Chronic Obstructive Pulmonary Disease: An Overview of the Problem in West Virginia

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The Problem of COPD in West Virginia: Highlights

General Facts

► While Chronic Obstructive Pulmonary Disease (COPD) has been the fourth leading cause of death in the United States since 1991, it was the third leading cause of death in West Virginia for eight of the nine years from 2000 through 2008.

► COPD comprises two major diseases: chronic bronchitis and emphysema.

► According to data from the 2008 National Health Interview Survey, approximately 10 million adults in the United States have been diagnosed with chronic bronchitis and four million have been diagnosed with emphysema. Approximately one-quarter to one-third of smokers have undiagnosed COPD.

► The annual cost of COPD in 2010 is estimated to be $49.9 billion; more than half of this cost ($29.5 billion) represents direct costs (medical goods and services).

► Tobacco smoking is the most important risk factor for COPD, accounting for about 80% of all cases. The remaining cases are attributable to environmental exposures and genetic factors. There is a direct association between secondhand smoke and COPD. Smoking cessation is the single most effective way to reduce the risk of COPD and its progression.

West Virginia Statistics

► The American Lung Association estimates that in 2008, 64,000 West Virginia residents had been diagnosed with chronic bronchitis and 27,000 had been diagnosed with emphysema.

► In 2009, one-fourth (25.5%) of the state's adult population reported smoking cigarettes either every day or some days, ranking the state 2nd among the 50 states and the District of Columbia in current smoking prevalence. The U.S. median in 2009 was 17.9%. One-half (50.0%) of West Virginia adults are either current or former smokers, putting themselves at risk for COPD.

► Although the rates of smoking among West Virginia students are higher than the U.S. median in both middle and high schools, the rate in the state is falling more rapidly than the national rate. The state rate among high school students declined from 38.5% in 2000 to 22.3% in 2009, a decrease of 42%. The rate among middle school students decreased 51% over the same time period.

► West Virginia's rates of hospitalization for COPD have consistently been higher than the comparable U.S. rates, in some years twice the national rate. In 2006, the state rate was 45.7 hospitalizations per 10,000 population, 103% higher than the national rate of 22.5.

► Women in West Virginia were hospitalized with COPD at higher rates than men consistently from 1998 through 2008. The rate for women in 2008 was 59.8 hospitalizations per 10,000 population, compared with a rate of 45.7 for men.

► Over two-thirds of the COPD hospital discharges in West Virginia in 2008 were paid for by Medicare, followed by Medicaid.

► Age-adjusted mortality rates for COPD have traditionally been higher in West Virginia than in the United States as a whole. The state's rate was the 3rd highest in the country in 2002-2005. West Virginia's mortality rates for COPD exceed the national rates for both men and women and among all age groups.

► The state's rate of COPD as a contributing cause of death in 2006 was also higher than the national rate for both sexes and all age groups.

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I. Chronic Obstructive Pulmonary Disease: An Overview of the Problem

Chronic Obstructive Pulmonary Disease (COPD), a major cause of morbidity and mortality in West Virginia and the United States, is a term that refers to a group of slowly progressive lung diseases that are characterized by shortness of breath caused by airflow restriction. While COPD has been the fourth leading cause of death in the United States since 1991, it was the third leading cause of death in West Virginia for eight of the nine years from 2000 to 2008. According to a 2009 report from the National Institutes of Health, the state ranks third among the 50 states in COPD mortality after Wyoming and Nevada (2003-2005 age-adjusted data) (1).

This report is a revised and updated version of a report originally published in 2003 by the West Virginia Bureau for Public Health entitled *Chronic Lower Respiratory Disease: A Growing State and National Problem*. The original report included information on all chronic lower respiratory disease (CLRD); the current report concentrates on COPD. It seeks to present an overview of the causes and components of COPD, with an updated examination of research to date on prevalence, risk factors, and costs of the disease, as well as the most recently available statistics on COPD hospitalization and mortality in West Virginia.

Chronic bronchitis and emphysema account for the majority of cases of COPD, and the two diseases often coexist. Chronic bronchitis usually precedes emphysema in cases where both exist, except in a few genetically determined cases. The obstruction in chronic bronchitis and emphysema is irreversible but treatable. COPD does not include asthma, another major cause of airflow obstruction, in which the obstruction is reversible.[1]

According to data from the 2008 National Health Interview Survey, approximately 10 million adults in the United States have been diagnosed with chronic bronchitis, about 4.4% of the adult population (2). Almost four million people have been diagnosed with emphysema, approximately 1.7% of the nation's adult population. Thirteen percent (12.7%) of nursing home residents in 2004 had diagnosed COPD (3). Women were significantly more likely to have been diagnosed with chronic bronchitis than men (5.6% versus 2.8%), while men and women reported similar rates of emphysema (1.7% and 1.6%, respectively) (4). Recent research, however, has found that COPD underdiagnosis and undertreatment is widespread; two separate studies involving long-term smokers found that approximately one-fourth to one-third of study participants had undiagnosed obstructive pulmonary disease (5, 6). Both studies recommended that primary care physicians encourage testing for patients with COPD symptoms.

COPD takes a terrible toll. Results from Confronting COPD in America, a telephone survey of 573 participants with diagnosed COPD (89%) or COPD symptoms (11%), showed that nearly one-half of those surveyed became short of breath while washing or dressing (44%) or performing light household tasks (46%) (7). Nearly one-third of respondents (32%) became breathless while talking; 28% were short of

[1] COPD and asthma are grouped together by the International Classification of Diseases used by the World Health Organization to code mortality and diseases in a category termed Chronic Lower Respiratory Disease (CLRD). The present report addresses COPD only; for information on asthma in West Virginia, see *The Burden of Asthma in West Virginia*, published by the WV Bureau for Public Health, West Virginia Asthma Education and Prevention Program, August 2007.

breath even when sitting or lying still. Almost one-fourth (23%) reported that COPD had made them an invalid, while 8% were unable to leave home.

Over half (51%) of the survey respondents reported that COPD limited their ability to work. Fifty-three percent (53%) said their disease limited their social activities, and 46% said it affected their family activities. The psychological effect of COPD on patients was devastating. Over half of those surveyed said they felt panic when they could not get their breath (58%) or felt they were not in control of their breathing (52%). Two-thirds (66%) said they expected their condition to get worse.

An Overview of COPD

Lung Anatomy. The lungs work by drawing air into our bodies, allowing the oxygen to be absorbed into the blood and then removing and expelling carbon dioxide and other gases. Rib cage muscles and the diaphragm, in particular, contract when we inhale, which allows air to be sucked into the lungs. These muscles then relax, causing the air to be expired. During the process of inhalation, air travels through the nose and mouth, where mucus membranes warm and moisten the air and trap particles of foreign matter. The air continues down the throat into the trachea, or windpipe, which divides into the left and right bronchi leading into the lungs. Each bronchus then divides over and over into progressively narrower airways. The smallest airways (bronchioles) end in the alveoli, some 300 million tiny air sacs arranged in clusters. During inhalation, the alveoli expand, deflating when the lungs relax. It is in the alveoli that the oxygen in the air is absorbed by tiny blood vessels surrounding the sacs and carried to the rest of the body. At the same time, carbon dioxide and other waste gases pass from the blood into the alveoli and are exhaled.

Lungs are designed to stay healthy by expelling inhaled toxins and particulate matter. The mucus in the airways traps foreign particles, which are then moved up the bronchi into the trachea by little hairs called cilia to be expelled by coughing. This process is aided by macrophages, specialized cells in airway tissues that ingest toxins as part of the body's infection-fighting system. The introduction of greater-than-normal exposures to particulates and toxins in smoke, dust, or fumes damages this process, leading to the development of pulmonary inefficiency.

