Arizona Comprehensive Lung Disease Control Plan
“The Arizona Department of Health Services initiated the development of this plan with the intent to set priorities by formulating objectives and suggesting strategies to address chronic obstructive pulmonary disease as a serious public health issue in Arizona.”
Dear Arizona Residents:

Chronic Obstructive Lung Disease (COPD) is a major health problem in Arizona and is the third leading cause of death in our state. In the United States, COPD is ranked the fourth leading cause of morbidity and mortality; the incidence of the disease is on the rise.

The Arizona Department of Health Services has developed the Arizona Comprehensive Lung Disease Control Plan. This plan sets the priorities, objectives, and strategies to reduce deaths attributable to COPD and to aid in reducing the adult population in Arizona who become disabled due to chronic lung disease. In addition, the plan promotes improvements in the system of care for individuals diagnosed with COPD, as well as promoting the prevention and early detection of lung disease.

The risk factors for COPD include genetic, physical wellness, behavioral, and environmental components: the greater being behavioral and environmental. It is estimated that 80% to 90% of COPD cases in the United States are attributable to tobacco smoking. This presents a tremendous challenge to the public health community in the areas of prevention and early detection given that smoking abstinence can prevent the occurrence of COPD, and smoking cessation has a powerful influence on determining the outcome for those who suffer from this disease.

The plan is designed to assist stakeholders, policymakers, health care professionals, educators, and public health workers in developing and coordinating approaches to address COPD among their constituents. The plan is an important step in raising awareness of the serious public health problem of COPD, and it provides a framework for action to reduce the impact of lung disease in Arizona.

Sincerely,

Susan Gerard
Director
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TRUST Commission
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Chronic Obstructive Pulmonary Disease (COPD), also known as Chronic Obstructive Lung Disease or Chronic Lower Respiratory Disease, is a major public health problem. It is projected to rank fifth in 2020 as a world-wide burden of disease according to the World Bank/World Health Organization.\textsuperscript{1} It is currently the fourth leading cause of morbidity and mortality in the United States,\textsuperscript{2} and in 2002, COPD was the third leading cause of death in Arizona.\textsuperscript{3} COPD is also the only major disease that is rising in prevalence and mortality while all other major causes of death are declining.\textsuperscript{4}

Although COPD is such a significant cause of mortality and morbidity worldwide, it has failed to receive commensurate attention from the healthcare community and government officials. This can be attributed to various factors. There is incomplete information about the causes and prevalence of COPD. Prevalence and mortality data greatly underestimate the total burden of COPD because the disease is usually not diagnosed until it is clinically apparent and moderately advanced.\textsuperscript{5} Mortality data also underestimate COPD as a cause of death because the disease is more likely to be cited as an underlying cause of death, or may not be cited at all, rather than being the attributed cause of death.\textsuperscript{6}

COPD is not characterized by the drama that surrounds sudden and life-threatening illness such as heart attack and stroke, which has brought the combined efforts of the public, governmental agencies, and the medical community to devise effective strategies for prevention and early treatment for these chronic diseases. By contrast, COPD lacks compelling symptoms or may be asymptomatic during years of progressive loss of lung function on the pathway to disabling dyspnea and premature mortality from acute and chronic respiratory failure.\textsuperscript{7} One of the most frightening aspects of this disease is the fact that many afflicted with COPD do not realize they have it until they have lost significant lung function, at which time mundane activities such as walking short distances can become difficult. Often the symptoms of COPD are attributed to a common cold, allergies,
lack of exercise, or aging and are therefore unrecognized by patients or underdiagnosed by physicians.

According to the American Lung Association, smoking causes 80 to 90% of COPD cases; and smokers are 10 times more likely than nonsmokers to die of the disease. The association of the disease with smoking has also attributed to its lack of importance, since until recently it was seen as a self-inflicted consequence of a bad habit, rather than the tragic outcome of an addiction.

There is also confusion around the term COPD among patients and healthcare professionals, which complicates epidemiologic studies of COPD. Patients may be diagnosed with smoker’s lung, emphysema, bronchitis, chronic bronchitis, chronic obstructive bronchitis or obstructive lung disease and not identify with the term COPD.

Self-report of physician diagnosis is a poor measure of COPD prevalence, as in reports of impaired lung function from the National Health and Nutrition Examination Survey (NHANES) III which carefully defined “obstructive” but included in the analysis those in whom the impairments may not have been “chronic” (e.g. viral infection), “pulmonary” (e.g. congestive heart failure), or “disease” (e.g. normal subjects with outlier laboratory values).

These factors have resulted in surprisingly little being known about the disease beyond its clinical nature. Studies of the disease burden on COPD patients are scarce, and the social and healthcare costs of the disease have not been well quantified. There are limited data about COPD symptoms and severity, disability or activity limitations, lifestyle impact, social and psychosocial consequences, healthcare utilization and current patterns of treatment.

The Arizona Department of Health Services initiated the development of this plan with the intent to set priorities by formulating objectives and suggesting strategies to address chronic obstructive pulmonary disease as a serious public health issue in Arizona. This plan begins with a detailed description of chronic obstructive lung disease followed by a discussion of proposed objectives and strategies. The overriding goals of this plan are those stated in the Healthy People 2010: Objectives for Improving Health. It is important that a comprehensive approach be taken in working toward these ambitious goals in Arizona, involving the collaboration of community stakeholders.

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**TABLE 1: Overriding Goals**

| Reduce the proportion of adults in Arizona whose activity is limited due to chronic lung disease. |
| Reduce deaths from COPD among Arizona adults. |
TABLE 2 Plan Objectives by Topic

**Epidemiology and Research**
1. Establish a surveillance system to accurately track the mortality and morbidity of COPD in Arizona, and also measure the impact on the economy of the state.
2. Support research into COPD etiology and clinical management, as well as healthcare policies and outcomes particularly as the activities relate to state issues.

**Treatment and Management**
3. Improve early detection and diagnosis of COPD.
4. Promote better care for patients with COPD in Arizona according to established guidelines.
5. Educate healthcare providers to manage patients with COPD to increase longevity and quality of life and reduce exacerbations of the disease.
7. Improve access to pulmonary rehabilitation programs for Arizonans with COPD in order to prevent and forestall premature morbidity and mortality.

**Patient Education and Quality of Life Issues**
8. Improve self-management knowledge and behavior in people with COPD, their families and other caregivers.
9. Provide social support for patients, families, and caregivers impacted by COPD.

**Prevention**
10. Promote healthy living practices, which provide the most effective method of preventing COPD (tobacco abstinence, periodic health checks, avoidance of unhealthy work environments).
11. Reduce exposure to environmental and occupational risk factors to prevent the onset and progression of COPD.

**Disparity**
12. Identify and eliminate disparities in COPD prevention, diagnosis, and management throughout the state.

**Collaborative Efforts**
13. Foster communication, collaboration and networking opportunities among patients, caregivers, healthcare professionals, public health officials and other stakeholders.

**Advocacy**
14. Advocate and support policies to reduce the prevalence of tobacco use and secondhand smoke exposure among Arizonans.

**Public Awareness**
15. Increase awareness of the medical community, public health officials and the general public that COPD is a serious public health problem in Arizona.
Definition:

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema. The airflow obstruction is generally progressive, accompanied by airway hyperreactivity, and may be partially reversible. In the past asthma was also classified as COPD, however it has been determined that the reversibility of asthma and the characteristic inflammation with participation of complex cellular and chemical mediators distinguishes it from these conditions and it has been separated. However, the obstruction in many patients with COPD may include a significant reversible component and some patients with asthma may go on to develop irreversible airflow obstruction indistinguishable from COPD.11

Chronic bronchitis is defined as the presence of chronic productive cough for three months in each of two successive years in a patient for whom other causes of chronic cough have been excluded.12

Chronic bronchitis is characterized by chronic cough and sputum production, intermittent wheezing with variable degrees of shortness of breath on exertion, recurring and continuing for months.

Chronic bronchitis results from inflammation and swelling of the cells, which line the bronchus. This inflammation causes the production of excessive mucus. Both the swelling and excess mucus contribute to the narrowing of the bronchi, making air exchange more difficult and increasing the risk of lung infections.

In chronic bronchitis, the mucous glands in the lungs become larger. The airways become inflamed, and the bronchial walls thicken. These changes and the loss of supporting alveolar (air space) attachments limit airflow by allowing the airway walls to deform and narrow the airway lumen (the inside of the airway tube).

Emphysema is defined as abnormal permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis. Destruction is
defined as lack of uniformity in the pattern of respiratory airspace enlargement; the orderly appearance of the acinus and its components is disturbed and may be lost.\textsuperscript{13} It is an abnormal, permanent enlargement of the air spaces (alveoli) located at the end of the breathing passages of the lungs (terminal bronchioles), accompanied by the damage to the walls between the air spaces in the lungs. This leaves less surface area for the normal exchange of oxygen and carbon dioxide.

Emphysema reduces the normal elasticity of the lung that helps to hold the airways open. As a result, the lungs lose their elasticity and exhalation becomes more and more difficult. Air remains trapped in the overinflated lungs. Those with emphysema experience progressive shortness of breath on exertion, variable degrees of coughing and wheezing, and irreversible airflow obstruction.

