

For patients with CF aged 5-18 years, a stepwise linear regresión análisis was performed in wich the outcome variables were FEV1, CVF and FEV1/CVF. To obtain predictor variables the comparisons of all variables were made using the unpaired Student's t test, ANOVA test and Spearman's rho correlation.

RESULTS

Of the 105 available patients, 97 (92.3%) were successfully contacted and followed. The participants demonstrated great interest in participating, and they received telephone recommendations for smoking prevention and cessation. Nine individuals (patients and parents) were seen in the clinic and utilized behavioural therapy; in one of them, nicotine substitution therapy was also used.

Table 1 shows the general characteristics of the contacted families at the moment of inclusion in the study and their actual tobacco consumption in 2008. Table 2 describes exposure to tobacco smoke during critical periods in the patient's life, ranging from the formation of the patient's germ cells (during the grandmothers' pregnancies) to the moment of the first telephone interview.

Active smoking reported in the questionnaire

Out of the 97 contacted patients, 58 patients (59.8%) were exposed to environmental tobacco smoke (ETS); 12.4% (n=12) had smoked at one time and 8.2% (n=8) were currently smoking; when considering patients over the age of 15, active smokers represented 14.3%. The mean age was 31.13 (with a range of 19 to 45). These patients smoked a mean 45.2 (95% CI 13.07-77.43) cigarettes per week. They started smoking at a mean age of 15.3 (95% CI 13.59-17.16), and had been smoking for 15.75 years (95% CI 7.42-24.08). The mean pack-years of smoking was 6.71 (95% CI 1.30-12.12). Of the patients that had smoked, only one did not have a first-degree smoker relative ($p<0.001$).

Passive smoking reported in the questionnaire

Of the 89 non-smoking patients, 56.2% (n=50) reported some type of ETS at home, school, work and/or places like bars and restaurants. 26.9% (n= 24) of them live with at least one smoker in the home. In those less than 15 years of age, 30.6% (n=11) accompany their parents to eat at a restaurant with tobacco smoke at least once a week. Table 3 shows the ETS exposure in and outside the home of non-smoking patients with CF.

Correlations with urinary cotinine levels

Cotinine levels were analyzed in 69 patients. 49.2% of patients had a cotinine level greater than 10ng/ml. The Spearman correlation is 0.77 ($p<.0001$) between cotinine levels and the tobacco smoke exposure of the patients reported in the questionnaire (3 categories: none, passive exposure, and smoker). Also, excluding patient smokers, the study showed that there was a difference in urinary tests for passive tobacco exposure if any parents or relatives smoked inside as opposed to outside the house (0.63, $p=0.003$). We found a negative correlation with family's monthly net income (0.41, $p<.001$) and no correlation with any educational level.

Cotinine levels were analyzed in 43 fathers and 52 mothers and significant differences were found with their distributions with smoking fathers and mothers, respectively ($p<.001$).

Smoking cessation

A total of 48 smokers were found (8 patients, 2 wives, 2 siblings, 17 fathers, and 19 mothers). At the 12-month follow-up, serious efforts were made to achieve smoking cessation in 13 people (4 patients, 1 father, 8 mothers), ultimately leaving 6 (12.5%) of them as non-smokers (2 patients, 1 father, and 3 mothers) during the last 6 months (cooximetry of 0 in patients). Two patients stopped smoking after going through a personalized consultation. Additionally, a reduction in smoking behaviour was reported by 10 people (2 patients, 8 fathers).

Lung funtion and gene-environment interactions

Table 4 shows the predictor variables that correlated significantly with each clinical end point. The number of copies of F508del was the most significant predictor of FEV1 and FVC. Mothers who smoked during pregnancy (cig/week) was the most significant predictor of FEV1/FVC.

DISCUSSION

Spain has one of the highest rates of smoking in Europe (29.5%), with an even higher rate in the Region of Murcia (33.9%). The prevalence is 40.7% among Murcian men and 26.9% among women.[7] The 14.3% (n=8) of the patients with CF in our study are active smokers, which is less than the rest of the Spanish population and intermediate among the 8%[8], 11%[9], 16%[10], and 21% that other published CF studies report. In general, it seems that CF was an important reason to not start smoking— an encouraging finding since the disease processes accompanying CF are likely to be exacerbated by tobacco use. Smoking is known to irritate mucosal linings and increase coughing and phlegm production in the respiratory tract, resulting in increased likelihood of bacterial infections, worsening of symptoms, and increased hospitalizations. Several studies have even found a dose-dependent relationship between the number of cigarettes smoked and the severity of respiratory disease among young patients with CF.[8, 11, 12]

Despite the absence of adolescent smokers in our study, about a third (36.5%) of CF patients under the age of 19 live with at least one smoker in the home, compared to 50% of healthy children in the Murcia Region.[13] Also, nearly 60% of our patients are exposed to tobacco smoke in places that should be more secure, such as at home or work (either because they themselves are smokers or because they are exposed to it by their relatives, colleagues, or others). These rates are alarming given that CF patients exposed to secondhand smoke in the home has been associated with a 10% reduction in pulmonary function.[14] Tobacco smoke demonstrates diminished nasal and sinus mucociliary clearance,[15] and it can also diminish the benefits of respiratory therapy in these patients, further limiting their cardio-respiratory capacity. Smoking may also result in appetite reduction and weight loss, which can be problematic in youngsters with CF who already have poor growth patterns and gastrointestinal complications.

