Common Environmental Exposures and COPD

Chronic obstructive pulmonary disease (COPD) is a major cause of illness and death throughout the world and currently a leading cause of worldwide mortality\(^1\). Unlike other common causes of mortality, the burden of COPD continues to increase with projections. The National Institutes of Health had predicted that COPD will be the third leading cause of mortality in the US by 2030\(^3,3\), but the December 2010 “Deaths: Preliminary Data for 2008,” report from the Centers for Disease Control (CDC) and Prevention’s National Center for Health Statistics (NCHS) confirm that COPD has already become the third leading cause of death in the U.S. for 2008.

Presently, COPD affects about 10% of the general population\(^4\). Smoking tobacco is clearly a dominant risk factor for disease development particularly in genetically susceptible individuals. Therefore, the major focus of both research endeavors and disease modification has appropriately been on tobacco smoke. However, we now recognize that approximately 25% of all patients with COPD are lifelong nonsmokers\(^5\). This observation strongly supports that factors other than tobacco smoke contribute to the development of COPD. There is growing evidence to support that common environmental factors, other than tobacco smoke, are associated with COPD in both developed and developing countries. Some of these environmental factors could be modified and provide a tremendous opportunity to impact human health.

Nontobacco environmental risk factors that are associated with the development of COPD include: indoor air pollution, outdoor air pollution, occupational exposure to dust and fumes, and poor nutrition\(^6\). Each of these environmental factors is relevant to both developed and developing countries. Clear understanding of how each of these modifiable environmental risk factors contributes to the development and progression of disease will provide new insights into how we could prevent or treat COPD. In addition to direct effects of exposure, the environment can have a lasting impact on the risk or severity of lung diseases such as COPD by modification of either genetic code or epigenetics. For example, epigenetic marks from exposures modify disease risk for lung diseases through regulation of genes independent of the genetic code. Furthermore, epigenetic marks can be transferred from one generation to the next. Reducing current exposures may not only help us breathe better today, but could help our children tomorrow.

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Studies of outdoor air pollution demonstrate a link between lower lung function and high levels of particulate matter, sulfur dioxide, and nitrogen dioxide in the air. More importantly, high air pollution levels are clearly associated with an increased risk of death\(^7\) and loss of lung function\(^6\). Regulatory control of emissions by the US Environmental Protection Agency has had a dramatic impact on the level of outdoor air pollution in the US. For example, the US Clean Air Act of 1970 markedly improved air quality in the US over the last 40 years and as a result, significantly contributed to an increase in the life expectancy of the general US population during that time\(^8\).

Workplace exposures are estimated to account for approximately 15% of nontobacco related COPD in the US9. Occupational exposures associated with COPD include farming, mining, drilling tunnels, automotive repair, transportation, metal foundry, and manufacturing (concrete, plastic, rubber, leather, textiles). In general, the

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increased risk of COPD in these work environments is primarily associated with a higher level of inhaled particles, fumes, or dust. Agencies such as OSHA and NIOSH can minimize occupational risks by limiting exposures and implementation of preventive strategies. Associations between occupational exposures and COPD additionally provide an opportunity to better understand the mechanisms that lead to loss of lung function.

Recent evidence supports that possibly the most important global risk factor for COPD is exposure to indoor air pollution. An estimated 3 billion people in the world breathe polluted indoor air from burning of wood, crop residues, dung or coal to cook food or heat their homes. This compares with 1 billion people who smoke tobacco. Indoor burning of biomass or coal is estimated to account for 50% of COPD related deaths in developing countries. Both women and children have increased exposure to indoor air pollution and are at increased lifelong risk for COPD. However, there are new opportunities to reduce indoor air pollution and improve the health of women and children around the world. The United Nations Foundation in September 2010 announced a new program called the Global Alliance for Clean Cookstoves (web link: http://cleancookstoves.org). This program has set a goal of having a 100 million new cookstoves in developing countries by the year 2020. This has the potential to improve the air quality for poor women and children and could reduce COPD and other health risks for millions of people.

The risk for COPD may not only be increased by the air that we breathe, but may also be modified by the food that we eat. The risk for COPD from our diet is less clear when compared to what we know about tobacco smoke or air pollution. However, the emerging research is intriguing. Like with so many dietary recommendations that we hear today about good health, eating fresh fruits and vegetables that contain vitamins such as A, C, E and D, as well as eating omega-3 rich seafood seem to reduce risk of COPD. In contrast, diets deficient in these ingredients or high in processed foods such as cured meats may increase the risk of COPD. One recent example

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of the effect of diet on lung function is highlighted by evidence from a chronically undernourished population in Nepal\textsuperscript{12}. This study demonstrated that supplementation with vitamin A in the mother improved lung function in the child. As the future helps us better understand these risks for COPD, it seems that a diet of fresh fruits, vegetables and omega-3 rich seafood is not only good for your heart, it may additionally be good for your lungs.

Today, we recognize that environmental risk factors associated with COPD extend beyond exposure to tobacco smoke. We recognized the dangers of tobacco smoke on lung function almost 50 years ago\textsuperscript{13} and appropriately focused our attention on effective smoking cessation strategies to reduce individual risk as well as on changes in public policy to reduce risks for the public as a whole. The U.S. has helped lead the global effort in the fight against tobacco and to clean the air. However, we now recognize that almost 25% of patients with COPD never smoked a cigarette\textsuperscript{5}. We know some of the other environmental and dietary risk factors for COPD, but we still do not know enough. It is possible to reduce the global burden of COPD through our continued efforts to improve air quality in the atmosphere, home, and workplace in both developed and developing countries. We can support research efforts into learning more about other environmental and dietary causes of COPD. We can help with policy changes from our local communities to the global community. COPD is predicted to be the third most common cause of death in the world by 2030. The increased prevalence of COPD represents a cautionary warning for the current condition of our global environment and the profound impact on respiratory health. We can all help. We can achieve a better future for ourselves, our children, and our grandchildren.

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References:


