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How to prevent the progression of chronic bronchitis: the role of smoking cessation prevention

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Abbreviations: FEV_1=forced expiratory volume in one second. COPD=chronic obstructive pulmonary disease. Ig=Immunoglobulin. IgM=M-type of immunoglobulin. IgG=G-type of immunoglobulin. IgA=A-type of immunoglobulin. IgE=E-type of immunoglobulin. C5=complement component. C9=complement component. C1=complement component

ABSTRACT

How to prevent the progression of chronic bronchitis: the role of smoking cessation prevention. M.C. Fiore

Cigarette smoking has been established as the chief avoidable cause of a number of pulmonary diseases, including emphysema, asthma, chronic bronchitis, and chronic obstructive pulmonary disease (COPD). In Italy, over 68,000 pulmonary deaths were attributed to tobacco use in 1987, compared to about 390,000 deaths in the United States. COPD accounts for the majority of these deaths in both Italy and the U.S., with 90 to 95% of all COPD deaths attributed to smoking. Quitting smoking results in dramatic health benefits, including a decrease in respiratory symptoms and acute respiratory infections, and a 5% improvement in pulmonary functioning. By using brief and effective smoking cessation interventions with patients who smoke, clinicians can play a central role in improving the pulmonary health of their patients. In addition, clinicians must take an active part in local, national, and international efforts to eliminate tobacco use.

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Introduction

Cigarette smoking, a behavior uncommon before this century, is now the most prevalence drug addiction world-wide, and the most important and preventable cause of chronic obstructive pulmonary disease.

Table 1. – Smoking related deaths in Italy 1987

Trachea, bronchi and lung cancer	23,000
Chronic bronchitis and emphysema	20,200
Cardiovascular pathologies	20,200
Larynx and mouth cancer	3,500
Esophagus cancer	1,200
TOTAL	**68,100**

In Italy, the health toll from tobacco is enormous. The mortality attributable to smoking in Italy in 1987, for example, was approximately 68,000 deaths including 23,000 with cancers of the respiratory tract, 19,200 with COPD, 20,200 with cardiovascular ailments, 3,500 with laryngeal and mouth cancers, and 1,200 with esophageal cancers (Table 1).

Moreover, cigarette smoking is the most prevalent drug addiction in Italy today. Recent data from 1986-87 showed that 40.8% of Italian men and 17.3% of Italian women smoked cigarettes (Table 2). The power of this addiction was underscored recently in Italy when employees of the Italian tobacco monopoly went on strike, leading to a country-wide outbreak of nicotine withdrawal. This strike received extensive world-wide press coverage as Italians, addicted to nicotine, struggled with their drug dependence. I think more than any recent event, the Italy tobacco workers' strike pointed out the power of nicotine addiction.

The prevalence of cigarette smoking in Italy has followed a pattern similar to the United States

Table 2. – Percent distribution (normalized by age) of smoking habits for Italian men and women of age 15 and older, for the three large geographic areas (ISTAT survey: Population Health Status 1986-1987)

	MALES			FEMALES		
	North Italy	Central Italy	South Italy	North Italy	Central Italy	South Italy
Non-smokers	44.4	43.9	43.7	76.0	76.6	84.6
Ex-smokers	16.9	16.5	12.5	4.4	3.7	2.0
Pipe/Cigar smokers	0.8	0.5	0.4	—	—	—
Cigarette smokers <15/day	15.6	14.5	14.9	12.8	12.5	9.2
15-24/day	17.3	18.7	22.4	6.2	6.2	3.6
>25/day	5.0	5.9	6.1	0.6	1.0	0.5

over the last 50 years. In fact, the rate of cigarette smoking in Italy today is virtually identical to that of the United States in 1964, the year of the release of the first U.S. Surgeon General's Report on the Health Consequences of Cigarette Smoking. At that time, approximately 42% of all adults smoked, very similar to recent Italy prevalence figures suggesting that approximately 35% of Italian adults currently smoke.

