Chronic Obstructive Pulmonary Disease

EXECUTIVE SUMMARY

Chronic obstructive pulmonary disease (COPD) is defined as a process characterized by the presence of chronic bronchitis or emphysema that may lead to the development of airways obstruction. COPD may range from mild to severe. Health care providers must be involved in the primary prevention of COPD through campaigns to eliminate cigarette smoking and other risk factors and secondary prevention through early detection of airflow obstruction in asymptomatic individuals at risk. They must also implement effective care for those who become symptomatic, and develop rehabilitation programs to slow disease progression among those with more serious airway obstruction.

COPD affects approximately 11% of the US adult population; further, its incidence is increasing, with greater rates of change for women than for men. Cigarette smoking is the most important and best documented risk factor for COPD; air pollution exacerbates the disease and may be a risk factor for its development. There is substantial evidence that childhood infections are also a risk factor for COPD. Further, COPD is aggregated in families, and there is good evidence that those with a severe hereditary deficiency in alpha-antitrypsin are more susceptible to COPD. Other risk factors include male sex, advanced age, airway hyperactivity, occupational exposure to dusts (particularly for cigarette smokers), and perhaps environmental exposures such as oxidant gases associated with low socioeconomic status. Secondary and tertiary risk factors include exposure to outdoor pollutants and intercurrent infection.

The clinical care of COPD includes avoidance of polluted air, regular influenza vaccinations, and vigorous programs for smoking cessation; augmentation therapy should be considered for those with severe alpha-antitrypsin deficiency. Symptomatic measures may include the use of bronchodilators, sympathomimetics, anticholinergics, theophylline, and selective use of corticosteroids and antibiotics. Supportive therapy involves nutrition programs to combat progressive weight loss, and psychological treatment for depression and anxiety. It includes comprehensive rehabilitation, home ventilator care, and long-term oxygen therapy for hypoxic patients. Lung transplantation is also now an option for advanced COPD. Providers should be actively involved in risk factor modification.

Education is essential for the development of new treatment programs for COPD patients. Some of the major targets for improved educational programs include the development of better smoking-cessation methods, dissemination of information on the benefits of early treatment, more widespread efforts to increase the use of influenza vaccinations, programs to enhance patient knowledge and self-care skills, and programs to disseminate information to health care providers. Patient education goals should be directed toward the development of better self-management programs for those with minimal illness, as well as those with more severe disability. Provider education programs should increase the following: (a) the number of physicians who know the risk factors for COPD and how to modify them; (b) the number of physicians who screen for airflow obstruction; and (c) the use of risk factor modification for those with secondary complications. These programs will require both public education campaigns and targeted continuing education programs. Further, these goals might be better achieved if prevention became consistently established in the medical school curriculum.

Health services research goals include the development of better outcome measures, expanded cost/efficacy studies, and research on the relationship between dysfunction and psychological depression. In addition, it will be important to focus more attention on the high prevalence and the rising incidence of COPD among minority groups and women. Determinants of provider behavior including documentation of appropriate and inappropriate test use will also be important. Finally, more research is necessary to delineate the determinants of patient behaviors, such as cigarette smoking and noncompliance with medical regimens. Experimental evaluations of interventions to remedy these problems should also be undertaken.

Research Recommendations

Epidemiologic Research

- Define interaction of nonmodifiable and environmental risk factors.
- Define host factors in determining impact of cigarette smoke on the lung.
- Define role of maternal smoking (in utero) on later development of COPD.
- Better define role of airway hyperresponsiveness on development of COPD.
- Define role of childhood exposure to ozone and acid aerosols in the development of COPD.
- Better define risk associated with socioeconomic status and race.

Biomedical Research

- Develop improved chemical markers of lung injury.
- Further develop the component parts of the protease-antiprotease systems in relation to lung injury.
Epidemiology: The magnitude of COPD is substantial and increasing. In the United States, the age-adjusted prevalence for men, as estimated from the National Health Interview Survey, was 11.0% in 1985 as compared to 9.4% in 1979. The comparable statistic for women was 11.9% in 1985 as compared to 8.8% in 1979. The increasing prevalence as well as differences between sexes can be largely accounted for by changing patterns of cigarette smoking, taking into account the substantial lag between onset of smoking and the manifestation of disease. Because there is a long asymptomatic period of disease, COPD may be underdiagnosed.

Similar trends exist for mortality from COPD. Data from the National Vital Statistics System indicate an age-adjusted death rate of 0.20% for men in 1985 as compared to 0.17% in 1979. The death rate due to COPD in women rose from 0.05% in 1979 to 0.08% in 1985. From age 40 and beyond, death rates increase rapidly with age. In 1985, COPD was the underlying cause in 3.6% of all deaths and a contributory cause in an additional 4.3%. Viewed from a broad perspective, these data are quite dramatic. In a 7-year period, death rates from COPD have increased approximately 16% in men and 73% in women, despite a decline in death rates from all causes, notably cardiovascular disease, and a plateauing in death rate from cancer other than that of the respiratory system.

The impact of COPD on society can be estimated from data on use of medical resources. Men increased their visits to doctors' offices for COPD by approximately 15% from 1979 to 1985; women by approximately 8%. Feinleib and co-workers report a dramatic difference in self-assessment of health status: patients with COPD are twice as likely to rate their health as fair or poor, and to have experienced recent limitation in activities in comparison to the general population; many report more frequent visits to physicians (46% as compared to 29% of the general population). They also report approximately twice the number of short hospital stays, days of restricted activity, and days of being confined to bed as do subjects from the general population.

There is general agreement that most of the morbidity and mortality due to COPD can be attributed to cigarette smoking. The FEV₁, commonly used measure of lung function, is predictive of future morbidity and mortality. When the level of FEV₁ reaches 35% to 40% of predicted normal, most individuals are limited for work and to some extent, for activities of daily living. As function deteriorates further, symptoms worsen and death often occurs from obstructive lung disease when the level of FEV₁ decreases below 25% of predicted level. Smokers have a more rapid decline in pulmonary function than do nonsmokers. When smokers stop smoking, they do not fully recover the function they have lost but may reduce their rate of loss to that of a nonsmoker. Thus, level of pulmonary function is an important predictor of morbidity and mortality.

Since not all smokers have similar rates of decline of pulmonary function for a given amount of smoking, one must consider additional risk factors that may put people on these different tracks. It is known that children tend to follow a set trend in the growth of their lung function. One hypothesis is that children who do not reach their predicted level of full lung function in early adult life are at greater risk of the effects of cigarette smoke. If this hypothesis is true, it becomes important to determine why some children exhibit these slower rates of growth and when this effect begins. Recent investigations have focused on the beginning stages of lung growth and have involved evaluation of both in utero as well as postnatal exposures to a variety of risk factors.

Other selected risk factors for obstructive airways diseases include recurrent respiratory infections in childhood, specific occupational exposures resulting in asthma-like syndromes (eg, from toluene diisocyanate, wood dusts, tea and coffee dusts, animal protein exposures, and solder fluxes), and chronic airways disease (eg, from welding fumes, cement dusts, and certain other occupational dusts). In addition, the severe form of hereditary alpha,-antitrypsin deficiency associated with the PiZZ genotype is associated, especially in smokers, with severe, early onset emphysema. The gene frequency of this phenotype is about 1/2,700 and is too rare to be responsible for much of the population burden of emphysema; however, it does serve as a paradigm of a genetic obstructive lung disease.

Primary Risk Factors for COPD

Considerable progress has been made in recent years toward the goal of distinguishing between genetic and environmental risk factors for COPD. Although the hereditary data are far from complete, one conclusion is inescapable: in the absence of cigarette smoking, severe deficiency of alpha,-antitrypsin, and bronchiectasis, COPD of sufficient severity to cause appreciable impairment of lung function is quite uncommon, at least in Western countries. This may not be true, however, in developing countries where COPD is seen with some frequency in nonsmokers as well as in smokers.

Smoking: Active smoking is known with certainty to be related causally to COPD. Not well understood yet is the importance of the composition of the smoke, the kind of cigarette, the way in which the individual puffs, and whether there is a clear dose-response relationship between amount smoked and the development of disease. The picture is less clear for passive smoking and maternal smoking. With the present state of knowledge, it is reasonable to conclude that children whose parents (especially mothers) smoke are likely to have increased rates of respiratory infections and respiratory symptoms and may have slightly reduced pulmonary function relative to their peers who have nonsmoking parents. It is not known whether these changes in pulmonary function result directly from environmental exposures in childhood or from in utero exposure from mothers who smoke while pregnant. Household exposure relates directly to the number of smokers in the household. How these exposures and effects translate into increased risk of COPD in adult life is not yet known.

Air Pollution: Air pollution is a complex mixture of gases, particles, and water vapor that results from the burning of fossil fuels or other combustible materials (biomass) and the interaction with climate and other factors in the environment. Over the past 40 years, several major population-based studies incorporating monitoring of ambient air pollutants, together with assessment of respiratory morbidity, have identified adverse health outcomes ranging from excess.
in some patients with COPD. Pollutants most likely to affect COPD patients are ozone, acid aerosols, and particulates. The principal indoor pollutant is cigarette smoke. Patients with hyperresponsive airways are sensitive to a variety of nonspecific irritants, and in some cases, to specific allergens.

Intercurrent infection plays a clear role in the exacerbation of COPD. The most important agents are the common respiratory viruses, the influenza virus, and bacterial agents such as Streptococcus pneumoniae, Hemophilus influenzae, and Legionella pneumophila.

Tertiary factors are those elements of disease which can be modified by therapeutic measures. The major factor in COPD is reversible airflow obstruction, which is due to increased bronchomotor tone and bronchial hyperreactivity. Inflammation of airways and narrowing of airways due to mucus is also commonly present. In more severely impaired patients, hypoxemia, loss of mass of skeletal muscles, (including those used for breathing), and respiratory failure frequently supervene. Important psychosocial problems occur in this group as well.

**Clinical Care of COPD**

The prevention of COPD and its sequelae require attention to tertiary, secondary, and primary risk factors.

**Principles of Control of COPD**

The clinical management of patients with established COPD and tertiary preventative techniques have recently been reviewed.37 Except when patients are suffering from acute respiratory failure or some other acute medical incident, the management of patients with COPD can be carried out in the ambulatory medical setting. However, given the importance of acute events as causes of death, it is essential that these patients have ready access to the hospital when they become acutely ill.

**Specific Measures: Smoking Cessation.** Although there is some debate (see Smoking Committee Report), the rate of decline of lung function, measured as decrease in FEV1 per year, is greater in smokers than nonsmokers, especially in smokers with COPD.38 Every effort should be made to help persons with COPD stop smoking at all stages and levels of disease. There may be a small improvement in lung function following smoking cessation, especially if function loss is mild. Of greater importance is that the rate of decline of FEV1 reverts in most exsmokers to that of nonsmokers, bronchiolar inflammation, luminal obstruction in subjects with COPD is fixed and irreversible.

**Symptomatic Measures:** Symptomatic therapy is directed against the relief of reversible airflow obstruction due to bronchiolar inflammation, luminal secretions, and smooth muscle spasm. Symptomatic therapy will be addressed under the subheadings of bronchodilator therapy, corticosteroids, antibiotics, and mobilization of airways secretions.

**Bronchodilator Drugs.** Although most of the airways obstruction in subjects with COPD is fixed and irreversible, there is a high prevalence of partial acute reversibility to inhaled isoproterenol. The amount of reversibility of FEV1 averages 15% of the baseline value, with about one third of patients showing responses of 62% or greater.48 COPD patients with the greatest bronchodilator responses have the lowest annual decline in FEV1, and the greatest 5-year survival. The hypothesis that regular bronchodilator therapy may slow deterioration of lung function is currently under investigation in a large clinical trial, the National Heart, Lung, and Blood Institute Lung Health Study.49

**Sympathomimetetics.** The beta-adrenergic agonists, given as
failure and on mechanical ventilators. Several controlled studies demonstrated improvement in such patients following nutritional repletion. However, long-term maintenance of weight gain has been difficult to establish.

**Psychosocial Aspects of Severe COPD.** Like all serious chronic illnesses that are slow and insidious in onset, COPD has a profound effect on patients, their families, and other care givers. The constant threat of dyspnea and the reality of increased dependency can lead to loss of self-esteem, somatic preoccupation, anxiety, irritability, and depression. Depression may magnify the distress from physical symptoms in COPD, including a worsening of dyspnea. Management requires a willingness by the health care provider to deal with the psychosocial, as well as the somatic elements of the illness. Encouragement of the patient, education in self-care, the setting of realistic achievable goals, counselling of family, members, and group interaction of patient and family members with groups of similar patients and their families are all useful. Participation in comprehensive pulmonary rehabilitation programs may be particularly effective in providing these elements in a cohesive manner. Pharmacologic treatment may be used to supplement these measures. However, psychoactive drugs frequently have side effects, such as sedation, which require special caution when used in the patient with COPD.

**Lung Transplantation.** With the advent of cyclosporin as an immunosuppressive agent, lung transplantation has become a potential treatment alternative for selected patients with end-stage COPD. Patients have generally been under age 50 years, and many have had severe alpha-antitrypsin deficiency as the cause of their emphysema. Although there have been some complications with double lung transplantation, there has been an increase in the use of single lung transplantation for patients with nonseptic obstructive disease. Continuing follow-up studies will be required in order to finally evaluate the benefits of single lung, double lung, and heart-lung transplantation; at present, the procedures appear to be promising for selected patients.

**Issues in Education**

The previous section provided a brief overview of the clinical care of established COPD patients. Since there is no cure for COPD, reducing the impact of these diseases requires preventive efforts. The remainder of the report will focus on primary and secondary preventive strategies. Improved preventive efforts require education directed toward patients, the general public, and health care providers.

**Barriers to Effective Prevention**

Success in the control of COPD begins with prevention. If this step fails, the next goal is early intervention, when airflow obstruction is still preventable or reversible. Although prevention and early intervention are desirable, they have proven difficult to achieve. Barriers to achieving these goals include the following:

**Inability to Achieve Smoking Cessation:** Although many established smokers have stopped smoking, over 50 million Americans (20% of the adult population) continue to smoke. As many as 50% of smokers try to quit in any year and most attempts are unsuccessful.

**Limited Awareness of the Benefits of Early Treatment and Risk Factor Modification:** The earlier COPD is identified and treated, the more likely it is that functional impairment will be avoided. However, most COPD patients do not seek treatment until they become dyspneic. At this time, changes resulting in airflow obstruction are typically widespread and not reversible.

**Limited Use of Immunization for Influenza:** It has been estimated that as few as 20% of high-risk patients receive immunization against influenza. Retrospective studies have shown that immunization will prevent illness in 20% to 45% of those vaccinated. Additional studies have shown that those not completely protected tend to experience milder symptoms and are less likely to require hospitalization. Mortality is also decreased. It has been estimated that as many as 10,000 patients die from influenza in a nonpandemic year, and as many as 30,000 after a major epidemic. If the number of "at risk" persons immunized increased to 80%, these deaths, hospitalizations, and related costs to the health care system should substantially decrease.

**Lack of Patient Knowledge, Skills, or Motivation to Follow Management Regimen:** COPD patients may lack the skills required to carry out their management regimen or to perform activities in an energy-efficient manner. The sensation of shortness of breath may lead to severe anxiety, avoiding activities that provoke dyspnea, and preoccupation with bodily complaints. These actions further compound the problems experienced.

**Lack of Knowledge by Health Care Providers:** Many COPD patients are cared for by health care professionals who are not specialists in pulmonary medicine. The state of the art in management of COPD has changed appreciably in the past decade. Application of current knowledge would be facilitated if physicians, nurses, and other health care professionals had easy access to educational materials which succinctly summarize these advances and present easy to follow strategies for improving the functional ability of COPD patients.

Educational strategies to resolve problems created by these barriers include initiatives directed toward education of the public, patient and family, and health care providers.

**Public Education**

Beyond patient education, general public education is also required. Strategies should include the following:

1. A clear, consistent, and repeated nonsmoking message should be delivered. A meta analysis of 39 controlled smoking cessation trials indicated that it was social reinforcement and support—by increasing the number of contacts, the types of contacts, and the number of people making the contacts—and not a specific intervention or delivery system that produced results. Likewise, it appeared that it was withdrawal of reinforcement that contributed to relapse. The conclusion drawn from this is that smoking cessation messages would be most productive if given clearly, repeatedly, and consistently through every feasible delivery system (eg, personalized advice, print materials, the mass media, and a smoke-free home, school, and work environment).

2. The development of smoking prevention and cessation programs should target adolescents and young adults with
Similarly, the role of atopy and airways responsiveness and the development of subsequent COPD need further clarification. Subjects with a history of atopy are at greater risk of having airways responsiveness; however, whether the causal pathway goes on to the development of COPD is not known.

Increased airways resistance and responsiveness have been found in subjects exposed to ozone and acid aerosols (H₂SO₄, HNO₃) in recent controlled laboratory exposures. Animal studies suggest these agents produce inflammatory changes in small airways. Concern has been raised about the role of these agents in producing respiratory effects in chronically exposed children, and if so, whether these children will become more susceptible to COPD in later life. These issues require further study.

Several recent studies suggest that occupational dust exposures result in COPD in some fraction of the exposed subjects. Occupational asthma has been characterized as a disease in which the specific agent, whether it be a sensitizer or irritant, can be identified. Unfortunately, in this setting, elimination from exposure only reverses the disease for a minority of patients. Thus, the onset of occupational asthma offers an opportunity to study the immunologic state of the host (potentially) before exposure begins and during the course of developing the disease. Obviously, tied to any such study must be industrial hygiene control methods to reduce exposure to a minimum. However, once sensitized, it may not be possible to reduce exposure sufficiently to protect the sensitized worker.

Although differences in socioeconomic status (SES) do not appear to be an important risk factor in the developed world at this time, experience suggests that in the past, SES was a surrogate for multiple environmental factors such as nutrition, smoke exposure, and crowding; these are potential risk factors that need to be explored in the developing world today.

Biomedical Research

Chemical Markers of Lung Injury: Over the last 23 years, scientists have developed evidence that neutrophil elastase causes at least some of the emphysema in smokers or in alpha,-antitrypsin deficient patients. Several companies have developed elastase inhibitors; however, current concepts of how a clinical trial of efficacy would have to be carried out dictate that these clinical trials would be enormously expensive. It is possible that chemical markers of lung injury may reduce the expense of a clinical trial enough to make it financially feasible. Two critical areas deserve serious attention: existing putative chemical indicators must be validated or discarded, and new indicators should be developed.

Expanding the Protease-Inhibitor Deficiency Hypothesis: Over the last 25 years, investigators studied various aspects of the protease-inhibitor deficiency hypothesis of emphysema. This theory contends that neutrophil elastase causes emphysema if it is not inhibited by alpha,-antitrypsin. The dominance of this theory may have too-narrowly-focused research in this area. It is important to stimulate new ideas about the pathogenesis of emphysema. Several areas may be worth further research. Several groups have determined that alveolar macrophages provide the enzymes which digest lung elastin and cause emphysema. Other groups have shown that proteoglycans are reduced in lungs with emphysema. Expanding current research to include other specific areas of lung destruction and tissue destructive mechanisms, as well as mechanisms of repair, may be fruitful.

Exploring Genetic Clues to Emphysema: Not all smokers develop emphysema. In addition, there are family clusters of patients with emphysema that are not due to alpha,-antitrypsin deficiency. New genetic, cell biological, and chemical methods have been developed to determine genetic loci in cystic fibrosis and phenylketonuria. Efforts should be made to identify kindreds with emphysema and to encourage them to participate in research designed to determine why they have a familial propensity to develop emphysema.

Therapeutic Research

There is a wide variety of issues in therapy that require further clarification and development. These issues include the following: mechanisms of pulmonary vasoconstriction and control of pulmonary vasoconstriction; the role of acute and chronic airways inflammation in COPD and the efficacy of anti-inflammatory agents in its treatment; the role of oxygen therapy during sleep; the role of bronchodilators; the mechanism of apparent airways hyperresponsiveness and its control; the role of infection and optimal methods for prevention of infection; the role of locomotor and respiratory muscle conditioning in improving function; development of improved methods for ventilatory support; and improvement of methods for single lung transplantation.

Research Related to Health Care Delivery

Health Services Research: Health services research focuses on problems related to the quality, delivery, and costs of health services. Some important efforts in health services research involve the definition and measurement of health outcomes. These outcomes are described as health-related quality of life (HRQOL). Evaluating health outcomes typically involves 2 components. One component is mortality which is estimated through standard life table techniques. The other aspect is quality of life and is typically assessed using questionnaires. Traditional medical measures, such as pulmonary function tests, are often regarded as mediators of these outcomes.