Chronic Bronchitis. The Merck Manual of Geriatrics defines chronic bronchitis as ". . . a clinical diagnosis . . . characterized by a productive cough occurring most days of the month for at least three months of the year for two consecutive years" (8). In chronic bronchitis, there is persistent inflammation and eventual scarring of the bronchial tubes. The inflammation causes excessive production of mucus and swelling of the bronchial walls. Acute bronchitis is triggered by viral or bacterial infections; the chief cause of chronic bronchitis, however, is cigarette smoking (including exposure to secondhand smoke). Long-term exposure to other irritants such as dust, fumes, and other pollutants and frequent prior viral or bacterial lung infections are also triggers. While smokers are most likely to develop chronic bronchitis, higher rates of the disease are also found among coal miners, grain handlers, metal workers, and others exposed to dust or irritating fumes.

Chronic bronchitis is often not treated properly until it has reached an advanced stage because sufferers do not realize its seriousness until their lungs have already been damaged. By this time, the lining of the bronchial tubes has thickened due to the persistent inflammation, excess mucus is constantly being produced, there is much coughing, airflow is increasingly hampered, and respiratory infections occur with greater frequency due to the ideal breeding conditions of the bronchi. Chronic

inflammation in the smaller, peripheral airways (those having a diameter of less than 2 mm) leads over time to scar tissue formation and additional fixed airway obstruction. In severe cases, the lung damage can produce pulmonary hypertension, leading to cor pulmonale, a failure of the right side of the heart due to increased workload on the right ventricle from the pulmonary hypertension. Overall, the 10-year mortality rate following a diagnosis of chronic bronchitis is >50% (9).

Emphysema. Emphysema is a pathological definition: abnormal permanent enlargement of the air spaces at the ends of the bronchioles, accompanied by the destruction of their walls. Most emphysema is caused by smoking; however, approximately 50,000 to 100,000 current U.S. cases, primarily among individuals of Northern European descent, are the result of an inherited condition known as alpha-1 antitrypsin (ATT) deficiency. ATT, a protein produced by the liver, can also be deactivated by cigarette smoke, causing an environmentally induced deficiency. What ever the cause of the deficiency, the results are the same. ATT normally works to inhibit the enzyme elastase, which acts to destroy a protein called elastin. Elastin ("elastic") forms the underlying structure of the lungs and is the material that gives the air sacs, or alveoli, their vital ability to stretch and recoil after filling with air. As elastin is destroyed, alveoli walls can break down, forming large, permanent distended air sacs. Emphysema sufferers must then forcefully expel the air from their lungs, increasing the use of their respiratory muscles. This puts pressure on the airways from the outside, causing even more damage to both the airways and the alveoli. Eventually the exchange of oxygen and carbon dioxide is compromised, producing hypoxemia (oxygen deficiency) and hypercapnia (excess carbon dioxide).

Emphysema develops gradually. The initial symptom of emphysema is dyspnea, or shortness of breath; a cough might also be present, as chronic bronchitis often precedes emphysema. As the disease progresses, even a short walk can result in difficulty in breathing. The most common conditions leading to death from emphysema are respiratory acidosis (alterations in blood pH due to buildup of carbon dioxide), cor pulmonale, and massive collapse of the lungs due to pneumothorax (accumulation of air in the pleural cavity surrounding the lungs).

The Economic Costs of COPD

According to the National Heart, Lung, and Blood Institute (NHLBI), the annual cost of COPD in 2010 is estimated to be $49.9 billion; more than half of this cost ($29.5 billion) represents direct costs (medical goods and services) (10).

A study presented at the American Thoracic Society International Conference in 2006 used a mathematical model to estimate future COPD-related costs based on the $31.4 billion in medical expenses estimated by the NHLBI in 2003. The model predicted that medical costs related to COPD would total about $832 billion over the next 20 years in the United States alone (11). The study, part of the Burden of Obstructive Lung Disease (BOLD) initiative established to examine the prevalence and burden of COPD worldwide, estimated that the disease would cost the United States $176.6 billion over the first five years of the projections, and $389.2 billion over the next 10 years. Even though tobacco use, the primary risk factor for COPD, is declining, the prevalence of COPD is expected to increase due to lag time between smoking exposure and the development of the disease.

Mapel et al. examined the direct medical costs of undiagnosed COPD in a 2008 article in *Value in Health*. Their findings showed that total utilization and direct medical costs were on average $1,182 higher per patient in the two years before an initial COPD diagnosis and $2,389 higher in the 12 months

just before the diagnosis, compared with matched controls (12). Most of the excess costs before diagnosis were due to hospitalizations. Following the diagnosis, outpatient and pharmacy costs were found to be 50% to 100% higher for COPD patients during a two-year follow-up study.

Risk Factors for COPD

Age and Gender. Older individuals are more likely to be diagnosed with COPD (it has been estimated that 10% of the population aged 55-85 have COPD [13]). COPD is a progressive disease; according to Dr. Stephen Rennard, a pulmonary specialist and participant in the Global Initiative for Chronic Obstructive Lung Disease (GOLD)[2], "Typically, it takes 30 to 40 years for COPD to develop fully enough to cause symptoms. Before that, it is present, but silent" (14). Chronic bronchitis is about twice as common among women as men; nationally in 2008 the rate among women was 57.6 per 1,000 compared with a rate of 28.6 per 1,000 among men (15). Emphysema rates have traditionally been higher among men in the United States, but the 2008 rate for women (17.3 per 1,000) was higher than that for men (16.3 per 1,000) for the first time (15).

Tobacco Smoking. Tobacco smoking is by far the most important risk factor for COPD, accounting for approximately 80% of all cases, according to American Lung Association (ALA) data (16). The latest statistics from the ALA estimate that male smokers are nearly 12 times as likely to die from COPD as men who have never smoked; female smokers are nearly 13 times more likely to die from COPD than women who have never smoked. Pipe and cigar smokers also have greater COPD morbidity and mortality than nonsmokers; however, their rates are somewhat lower than those for cigarette smokers (16). Even given these alarming statistics, a 2004 ALA survey found that nearly two-thirds (64%) of smokers were not concerned about developing COPD. Even more surprising, 55% of these smokers were experiencing at least one symptom of COPD at least once a week (17).

Cigarette smoke affects the lungs in several ways. It increases mucus production in the airways (even contributing to an increase in the number of mucus glands), while at the same time stopping the cilia from working, allowing the buildup of mucus and the contaminants it contains. Over time this results in obstruction of the airways, causing chronic bronchitis. The smoke also attracts inflammatory cells into the lungs, which release elastase. Elastase breaks down elastin, normally protected by the inhibitor ATT; however, cigarette smoke attracts more cells and stimulates the release of more elastase than can be countered by the ATT. In addition, the smoke itself inactivates ATT, and it is the imbalance between elastase and ATT that leads to the development of emphysema.

A 2009 study presented at the 105th International Conference of the American Thoracic Society provided strong evidence that women are more susceptible to the lung-damaging effects of cigarettes than men (18). The study included current or ex-smokers and COPD subjects with moderate or severe disease. Overall, there were no gender differences in lung function and COPD severity; however, female subjects were on average younger and had smoked significantly less than men. The difference in susceptibility to COPD was found to be greater when smoking exposure was lower. According to the study's lead author, the differences may be explained by the fact that "women have smaller airways; therefore each cigarette may do more harm. Also, there are gender differences in the metabolism of cigarette smoke. Genes and hormones could also be important."

[2] GOLD is a collaborative project of the U.S. National Heart, Lung, and Blood Institute (NHLBI) and the World Health Organization (WHO).

While 80% to 90% of COPD is caused by long-term smoking, only about 25% of chronic tobacco smokers develop COPD, leading scientists to conclude an overall varying genetic susceptibility to lung damage may exist (19). Susceptible individuals show an accelerated rate of lung function decline that can result in severe COPD. Even if these individuals stop smoking, the progression of the disease will slow down, but existing symptoms will not improve. This overall (non-gender-specific) genetic predisposition to bronchial hyperresponsiveness is known as the "Dutch Hypothesis" (20). A 2009 study also looked at a genetic link between smoking and the development of COPD in later life. The researchers compared long-term smokers with COPD with a control group of long-term smokers without COPD (19). They found DNA sequence variations in a gene known as ADAM33 that were more frequent in the COPD group than the control group. One variation, called S1, had a particularly strong link to lung abnormalities. Studies continue on possible genetic differences between smokers who develop COPD and those who do not.

Passive smoke, also known as secondhand smoke, is a mixture of mainstream smoke that is exhaled by a smoker and sidestream smoke that comes from the lighted cigarette, pipe, or cigar (and actually is higher in concentrations of toxins than exhaled smoke). Secondhand smoke is a major polluter of indoor air. A recent (2007) study published in *Lancet* found a significant association between passive smoke exposure during adulthood and the prevalence of COPD (21). Duration of exposure to passive smoke was examined among older adults (age 50+), with those never smokers who were exposed at least 40 hours per week for more than five years significantly more likely to develop COPD than those with less exposure. Eisner et al. found that directly measured secondhand smoke was associated with worse COPD severity, independent of personal smoking (22).

Other Risk Factors. That exposure to vapors, gases, dust, or fumes on the job can also cause COPD independently of cigarette smoking and increase the risk and severity of disease among smokers is well-documented and supported by good evidence (23, 24). A 2002 study by the National Institute for Occupational Safety and Health (NIOSH) suggested that nearly one in five (19.2%) cases of COPD were attributable to occupational exposures and that nearly one-third (31.3%) of COPD cases among never smokers were job-related (25). The NIOSH study found COPD to occur more frequently in certain industries, including rubber, plastic, and leather manufacturing, utilities, and office building services (laborers, cleaners, material handlers, and maintenance workers), even after controlling for smoking and socioeconomic factors. By job, the NIOSH researchers found freight, stock, and material handlers and members of the armed forces to be at greatest risk.

Researchers in a large international study examined the relationship between occupational exposures and chronic bronchitis among 13,253 men and women aged 20 through 44 in 14 industrialized countries (26). Even at relatively young ages, occupational exposure to vapors, gases, dust, or fumes was found to be associated with chronic bronchitis among smokers. An increased risk was found among both smokers and nonsmokers who were agricultural, textile, paper, wood, chemical, or food processing workers, with the risk more pronounced among smokers.

The relationship between coal mining and obstructive lung disease has long been recognized (27). An in-depth study by NIOSH examined additional determinants of declining lung function among coal miners (28). The study, involving 264 underground coal miners whose health was followed for 11 years, examined occupational and non-occupational exposures, smoking, personal and family medical histories, and childhood living conditions. Certain jobs within the mining industry were found to be more conducive to lung function declines, i.e., roof bolting, exposure to explosive blasting, and exposure to water used for spraying (to control dust) that was stored in holding tanks. Non-mining factors found

to be associated with COPD were smoking, body mass index, weight gain in adulthood, childhood pneumonia, and childhood exposure to passive smoke in the home, as well as smoke from wood and coal-burning stoves.

Genetic factors such as the ATT deficiency discussed earlier and any factors that affected lung growth during gestation and childhood, as well as frequent childhood respiratory infections, can also influence the risk of developing COPD.

Diagnosis of COPD

While early diagnosis cannot reverse the damage already done to an individual's lungs by COPD, it can result in interventions that can slow down the progression and improve symptoms. A diagnosis of COPD should be suspected when there is a history of exposures to risk factors accompanied by some airflow limitation, with or without other symptoms. The *GOLD Pocket Guide to COPD Diagnosis, Management, and Prevention* recommends that all patients who have shortness of breath, a chronic cough, and sputum production, along with risk factor exposure, should be tested for airflow limitation (29).

Lung function is most commonly assessed by spirometry, a test that measures how much and how quickly air is expelled from the lungs after a patient takes a deep breath. The patient breathes out forcefully into a device called a spirometer. Two measures, the total amount of air that can be expelled following taking the breath (forced vital capacity [FVC]) and the amount of air that can be expelled in one second (forced expiratory volume in one second [FEV_1]), are normally used for assessment. The ratio of FEV_1 to FVC (FEV_1/FVC) is indicative of the degree of a patient's airflow obstruction. If obstruction exists, the air will not be expelled as quickly, and the percentage will be lower. Normally, 75% to 85% of the air in a person's lungs can be expelled in the first second, but this depends on the individual's age, gender, height, and race. The expected percentage is higher among persons who are younger, tall, male, and white. A number that is smaller than 85% of the expected percentage is considered low, or abnormal.

The severity of COPD is classified by FEV_1/FVC values. A person is considered at risk for COPD if he or she has a normal spirometry reading but has chronic symptoms, i.e., cough and sputum production. Stage I (mild), Stage II (moderate), Stage III (severe), or Stage IV (very severe) COPD diagnoses are based on FEV_1/FVC values as a percentage of predicted values for the individual. Arterial blood gas levels, typically abnormal in patients with moderate and severe COPD, are used to diagnose hypoxemia (low blood oxygen) and concomitant hypercapnia (too much carbon dioxide in the blood) as the disease progresses.

Treatment of COPD

Smoking Cessation. The advice from the multinational GOLD initiative is unequivocal: "Smoking cessation is the single most effective and cost-effective way to reduce the risk of developing COPD and slow its progression" (29). Secondhand smoke should also be avoided, as well as occupational exposures and other indoor and outdoor pollutants.

The Lung Health Study Group studied 3,926 smokers with mild-to-moderate airway obstruction who were separated into a smoking cessation group or a nonintervention group. Their lung function was measured annually for five years. Subjects who quit smoking experienced an improvement in their FEV_1 in the year after they quit, and the subsequent rate of decline in FEV_1 was half that of participants who continued to smoke, comparable with that of never-smokers (30). The benefits the quitters derived were independent of advanced age, poor baseline lung function, airway hyperresponsiveness, or amount previously smoked.

Researchers in the Boston Early-Onset Study found that patients with severe COPD who stop smoking may be able to improve their rate of survival despite the severity of their disease (31). Participants in the study who continued to smoke had a risk of mortality that was nearly three times that of the participants who stopped smoking. According to the lead author, the study ". . . confirms the importance of smoking cessation even in patients with the most advanced stages of lung disease."

Medications. To date, no medications for COPD exist that modify the long-term decline in lung function. Drug therapy, however, can be helpful in alleviating symptoms and complications of the disease. Bronchodilators, which can be administered as inhalers, pills, or liquids, relax and open air passages in the lungs and are the most commonly used medications in treating symptoms. There are different types of bronchodilators; some are short-acting, others long-acting. They can be used intermittently to provide symptom relief or routinely to prevent or reduce symptoms, depending on the patient and the stage of the disease.

In moderate and severe disease, inhaled glucocorticosteroids, which reduce inflammation, swelling, and mucus production, may be prescribed. Regular treatment with glucocorticosteroids does not modify the long-term decline in lung function, but can reduce the frequency of exacerbations. Chronic use, however, can result in a heightened risk of adverse effects, including osteopenia, cataracts, hyperglycemia, secondary infection, cardiovascular disease, and behavioral changes; therefore, long-term treatment is not recommended.

Antibiotics are an important component of COPD treatment when infectious exacerbations occur, but are not recommended for long-term use. Influenza and pneumococcal vaccines can be beneficial in reducing serious illness and mortality in COPD patients. Influenza vaccines have been shown to reduce COPD morbidity and mortality by 50% (32). The results of a study of a new vaccine designed to reduce the number and severity of exacerbations in patients with severe COPD were published in *Chest* in April 2010. The results showed significant reductions in moderate to severe exacerbations in terms of duration, antibiotic use, and hospital admissions (33).

Pulmonary Rehabilitation. Pulmonary rehabilitation includes exercise training, education on disease management, nutritional counseling, and psychosocial support. Regular physical activity has been shown to improve the quality of life for COPD patients in all severities of the disease (34). A population-based study published in *Thorax* in 2006 found that participants with COPD who had some level of regular physical activity, whether low, moderate, or high, had a lower risk of both hospital admissions and mortality than patients who reported very low physical activity (35).

Oxygen Therapy. Long-term oxygen therapy is usually initiated in Stage IV COPD or when there is evidence of congestive heart failure, pulmonary hypertension, or polycythemia. Participants in the Medical Research Council trial, a study of hypoxemic COPD patients, i.e., those with inadequate blood oxygen levels, received either continuous oxygen (at least 15 hours/day) or no supplemental oxygen.

Those who received oxygen showed a survival advantage of five years (36). Another study of hypoxemic COPD patients showed that continuous oxygen therapy (at least 17.7 hours/day) was more beneficial to long-term survival than fewer hours, such as nocturnal oxygen only. Those patients receiving continuous oxygen also reported fewer hospitalizations (36).

Ambulatory oxygen refers to supplemental oxygen used during exercise and the activities of daily living. A review of 31 studies examining the overall benefit of ambulatory oxygen showed that it did improve exercise performance in terms of endurance and maximal exercise capacity; however, the reviewers concluded that more research was necessary to establish the net long-term benefit in patients with different levels of hypoxemia (37).

Surgery. For severe emphysema sufferers, there are three surgical options available that can provide improvement in their symptoms and prevent complications. A bullectomy is the surgical removal of a bulla, a large distended air space in the lung that is greater than one centimeter in diameter. Bullae, which form as the result of tissue damage and do not contribute to the exchange of oxygen and carbon dioxide, compress the healthy alveoli around them, limiting their ability to function. Bullae may also rupture, causing pneumothorax, or collapsed lung. When a bulla is removed, the healthy air sacs can expand and the muscles used to breathe are able to function better, resulting in improved respiration. There is usually only one large bulla removed during the procedure.

Lung volume reduction surgery (LVRS) is performed on patients who have smaller, nonfunctioning air sacs distributed more widely through the lung. In this procedure, badly diseased tissue is removed, approximately 30% to 40% of one lung. LVRS improves lung function by increasing the space in the chest cavity, allowing the patient's diaphragm to better function while pumping air into and out of the lungs, and by restoring elastic recoil in the alveoli. The surgery works best for patients whose emphysema is confined to the upper parts of the lungs and who have very low exercise tolerance. Those patients will have a longer survival with surgery than with medical treatment only (38).

Lung transplantation is a complicated and expensive procedure that is normally the last resort for emphysema sufferers. Transplantation involves the removal of one or both diseased lungs from a patient and the replacement of the lungs with healthy organs from a donor. Post-operative complications can include infection, organ rejection, kidney damage, hypertension, osteoporosis, and lymphoma. According to the Organ Procurement and Transplantation Network (OPTN), there were over 1,600 lung transplants performed in the United States in 2009; in June 2010, there were approximately 1,800 individuals on the waiting list (39). Data from the OPTN show, three years post-transplant, a 60.7% survival rate for single-lung recipients and a 64.9% survival rate for double-lung recipients who received their transplants from 1999-2002 (40).

II. Chronic Obstructive Pulmonary Disease: The West Virginia Problem

COPD is a serious problem in West Virginia due to the presence of several of the major risk factors associated with the condition. In 2008, West Virginia residents had the third highest median age among all the states (40.6) (41), in addition to high rates of smoking among its adults and adolescents. To compound this already high-risk situation, the American Lung Association's *State of the Air 2010* lists four West Virginia counties among the 25 highest counties in the nation in terms of being most polluted by year-round particle pollution: Kanawha, Brooke, Cabell, and Berkeley (42). Nearly one-fourth (23%) of the state's residents live in one of these counties.

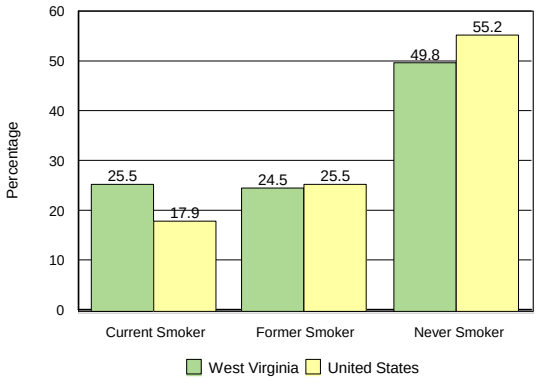
Prevalence of Chronic Bronchitis and Emphysema. The 2008 National Health Interview Survey included two questions addressing COPD: (1) Have you been told by a doctor or other health professional that you had chronic bronchitis in the past 12 months? and (2) Have you EVER been told by a doctor or other health professional that you had emphysema? An estimated 91,000 residents answered "yes" to one or both of these questions, 64,000 reporting chronic bronchitis and 27,000 reporting emphysema (43). The estimated prevalence of chronic bronchitis was unchanged from 2002, while that for emphysema was approximately 20% higher than the 2002 estimate.

COPD and Smoking

As noted, cigarette smoking is the most important risk factor for COPD. West Virginia has traditionally reported high rates of smoking among both its adult and adolescent populations. In 2009, one-fourth (25.5%) of the state's adult population reported smoking either every day or some days, according to data from the Behavioral Risk Factor Surveillance System (BRFSS), ranking the state 2nd among the 50 states and the District of Columbia in smoking prevalence (44). As shown in Table 1, the state's rate of smoking in that year exceeded the national median regardless of gender, age, educational level, or household income level.

Former smoking is also a risk factor for chronic bronchitis and emphysema, although risk decreases after quitting smoking. One-half (50.0%) of West Virginia adults are either current or former smokers, compared with a national median of 43.4%, a marked difference (Fig. 1). As depicted in Figure 2, the state's percentages of current, former, and never smokers have changed little in the 15 years from 1995 through 2009.

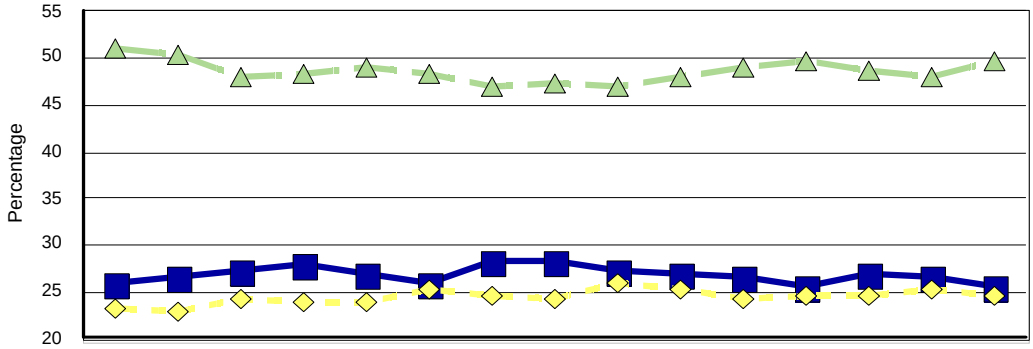
Fig. 1. Prevalence (%) of Adults by Smoking Status
West Virginia and United States* BRFSS, 2009



*US median

Table 1. Prevalence of Current Cigarette Smoking among Adults by Selected Demographics West Virginia and United States, 2009 Behavioral Risk Factor Surveillance System			
	West Virginia		United States
Demographic	Prevalence (%)	(95% CI)	Median (%)
Total	25.5	(23.9-27.2)	17.9
Gender			
Male	27.6	(24.9-30.3)	19.6
Female	23.6	(21.7-25.5)	16.8
Age			
18-24	30.6	(22.8-38.4)	23.1
25-34	41.6	(36.5-46.6)	23.7
35-44	26.9	(23.1-30.7)	18.2
45-54	27.3	(24.0-30.6)	20.5
55-64	21.6	(19.0-24.2)	16.1
65+	10.6	(8.9-12.4)	8.2
Educational Level			
<12 years	39.8	(34.9-44.7)	31.4
12 years or GED	28.3	(25.6-31.0)	24.8
13-15 years	25.0	(21.7-28.3)	19.9
16+ years	11.5	(9.2-13.9)	8.2
Household Income			
<$15,000	35.1	(29.8-40.4)	31.3
$15,000-$24,999	34.6	(30.3-38.9)	28.0
$25,000-$34,999	31.5	(26.8-36.2)	24.0
$35,000-$49,999	24.3	(20.6-28.0)	19.6
$50,000+	15.7	(13.2-18.1)	12.1

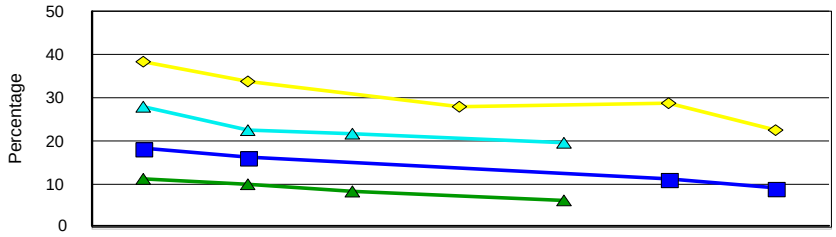
Fig. 2. Prevalence (%) of Adults by Smoking Status
West Virginia BRFSS, 1995-2009



	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Current	25.8	26.6	27.4	27.9	27.1	26.1	28.2	28.4	27.3	26.9	26.7	25.7	26.9	26.5	25.5
Former	23.3	22.9	24.3	23.8	23.8	25.3	24.7	24.2	25.8	25.1	24.3	24.7	24.5	25.4	24.5
Never	51.1	50.5	48.2	48.3	49.0	48.5	47.1	47.4	46.9	48.0	49.0	49.6	48.6	48.1	49.8

Youth Smoking Prevalence. While data from the BRFSS on adult smoking in the state show little change over the years the behavior has been monitored, the data on youth smoking from the West Virginia Youth Tobacco Survey (45) are more encouraging. Figures 3 and 4 illustrate the decline in smoking among middle and high school students since 2000. Although the rate for West Virginia students is higher than the United States median in both middle and high schools, the rate in the state is falling more rapidly than the national rate. The state rate of current smoking among high school students decreased from 38.5% in 2000 to 22.3% in 2009, a 42% decline; the rate among middle school students decreased 51% over the same time period. The national median of current smoking declined 30% among high school students between 2000 and 2007.

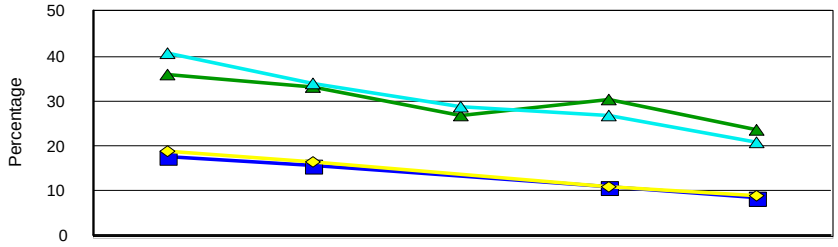
Fig. 3. Prevalence* (%) of Current Smoking Among Middle School (MS) and High School (HS) Students WV and US** Youth Tobacco Survey, 2000-2009



	2000	2002	2004	2005	2006	2007	2009
WV MS	18.1	16.3				11.1	8.9
WV HS	38.5	33.7		27.8		28.8	22.3
US MS	11.0	9.8	8.4		6.3		
US HS	28.0	22.5	21.7		19.7		

*Blank cells indicate no data are available in those years for MS and/or HS students
**US median

Fig. 4. Prevalence (%) of Current Smoking Among Middle School (MS) and High School (HS) Students By Gender, WV Youth Tobacco Survey, 2000-2009



	2000	2002	2005*	2007	2009
MS Boys	17.5	15.8		11.1	8.7
MS Girls	18.8	16.6		10.9	8.9
HS Boys	36.0	33.3	26.6	30.2	23.7
HS Girls	40.6	34.1	28.9	26.6	20.9

*Data not available for MS students

COPD Hospitalizations

Hospitalization data provide important information on the burden of specific conditions on a population, both in terms of human suffering and financial cost. Data were obtained from the West Virginia Health Care Authority (WVHCA)[3] to estimate COPD hospitalizations among state residents from 1998 through 2008 (50), with comparisons with U.S. data through 2006. National data were obtained from the National Hospital Discharge Survey (15). All data reflect COPD as a first-listed, or principal, diagnosis only.

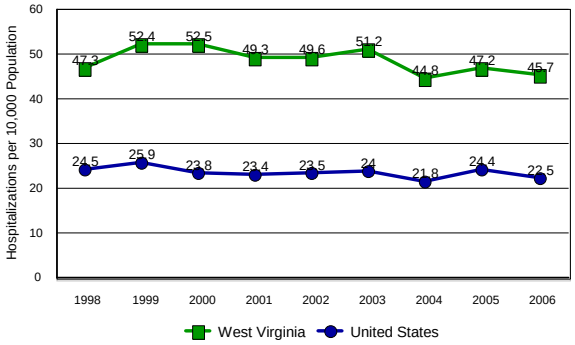
As shown in Figure 5, West Virginia's rates of hospitalization for COPD have consistently been higher than the comparable U.S. rates, in some years more than twofold the national rates. In 2006, the latest year available for U.S. data at the time this report was prepared, the state rate was 45.7 hospitalizations per 10,000 population, 103% higher than the national rate of 22.5.

West Virginia women have been hospitalized with a first-listed diagnosis of COPD at a higher rate than men during the decade from 1998 through 2008 (Fig. 6). The highest rates for both sexes were reported in 2008, with a rate of 59.6 hospitalizations per 10,000 population for women and a rate of 45.7 for men.

Women with a first-listed diagnosis of COPD also have a slightly longer average length of stay (ALOS) than men with a comparable diagnosis. In 2008, the ALOS for women with a COPD diagnosis was 5.0 days, compared with an ALOS of 4.8 for men. The ALOS for all discharges in 2008, however, was higher among men than among women, 5.2 days versus 4.7 days, respectively.

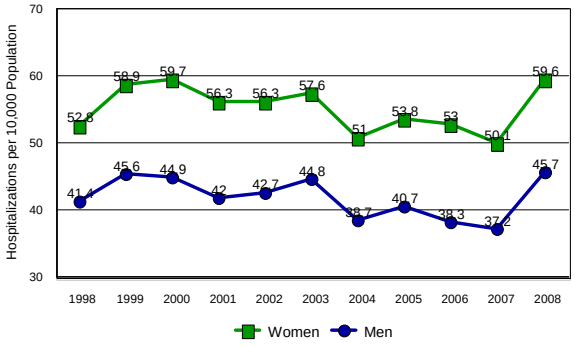
Figure 7 illustrates the distribution of discharges from acute care facilities by payer. Over two-thirds (67.6%) of the discharges were reimbursed by Medicare, followed by Medicaid (13.1%).

Fig. 5. COPD First-Listed Hospital* Discharge Rates**
West Virginia and United States, 1998-2006



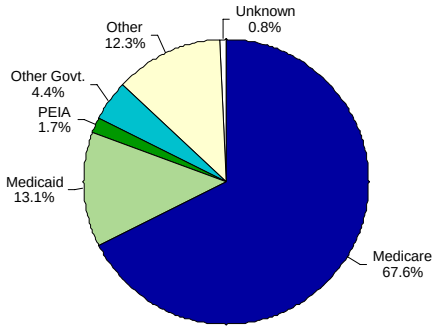
*Acute care facilities
**Rates per 10,000 population
Note: ICD-9 codes 490-492, 494-496

Fig. 6. COPD First-Listed Hospital* Discharge Rates**
By Gender, West Virginia, 1998-2008



*Acute care facilities
**Rates per 10,000 population
Note: ICD-9 codes 490-492, 494-496

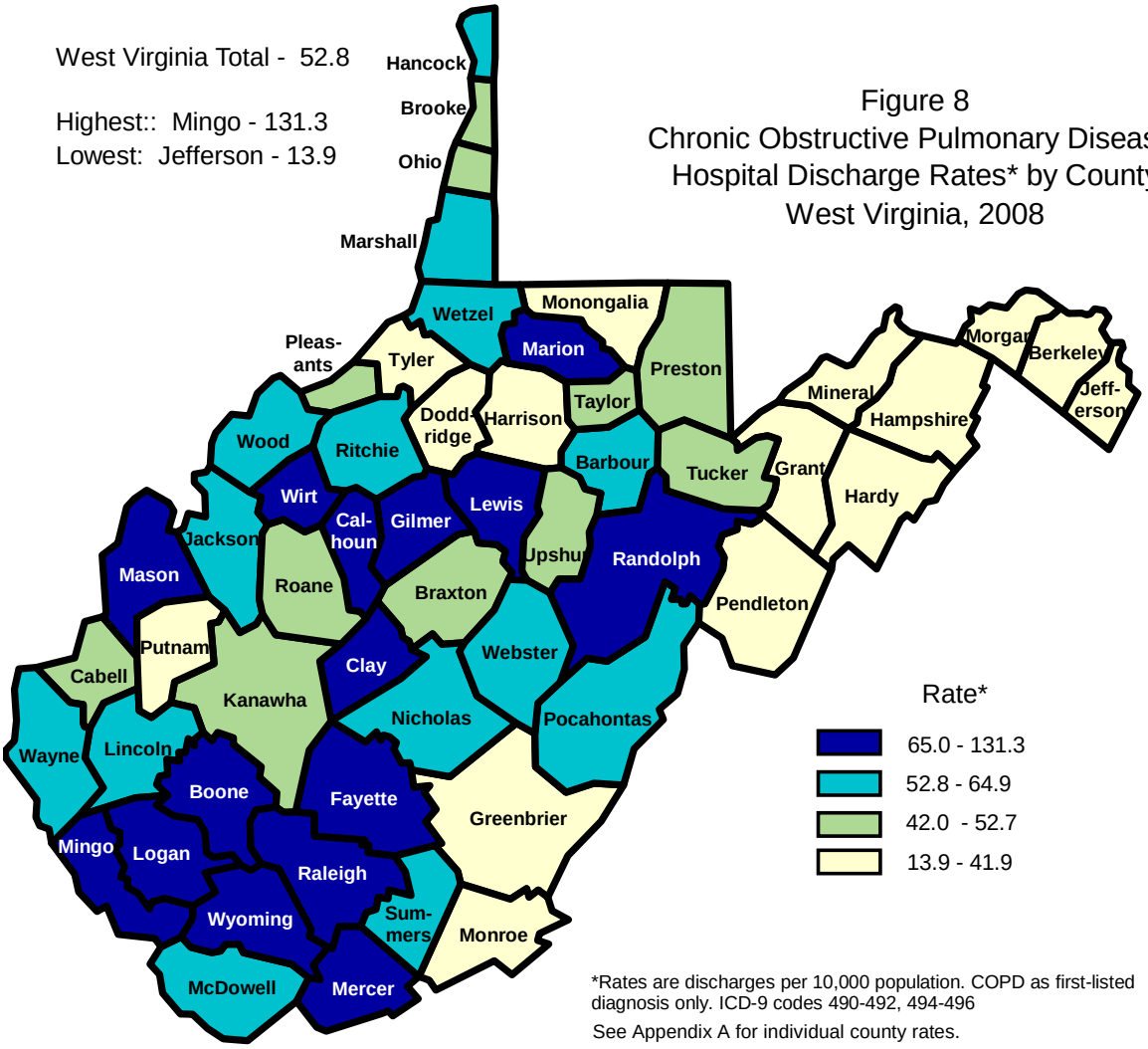
Fig. 7. Distribution of Hospital* Discharges** with a First-Listed Diagnosis
Of COPD by Payer, West Virginia Inpatient Records, 2008



*Acute care facilities
**9,574 total discharges
Note: ICD-9 codes 490-492, 494-496

[3] The WVHCA collects inpatient data from all nonfederal licensed hospitals in the state, as well as Medicare data on West Virginia residents hospitalized in out-of-state hospitals. The data presented in this report reflect inpatient hospitalizations in acute care, or short-stay, hospitals only.

Rates of COPD hospitalizations varied greatly around the state in 2008. Figure 8 illustrates those differences by county. The rates (hospital discharges with a first-listed diagnosis of COPD per 10,000 population) ranged from a low of 13.9 hospitalizations per 10,000 in Jefferson County to a high of 131.3 in Mingo County. The highest rates are seen in the southern and central counties of the state. Individual county rates can be found in Appendix A.



COPD Mortality

While COPD has been the fourth leading cause of death in the United States since 1991, it has been the third leading cause of death in West Virginia for eight of the nine years between 2000 and 2008. The seriousness of the problem of COPD in the state is reflected in the disease's morality rates: In 2006, the age-adjusted COPD mortality rate in West Virginia was 54.8 deaths per 100,000 population, compared with a national age-adjusted rate of 39.3 (46).

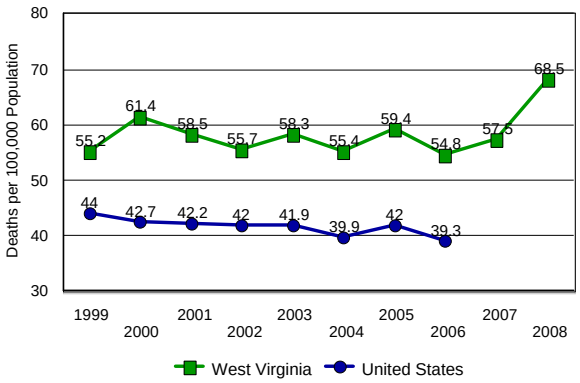
As Figure 9 shows, West Virginia's age-adjusted mortality rates for COPD were consistently higher than U.S. rates from 1999 through 2006. The state's rates increased in both 2007 and 2008, to a high of 68.5 deaths per 100,000 population in 2008, a 19% increase from the previous year.

When examined by gender and race, in 2006 West Virginia had higher age-adjusted mortality rates due to COPD in every group examined (Fig. 10). The state rate among men exceed the national rate by over 35% (63.1 deaths per 100,000 males vs. 46.7), while the state rate among women was 43% higher than the comparable national rate (49.8 deaths per 100,000 females vs. 34.9). White residents had a rate that was about 35% higher than their counterparts nationwide (56.1 vs. 41.6). African Americans in the state had a mortality rate that was slightly higher (7%) than the comparable

U.S. rate (27.0 vs. 25.3).

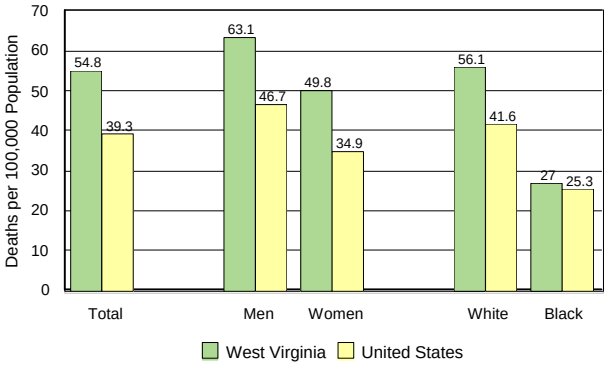
Figure 11 presents 2006 age-specific mortality rates for persons aged 45 and older. As shown, West Virginia's rates exceeded national rates in every age group. The state's rate in the youngest group shown, those aged 45-54, was over twice the U.S. rate. The percentage difference in rates lessened as the ages increased; however, the rate for ages 85 and older was still 16.2% higher in the state than in the nation as a whole.

Fig. 9. COPD Mortality Rates*
West Virginia and United States, 1999-2008



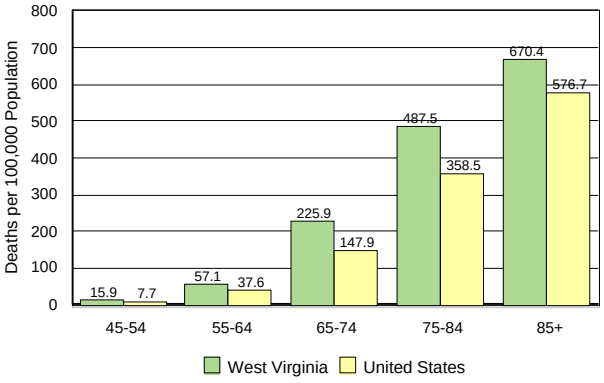
*Rates are age-adjusted to the 2000 U.S. standard million.
Note: ICD-10 codes J40-J474, J47

Fig. 10. COPD Mortality Rates* by Gender and Race**
West VIrginia and United States, 2006



*Rates are age adjusted to the 2000 U.S. standard milllion.
**Number of deaths among other races was too small to meet standard of reliability.
Note: ICD-10 codes J40-44, J47

Fig. 11. COPD Mortality Rates* by Age Group
West VIrginia and United States, 2006



*Age-specific crude rates
Note: ICD-10 codes J40-44, J47

The highest rates of COPD mortality in 2008 were primarily clustered among the southern and central counties of West Virginia (Fig. 12). Age-adjusted rates ranged from a high of 127.9 deaths per 100,000 population in Mingo County to a low of 34.6 in Hardy County. A complete list of county rates can be found in Appendix B.

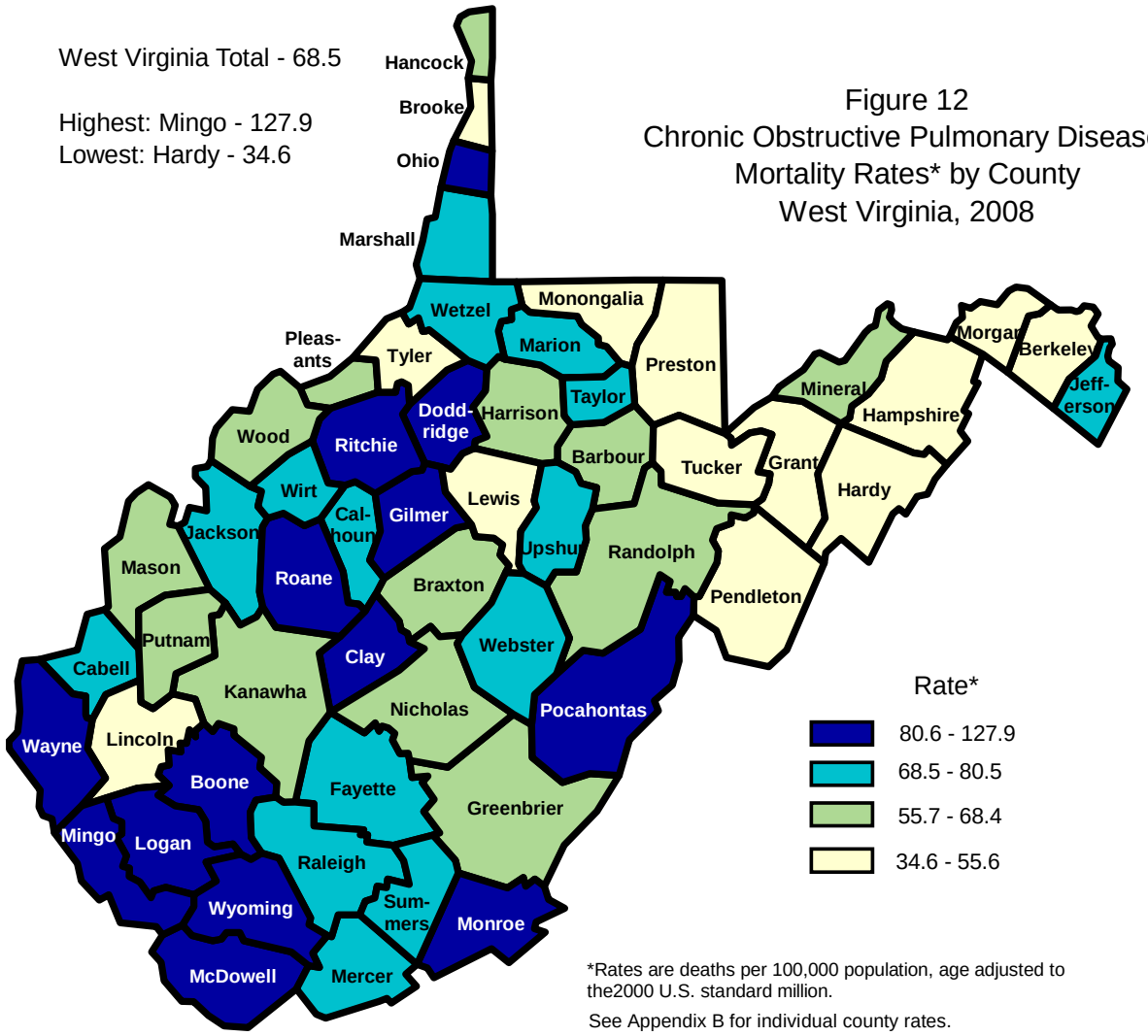


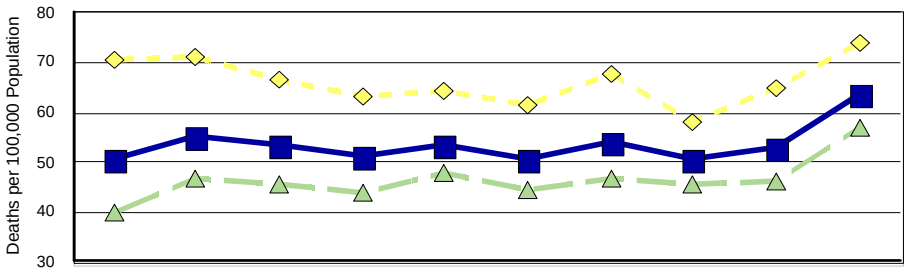
Figure 12
Chronic Obstructive Pulmonary Disease
Mortality Rates* by County
West Virginia, 2008

*Rates are deaths per 100,000 population, age adjusted to the2000 U.S. standard million.

See Appendix B for individual county rates.

Figures 13 and 14 illustrate the trends in chronic bronchitis and emphysema mortality from 1999 through 2008. The state's overall age-adjusted rate for deaths from chronic bronchitis increased from 50.7 deaths per 100,000 population in 1999 to 63.6 in 2008. The increase in chronic bronchitis deaths occurred among both men and women. The overall rate for emphysema mortality has fluctuated little since 2000, as has the rate among women. The rate among men has decreased since 2004, with a small increase in 2008.

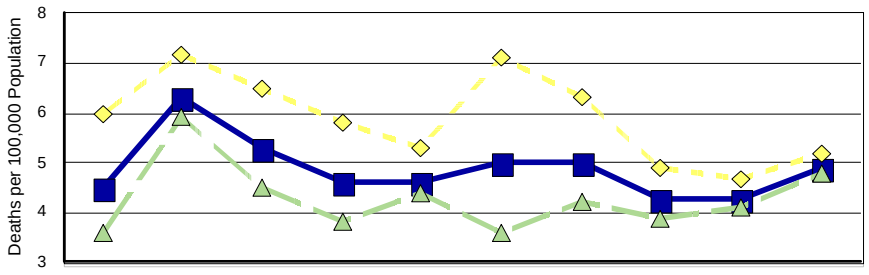
Fig. 13. Mortality Rates* for Chronic Bronchitis by Gender
West Virginia, 1999-2008



	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008
Total	50.7	55.1	53.3	51.1	53.7	50.5	54.4	50.5	53.2	63.6
Men	70.8	71.2	66.9	63.5	64.1	61.3	67.7	58.3	64.8	74.1
Women	39.8	46.8	45.9	44.0	47.9	44.6	47.0	45.9	46.1	56.8

*Rates are age adjusted to the 2000 U.S. standard million.
Note: ICD-10 codes J40-J42, J44, J47

Fig. 14. Mortality Rates* for Emphysema by Gender
West Virginia, 1999-2008



	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008
Total	4.5	6.3	5.3	4.6	4.6	5.0	5.0	4.3	4.3	4.9
Men	6.0	7.2	6.5	5.8	5.3	7.1	6.3	4.9	4.7	5.2
Women	3.6	5.9	4.5	3.8	4.4	3.6	4.2	3.9	4.1	4.8

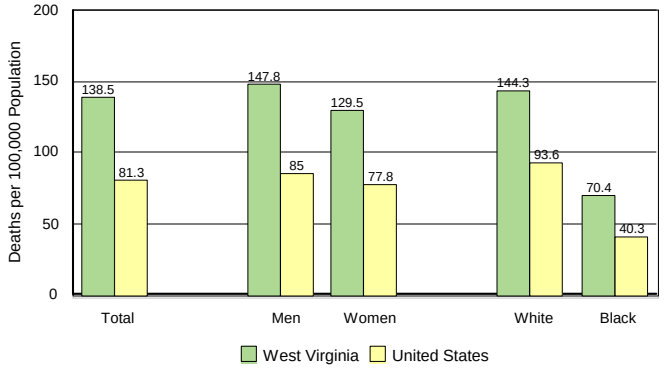
*Rates are age adjusted to the 2000 U.S. standard million.
Note: ICD-10 code J43

COPD-Related Mortality. The National Center for Health Statistics releases annual multiple-cause-of-death data that include decedent's age, sex, race, and state of residence; the underlying (primary) cause of death, and up to 19 contributing causes of death. This section examines COPD as a contributing cause (not underlying) of death in West Virginia and the United States in 2006.

The age-adjusted rate in West Virginia for deaths with COPD listed as a contributing cause was 138.5 deaths per 100,000 population, compared with 81.3 for the United States as a whole (Fig. 15). Excess West Virginia deaths were found among both men (a state rate of 147.8 vs. a U.S. rate of 85.0) and women (a state rate of 129.5 vs. a U.S. rate of 77.8). The rate among white residents was 144.3, compared with a national rate of 93.6. The rate among African American residents was 70.4, compared with a U.S. rate of 40.3.

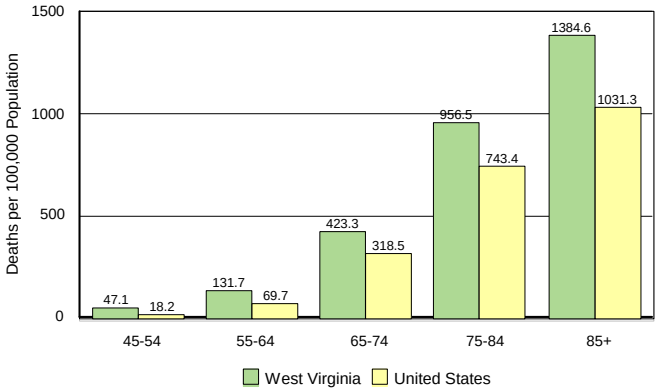
West Virginians of all ages were more likely to have COPD listed as a contributing cause on their death certificates than their peers nationwide (Figure 16). In terms of percentage differences between rates, the largest differences between state and national rates were found among 45-54 year olds (61.3% higher among West Virginians) and 55-64 year olds (47.1% higher).

Fig. 15. Rates* of COPD as a Contributing Cause of Death
By Gender and Race
West Virginia and United States, 2006



*Rates are age adjusted to the 2000 U.S. standard million.
Note: ICD-10 codes J40-44, J47

Fig. 16. Rates* of COPD as a Contributing Cause of Death by Age
West Virginia and United States, 2006



*Rates are age adjusted to the 2000 U.S. standard million.
Note: ICD-10 codes J40-J44, J47

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Appendix A COPD* Hospital Discharge Rates** by County West Virginia Inpatient Records, 2008					
County	**Rate**	**Rank**	**County**	**Rate**	**Rank**
Barbour	57.7	22	Monongalia	28.8	47
Berkeley	18.9	54	Monroe	24.0	49
Boone	73.3	15	Morgan	19.0	53
Braxton	44.9	37	Nicholas	59.3	18
Brooke	42.1	40	Ohio	51.0	31
Cabell	45.3	35	Pendleton	21.1	51
Calhoun	80.4	9	Pleasants	49.0	32
Clay	74.4	12	Pocahontas	55.5	25
Doddridge	19.4	52	Preston	44.9	36
Fayette	77.0	11	Putnam	33.7	43
Gilmer	82.9	8	Raleigh	97.0	3
Grant	34.9	42	Randolph	95.5	4
Greenbrier	24.9	48	Ritchie	58.2	21
Hampshire	30.6	45	Roane	47.5	33
Hancock	57.0	24	Summers	60.7	17
Hardy	31.6	44	Taylor	42.7	38
Harrison	30.1	46	Tucker	42.2	39
Jackson	59.0	19	Tyler	21.5	50
Jefferson	13.9	55	Upshur	51.3	30
Kanawha	46.5	34	Wayne	53.1	29
Lewis	126.7	2	Webster	53.2	28
Lincoln	64.3	16	Wetzel	58.8	20
Logan	90.6	5	Wirt	86.9	6
McDowell	54.6	27	Wood	55.4	26
Marion	74.0	13	Wyoming	77.3	10
Marshall	57.7	23	Total WV	52.8	
Mason	83.3	7			
Mercer	73.7	14			
Mineral	36.6	41			
Mingo	131.3	1			

*ICD-9 codes 490-492, 494-496

**Rates per 10,000 population

Ranked highest (1) to lowest (55)

Actual number of discharges available from the West Virginia Health Statistics Center

Appendix B COPD* Mortality Rates** by County West Virginia Residents, 2008					
County	**Rate**	**Rank**	**County**	**Rate**	**Rank**
Barbour	68.3	29	Monongalia	51.5	47
Berkeley	54.3	43	Monroe	91.3	8
Boone	115.1	3	Morgan	40.7	53
Braxton	63.5	37	Nicholas	55.7	42
Brooke	49.0	49	Ohio	80.9	12
Cabell	77.4	18	Pendleton	50.3	48
Calhoun	71.3	22	Pleasants	67.0	32
Clay	86.2	11	Pocahontas	86.2	10
Doddridge	102.6	4	Preston	54.3	44
Fayette	70.7	25	Putnam	68.0	30
Gilmer	92.1	6	Raleigh	73.8	19
Grant	34.7	54	Randolph	60.8	41
Greenbrier	63.8	36	Ritchie	95.7	5
Hampshire	45.1	51	Roane	90.7	9
Hancock	64.3	35	Summers	77.7	17
Hardy	34.6	55	Taylor	70.8	24
Harrison	61.2	39	Tucker	53.0	45
Jackson	70.4	26	Tyler	48.1	50
Jefferson	66.5	33	Upshur	71.7	20
Kanawha	61.2	40	Wayne	80.6	14
Lewis	43.1	52	Webster	71.1	23
Lincoln	51.7	46	Wetzel	78.8	15
Logan	80.6	13	Wirt	69.3	28
McDowell	119.7	2	Wood	63.5	38
Marion	71.3	21	Wyoming	91.5	7
Marshall	69.3	27	Total WV	68.5	
Mason	67.1	31			
Mercer	77.8	16			
Mineral	65.3	34			
Mingo	127.9	1			

*ICD-10 codes J40-J44, J47

**Rates per 100,000 population and age adjusted to the U.S. 2000 standard million

Ranked highest (1) to lowest (55)

Actual number of deaths available from the West Virginia Health Statistics Center

For more information about Health Statistics Center statistics and publications (hard copy and WEB versions), visit our website: http://www.wvdhhr.org/bph/hsc
or call (304) 558-9100