I believe the American experience in fighting tobacco addiction over the last 30 years may provide important lessons for our Italian colleagues as they attempt to decrease the enormous toll resulting from cigarette smoking in their country. In the U.S., progress has been slow with the most recent figures showing that overall smoking prevalence has fallen to approximately 25% of all adults. This slow rate of decline (about one-half of one percentage point per year) reflects, I believe, two factors — the addictive power of nicotine and the political and economic power of the U.S. tobacco industry. Any further success, both in the U.S. and in Italy must also address these two factors — treating nicotine addiction and directly fighting the tobacco industry, particularly against their efforts to urge children and young adults to start smoking.

In the U.S., 90% of all smokers become addicted before age 18; 50% become addicted before age 16. In fact, in the U.S., a country with 45 million smokers today, 1.3 million Americans stop smoking each year (Table 3).

Table 3. – United States, 1993

45 Million Current Smokers
Each Year
1.3 Million Smokers Quit
1 Million Children Start to Smoke

Unfortunately, they are replaced by 1 million young people who start to smoke each year. Therefore, any efforts to limit the damage from tobacco must address smoking cessation, but also the prevention of smoking initiation by children and young adults.

COPD and Cigarette Smoking

Although it is associated most often with COPD, cigarette smoking has been associated etiologically with the full spectrum of obstructive airway diseases including emphysema, asthma, chronic bronchitis, and COPD. Despite the fact that they possess distinct clinical features, overlap among these conditions is extremely common, and most patients demonstrate clinical evidence of more than one of them.

The development of clinically relevant COPD in smokers has been hypothesized to result from an accelerated rate of decline in lung function among that subsegment of smokers who are predisposed to develop COPD. Speizer and Tager presented a schematic representation of the changes in lung function among never-smokers and smokers who will and will not develop COPD (fig. 1). In this representation, smokers overall (B) demonstrate an accelerated, although not clinically apparent, decline in lung function when compared with nonsmokers (A). Among a subsegment of smokers (C), the decline in lung function is accelerated further, though not yet fulfilling clinical criteria for COPD. A smaller seg-

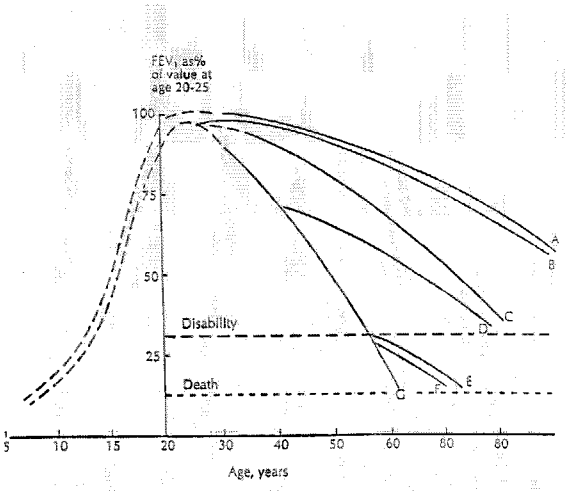

Fig. 1. – Theoretical curves depicting varying rates of decline of FEV_1. Curves A and B represent never-smokers and smokers, respectively, declining at normal rates. Curve C shows increased decline without development of COPD. Rates of decline for former smokers are represented by curves D and E for those without and with clinical COPD, respectively. Curves F and G show rates of decline with continued smoking after the development of COPD (from Speizer F.E., Tager I.B.).

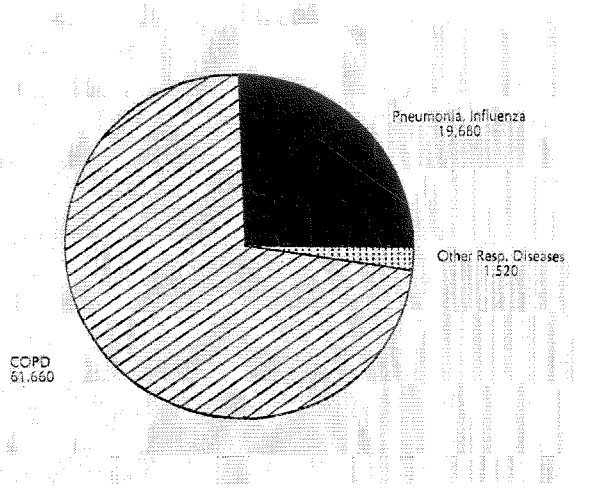

Fig. 2. – Pulmonary deaths each year attributable to smoking, United States

ment of smokers (E), will develop clinically apparent COPD resulting in disability. The final group of smokers (G) will develop COPD that rapidly and prematurely results in death. Genetic predisposition and childhood influences such as exposure to environmental tobacco smoke, other toxins, or both may influence and individual's predisposition to develop COPD. Independent of these predisposing factors, cigarette smoking in the United States has been established as a cause of 90 to 95% of all COPD resulting in 62,000 deaths (fig. 2). Additionally, 20,000 other, non-carcinomatous deaths in the U.S. are directly attributable to cigarette smoking.

In addition to these changes in lung function, cigarette smoking resulting in a myriad of pathophysiologic changes promotes COPD and other diseases (Table 4).

Table 4. – Summary of Pathophysiologic Changes Resulting from Cigarette Smoking

I. **Inflammatory changes**
   a) Increase in inflammatory cells (neutrophils and alveolar macrophages)
   b) Elastase release in the lung
   c) Decreased α1-antiprotease activity

II. **Altered immune function**
   a) Depressed antibody production
   b) Altered cellular immune response
   c) Decreased mitogen responsiveness
   d) Decreased alveolar macrophage responsiveness to lymphokine and macrophage migration inhibitory factor
   e) Depressed phagocytosis and intracellular killing
   f) Higher peripheral leukocyte count
   g) Higher levels of C5, C9, C1-inhibitor, C-reactive proteins, and autoantibodies (antinuclear and rheumatoid factors)
   h) Lower levels of IgG, IgM, IgA
   i) Higher levels of IgE
   j) Blunted immune response to influenza vaccination

III. **Heightened airway responsiveness**

These include: 1) Inflammatory changes including increase in inflammatory cells, elastase release in the lungs and decline in alpha-1 antiprotease activity, and; 2) Altered immune function including depressed antibody production, altered cellular immune response, decreased mitogen responsiveness, decreased alveolar macrophage responsiveness to lymphokine and macrophage migration inhibitory factory, depressed phagocytosis and intracellular killing, higher peripheral leukocyte count, higher levels of C5, C9, C1-inhibitor, C-reactive proteins and autoantibodies, lower levels of IgG, IgM, IgA, higher levels of IgE, and blunted immune response to influenza vaccination; and heightened airway responsiveness.

The Pulmonary Benefits of Smoking Cessation

Quitting smoking causes pulmonary benefits, both symptomatically and pathophysiologically. Buist and coworkers reported that smoking cessation was associated with a dramatic reduction in respiratory symptoms (e.g., cough, expectoration, shortness of breath and wheezing) within one month of cessation. These finding have been substantiated in other clinical, cross-sectional, and longitudinal studies that assessed the impact of smoking cessation on respiratory symptoms, although sex- and age-specific differences have been noted. The results of these studies contributed to the 1990 United States Surgeon General's Report on the Benefits of Smoking Cessation. That report (Table 5) noted that quitting smoking results in a rapid and significant decrease in respiratory symptoms, irrespective of the quantity smoked or the length of time smoking.

Table 5.

In the United States, more than 38 million Americans have quit smoking; almost half of all smokers. The United States Surgeon General's Survey is devoted to "Health Benefits of Smoking Cessation." Its main conclusions can be summarized in five points:

1. Smoking cessation has major and immediate health benefits for men and women of all ages. Benefits apply to persons with and without smoking related diseases.
2. Former smokers live longer than continuing smokers. For example, persons who quit smoking before the age of 50 have only half the chance of dying in the next 15 years, compared with continuing smokers.
3. Smoking cessation decreases the risk of lung cancer and other cancers, heart attack, stroke, and chronic pulmonary diseases.
4. Women who stop smoking before pregnancy or during the first three to four months of pregnancy reduce their risk of having a low birth-weight baby.
5. The health benefits of smoking cessation far exceed any risk from the average 5-pound (2-3 kg.) weight gain or any adverse psychological effects that may follow quitting.