There has been relatively little work evaluating HRQOL in COPD patients. Some of the approaches have used measures specific to lung disease, such as the dyspnea index and the chronic respiratory disease questionnaire. The nocturnal oxygen therapy trial (NOTT) used a general quality of life measure and demonstrated systematic relationships between degree of disease severity and quality of life outcomes. This measure was also used in a clinical trial evaluating the cost effectiveness of home care for COPD patients. A related approach known as the quality of well-being scale has also been used in several trials with COPD patients. This measure provides a generalized assessment of health status that can be used in cost effectiveness evaluations. There has been little research that attempts to link measures of pulmonary function or disease severity to measures of health status or patient outcome.

There appears to be a major trend toward the use of health outcome measures in both clinical and policy re-
Determined of Patient Behavior

Smoking in COPD: More than 25 years ago, the Surgeon General released the first report on smoking and health. Over the last quarter century, the case against the use of cigarettes has become increasingly strong. In 1989, the Surgeon General released a new report entitled, “Reducing the Consequences of Smoking: 25 Years of Progress.” As the new report suggests, the evidence on the detrimental effects of cigarette use is truly overwhelming. Nevertheless, the prevalence of smoking was still 29% in 1987, and techniques to reduce tobacco use in this group remain marginally effective and largely unsatisfactory. The need for research in this area remains urgent and is discussed elsewhere in this report.

Compliance Behavior: Despite major advances in medical diagnosis and the use of medical therapeutics, patients frequently do not receive the optimal benefit from contemporary medical care because they fail to follow treatment recommendations. Nearly all encounters between physicians and patients end with advice. The patients are told to stop smoking, use their medications in a specific way, exercise, and so on. Noncompliance or nonadherence is failure to follow this advice. Published studies suggest that rates of noncompliance range between 15% and 93%, with rates toward the higher end for those with diagnosed chronic diseases. Attempts to predict noncompliant behavior on the basis of psychological or demographic characteristics have been largely unsuccessful. In addition, there have been curious relationships between compliance and outcome. For example, Epstein reviewed the literature and discovered that compliance is associated with better health outcomes whether or not patients are receiving active drugs or placebo.

There have been relatively few studies on compliance behavior among patients with COPD. In one recent review, only a handful of studies was identified. Among the published studies, some findings specific to COPD patients emerged. For example, several studies identified overcompliance with medications such as corticosteroids that provide symptomatic relief. To date, commentaries on compliance with lung disease treatment exceed empirical studies. Some recent attention has been devoted to the advantages of transtracheal oxygen therapy. One study, for example, demonstrated that transtracheal oxygen can increase the number of patients who use therapy 24 h per day. Using the results of the NOTT trial, this increased compliance might be translated into estimates of greater life expectancy. Other studies have demonstrated that simple counseling interventions by pharmacists might increase compliance with medications commonly used by COPD patients.

There appears to have been relatively little research investigating the potential of increasing patient compliance among those with COPD. Physicians have little training in identifying the determinants of noncompliance and in assessing the magnitude of the problem. More research is necessary in order to identify counseling methods for enhancing compliance among COPD patients.

Conclusions

Chronic obstructive pulmonary disease is a major cause of death and disability in the United States. The most important risk factor for COPD is cigarette smoking. Thus, COPD is largely a preventable condition. Other risk factors may include air pollution, childhood infections, heredity, advanced age, airway hyperresponsiveness, and occupational exposures. Some risk factors, including male sex and socioeconomic status, may gain their influence through associations with cigarette smoking or living conditions. More attention to primary, secondary, and tertiary prevention of COPD is required.

In order to reduce the burden of illness associated with COPD, greater efforts in smoking prevention and smoking cessation are required. Other measures include reductions in air pollution, influenza vaccination programs, and augmentation therapy for those with severe alpha-antitrypsin deficiencies. Once persons are afflicted with COPD, high quality medical care is essential.

Improved efforts in the prevention and management of COPD will require education directed toward patients, the general public, and health care providers. Considerably more research in education and prevention is necessary; this includes epidemiologic research to clarify the risk factors for dysfunction, biomedical research to substantiate information on the chemical markers of lung injury, expansion of the protease-inhibitor hypothesis, and evaluation of genetic predisposition toward the disease. Health services research will be required in order to create improved measures of health outcome, evaluate the cost-effectiveness of treatment programs, and improve the efficiency and effectiveness of both preventive and tertiary treatment programs. More research is necessary on cigarette use, access to care, and occupational exposures among members of underrepresented minority groups. Finally, it will be necessary to conduct more research to identify methods to help the general public adhere to preventive measures and to help COPD patients adhere to prescribed therapies.

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