The effect of prenatal tobacco exposure on infant pulmonary function is well known. We have shown a negative association between smoking during pregnancy and lung function in young patients with CF. We speculate that active maternal smoking during pregnancy negatively affects critical periods of lung development. For every additional 10 cigarettes smoked per week, the FEV1/FVC decreased by about 3-4%. A difference of our findings from previous studies is that the gene that causes CF does not amplify the negative effects of secondhand smoke exposure. [16]

This study has several limitations, most notably the small sample size and the difficulty of characterizing environmental tobacco exposure through a telephone interview. Recall bias is

a concern when information is collected retrospectively. The ubiquity, widespread exposure, and temporal dimension of tobacco smoke makes it difficult to interpret the concept of passive tobacco exposure and explains the large differences found in the literature, ranging from 23%[14] to 76%[8]. In order to verify the tobacco use and exposure reported in the questionnaire, we tried to reduce recall by conducting interviews by one trained professional. The calls were made by one nurse trained in tobacco cessation and motivational interviews, and the cotinine levels of patients and other family members were collected given that previous studies have found elevated urinary cotinine levels in CF patients who smoked compared to controls.[12] The correlation between grade of exposure among the patients via the questionnaire and their urinary cotinine levels was strong (0.77). We think that this is due to the experience and training applied when collecting the information. The cotinine levels are a valuable measure of exposure that could be collected annually since it is rapid, non-invasive and easy to interpret.

In order to further reduce recall bias, linear regression analysis with all these variables and other clinical endpoints of lung function were limited to patients younger than 18 years old.

Finally, the effect of other confounding variables could also be present such as nutritional status and others. This study is an ongoing project and we hope conduct further analyses in the future.

Telephone counselling (OR 1.56, CI: 1.38-1.77) and nursing interventions (OR 1.47, CI: 1.29-1.67) are effective approaches to smoking cessation,[17] and nurses trained in these methods have been shown to improve the results of interventions.[18] Pehsu can help to train and support nurses in this task.[19-21] In light of this, one nurse specially trained in smoking cessation and environmental health conducted the telephone interviews carried out in this study. According to participative response from participants, the frequency of phone calls seems to be adequate in our population.

The lack of a control group in this study prevents an estimation of the real impact of this approach. However, due to ethical considerations, we do not believe it would have been appropriate to deny available treatment to patients exposed to tobacco smoke. Despite this fact,, our minimal-cost interventions resulted in serious attempts to quit smoking by 13 people, 6 of which were ultimately successful, and smoking reductions in 10 others. While we present encouraging follow-up results for the first year of the programme, it is necessary to consider that smoking cessation interventions need to be maintained and prolonged in time. Relapses can be frequent, and relatives who smoke need continual smoking cessation advice and support from the paediatric multidisciplinary team in order to stop smoking and eliminate the damaging effects of tobacco on their child. CF units should also actively discourage smoking initiation in their younger patients, and if they do start smoking, a comprehensive cessation programme should be in place. Our interventional model complements the activities of CF units with the actions and support of a Pehsu. The European Environment and Health Action Plan (CEHAPE) recognizes that Pehsu should be created.[22] These centres can play a pivotal role in eliminating CF patients' exposure to tobacco—a life-threatening environmental toxin in this vulnerable population. They could help patients by integrating information and human resources that are trained and can provide adequate follow-up, smoking cessation services, help in finding the most appropriate cessation intervention, and a wide range of specific and personalized information on smoking and health-related needs in order to prevent smoking before it starts and help patients and relatives remain abstinent.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Socio-demographic variables

		Patients	Mothers	Fathers
CF Patients per family				
Only one	74			
Two siblings	7			
Three siblings	3			
Sex				
Male		55 (56.7%)		
Female		42 (43.3%)		
Age (mean)		18.66 (16.22-21.10)	46.87 (44.27-49.47)	48.53 (45.94-51.12)
Number of smoked cigarettes/week (mean)		45.25 (13.07-77.43)	89.66 (54.87-124.46)	138.15 (98.53-177.77)
Pack/years (mean)		6.71 (1.30-12.12)	10.28 (5.47-15.09)	29.88 (20.82-38.93)
Smoker (2008)				
Yes		8 (8.2%)	21 (21.6%)*	24 (26.1%)*
No		89 (91.8%)	76 (78.4%)	68 (73.9%)
Smokers in the home				
None	65 (67.0%)			
Only patient smokes	5 (5.2%)			
Patient and others smoke	3 (3.1%)			
Only others smoke	24 (24.7%)			
Cotinine Level (ng/ml)				
<10		35 (50.7%)	20 (38.5%)	10 (23.3%)
10 – 50		25 (36.2%)	16 (30.8%)	16 (37.2%)
51 – 200		2 (2.9%)	4 (7.7%)	7 (16.3%)
201 – 400		2 (2.9%)	1 (1.9%)	1 (2.3%)
> 400		5 (7.2%)	11 (21.2%)	9 (20.9%)
Level of Education				
None		32 (36.0%)	16 (19.0%)	16 (19.3%)
Primary		34 (38.2%)	39 (46.5%)	33 (39.7%)
Secondary		10 (11.2%)	19 (22.6%)	16 (19.3%)
University Studies Initiated		13 (14.6%)	10 (11.9%)	18 (21.7%)
family's monthly net income (€)				
<800 €	11 (15.1%)			
800-1500 €	24 (32.9 %)			
1500-2000 €	16 (21.9%)			
2000-2500 €	10 (13.7%)			

		Patients	Mothers	Fathers
2500-3500€	11 (15.1%)			
>3500 €	1 (1.4%)			

* Only 19 mothers and 17 fathers are smokers living with patients at home.

Table 2

Environmental tobacco smoke exposure during different Critical Periods in the Patient's Life

	Periconceptional Period	Pregnancy	Postnatal Period	Post-diagnosis	At the present time (2008)
Smoking Mothers	21 (21.9) 71.0 cig/week	15 (15.6) 38.3 cig/week	23 (24.0)	21 (21.9)	21 (21.6) 89.6 cig/week
Smoking Fathers	43 (45.7) 149.8 cig/week	43 (45.7) 147.8 cig/week At home: 38.6 cig/week	44 (46.8)	34 (35.4)	24 (26.1) 138.1 cig/week
Tobacco smoke exposure Patients	During Oogenesis 56 (58.3) During Spermatogene sis 51 (53.1)	73 (76.0)*	53 (55.8)**	45 (47.4) ***	Active smoker 8/97 = 8.2% 8/56 (>15 yrs) =14.3% 24 (24.7)*** 45.25 cig/week

* Refers to intrauterine tobacco exposure (a smoker at home, work, etc.).

** At least one smoker in the home after birth.

*** At least one smoker in the home (not including the patient).

Table 3

Environmental tobacco smoke exposure in non-smoking patients with CF (2008)

Sources of Tobacco Smoke	No smokers in the home (n=65)	At least one smoker in the home (n=24)
Restaurants	**19 (29.2)**	**6 (25.0)**
</= 1 time/week	0 (0.0)	0 (0.0)
2-3 times/week	13 (68.4)	4 (66.7)
>3 times/week	6 (31.6)	2 (33.3)
Pubs, Bars, Clubs	**8 (12.3)**	**3 (12.5)**
</= 1 time/week	7 (87.5)	2 (66.7)
2-3 times/week	1 (12.5)	1 (33.3)
>3 times/week	0 (0.0)	0 (0.0)
Other places with smoke (houses of friends, family, etc.)	**14 (21.5)**	**7 (29.1)**
</= 1 time/week	12 (85.7)	5 (71.4)
2-3 times/week	2 (14.3)	1 (14.3)
>3 times/week	0 (0.0)	1 (14.3)

Table 4

Stepwise linear regression of clinical end point with predictor variables in patients aged 5-18 years

Clinical end point*	Predictor variable	Regression coefficient	95% Confidence interval	p Value
FEV1	F508del homozygotes	-0,511	-1,016 to -0,048	0,034
	Pseudomonas +	-0,376	-0,741 to -0,010	0,044
FVC	F508 del homozygotes	-0,551	-1,026 to -0,072	0,027
	F508 del heterozygotes	-0,584	-0,943 to−0,220	0,003
FEV1/FVC	Smoking mother during Pregnancy (Cig/week)	-0,385	-0,416 to -0,007	0,043

* Expressed as percentage predicted. The following predictor variables were included: cotinine concentration, mother smoking during pregnancy (dicotoumus and cig/week), F508del groups, pseudomonas colonization, smoking father at home, father's packet per years. To obtain predictor variables the comparisons of all variables were made using the unpaired Student's t test, ANOVA test and Spearman's rho correlation.