In addition to its impact on respiratory symptoms, smoking cessation also results in a decrease in the rate of acute respiratory infections compared to persons who continue to smoke. In the first American Cancer Prevention Study (ACS-CPS-I), a study involving more than 1 million Americans from 1959 to 1963, mortality from influenza and pneumonia was 1.3 and 1.9 times more likely among ever-smokers when compared to never-smokers. In the second American Cancer Society Cancer Prevention Study (ACS-CPS-II), mortality from influenza and pneumonia decreased among former smokers when compared with continuous smokers. Improvements in immune function is a chief reason for the decrease in respiratory infections following smoking cessation.

While modest, smoking cessation also results in an improvement in pulmonary function as measured by pulmonary function tests. The U.S. Surgeon General, in her 1990 report concluded that, "For persons without chronic obstructive pulmonary disease, smoking cessation improves pulmonary function about 5% within a few months after cessation. Moreover, with abstinence from smoking in persons with and without COPD, the rate of decline in pulmonary function among former smokers returns to that of never-smokers. Finally, with sustained abstinence, the COPD mortality rates among former smokers decline in comparison with continuing smokers." Unlike the risk of death from an acute myocardial infarction, however, the risk of death from respiratory causes among former smokers continues to exceed that among never-smokers. However, it decreases significantly compared with individuals who continue to smoke.

Clinical Intervention with Patients Who Smoke

Cigarette smoking in both Italy and the United States is the single most important preventable cause of pulmonary diseases, especially COPD. For that reason, clinicians must play a central role in eradicating this cause of morbidity and mortality. The U.S. National Cancer Institute urges physicians to follow a four step program (Table 6). Ask about smoking with every patient on every clinic visit regardless of the problem that brought them to see you.

Arrange follow-up: The patient should be evaluated within one-week of quitting to assess success and to aid the patient with any problems.

At the University of Wisconsin, we have developed criteria for deciding who should use the nicotine patch. The only absolute criteria is that the patient must be motivated to quit. If motivated, and the patient answers yes to any of the following three questions, we use the nicotine patch: 1) Twenty or more cigarettes per day; 2) First cigarette within 30 minutes of awakening; 3) Previous quit attempts-strong physical withdrawal during first week.

In addition to treating the smoking patient, clinicians must also be at the forefront of local, national and international efforts to eliminate tobacco use (Table 7).

Controlling access to children, higher taxes on tobacco products, bans on tobacco advertising, and eliminating exposure to environmental tobacco smoke are the cornerstones of effective tobacco control activities.

We have clearly identified the cause of most COPD in our society. Most of us would never refuse to treat a sick patient with this debilitating disease. We need to have a similar commitment to treating the underlying cause of this problem-eliminating the use of tobacco in our own communities and worldwide.

Table 6. - Guidelines for Physicians in Promoting Smoking Cessation Among Clinic Patients

Ask every patient about smoking during every visit
- Use a smoking status vital sign

Advise all smokers to quit
- Advice must be clear, firm, unequivocal
- Personalize the stop-smoking message: as a parent, as a professional person, as a pregnant woman, as a person who lost a father to lung cancer, as a person with lung, heart, etc. disease. You must stop smoking now.

Assist the patient in stopping
- Set a quit date within the next month
- Sign a stop-smoking contract
- Provide self-help materials
- Assess the degree of nicotine addiction, then consider adding nicotine replacement therapy (patch or gum): signs of nicotine addiction include those who smoke more than one pack of cigarettes per day and smoke their first cigarette within 1 half-hour of awakening.
- For those not willing to try to quit, ask on next visit

Arrange follow-up visits
- Set a follow-up visit within 2 weeks of the quit date
- Reinforce the quitting commitment with a telephone call
- Second follow-up visit within 2 months of the quit date

Advise: Advise ALL patients, in a clear way that they must stop smoking now and provide them with specific advise on how to quit.
- Assist: Assist them in quitting:
  Set a quit date
  Provide self-help materials
  Use the nicotine patch when appropriate (see below)

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