**Burden of COPD in Arizona**

According to the Arizona Department of Health Services Public Health Prevention Services, “Arizona Chronic Disease Surveillance Indicators Report,” September 2004, chronic lower respiratory disease (synonymous with chronic obstructive pulmonary disease) was the third leading cause of death in Arizona for 2002, and accounted for 6\% of all deaths. Hospital discharge data for that year reveals 13,638 hospitalizations for COPD, which is a rate of 249.2 per 100,000 population. Estimates of prevalence derived from the National Health Interview Survey (NHIS) conducted by the National Center for Health Statistics, Centers for Disease Control and Prevention in 2001 and based on Department of Economic Security population estimates for 2004 indicate that the total number of Arizonans with COPD is 290,124.\textsuperscript{14}

Figure 1 and Figure 2 from the Arizona Health Status and Vital Statistics Report of 2003 illustrates the burden of COPD (referred to as chronic lower respiratory diseases) in Arizona:\textsuperscript{15}

Figure 2 illustrates the burden as distributed among racial and ethnic groups in Arizona.\textsuperscript{16}

The number of deaths in Arizona from chronic lower respiratory diseases by county is illustrated in Figure 3.\textsuperscript{17}

There are significant problems in the accuracy of this data. Reporting on cause of death often will not indicate COPD as a cause of death, since it may be seen only as a contributing factor to death due to cardiopulmonary failure, pneumonia, or influenza, or it may not be listed at all. The same is true with hospitalization data, which in addition does not include emergency department or urgent care visits, nor does it include those Arizona residents receiving care in any Federal facility.

The NHIS data is dependent on the patient self-identifying that they have been diagnosed with either chronic bronchitis or emphysema. The question regarding emphysema asks whether the individual had “ever” been told by a doctor or other
**Twelve Leading Causes of Death Among Arizona Residents in 2003**

**BASED ON THE NUMBER OF DEATHS:**

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Number of Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Diseases of heart</td>
<td>10,649</td>
</tr>
<tr>
<td>2. Malignant neoplasms</td>
<td>9,451</td>
</tr>
<tr>
<td>3. Chronic lower respiratory diseases</td>
<td>2,522</td>
</tr>
<tr>
<td>4. Accidents (unintentional injuries)</td>
<td>2,466</td>
</tr>
<tr>
<td>5. Cerebrovascular disease</td>
<td>2,356</td>
</tr>
<tr>
<td>6. Alzheimer's disease</td>
<td>1,691</td>
</tr>
<tr>
<td>7. Influenza and pneumonia</td>
<td>1,248</td>
</tr>
<tr>
<td>8. Diabetes mellitus</td>
<td>1,124</td>
</tr>
<tr>
<td>9. Intentional self-harm (suicide)</td>
<td>807</td>
</tr>
<tr>
<td>10. Chronic liver disease and cirrhosis</td>
<td>625</td>
</tr>
<tr>
<td>11. Nephritis, nephrotic syndrome and nephrosis</td>
<td>550</td>
</tr>
<tr>
<td>12. Assault (homicide)</td>
<td>480</td>
</tr>
</tbody>
</table>

**BASED ON AGE-ADJUSTED* MORTALITY RATES:**

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Diseases of heart</td>
<td>197.1</td>
</tr>
<tr>
<td>2. Malignant neoplasms</td>
<td>169.4</td>
</tr>
<tr>
<td>3. Chronic lower respiratory diseases</td>
<td>45.4</td>
</tr>
<tr>
<td>4. Accidents (unintentional injuries)</td>
<td>44.8</td>
</tr>
<tr>
<td>5. Cerebrovascular disease</td>
<td>43.9</td>
</tr>
<tr>
<td>6. Alzheimer's disease</td>
<td>32.5</td>
</tr>
<tr>
<td>7. Influenza and pneumonia</td>
<td>23.4</td>
</tr>
<tr>
<td>8. Diabetes mellitus</td>
<td>20.3</td>
</tr>
<tr>
<td>9. Intentional self-harm (suicide)</td>
<td>14.6</td>
</tr>
<tr>
<td>10. Chronic liver disease and cirrhosis</td>
<td>11.5</td>
</tr>
<tr>
<td>11. Nephritis, nephrotic syndrome and nephrosis</td>
<td>10.1</td>
</tr>
<tr>
<td>12. Assault (homicide)</td>
<td>8.3</td>
</tr>
</tbody>
</table>

*Number of deaths per 100,000 population age-adjusted to the 2000 U.S. standard.
Note: the cause-of-death titles are according to the Tenth Revision of the International Classification of Diseases (ICD-10)
health professional that they had the condition. The chronic bronchitis question asks if they had been told by a doctor or health professional if they have had the condition in the past 12 months. As we see from the American Thoracic Society (ATS) definition of chronic bronchitis, it is diagnosed over two successive years, so a “yes” answer may represent confusion with a viral bronchial illness.

Unfortunately, since diagnosis of COPD usually takes place when it is well advanced due to late onset of symptoms, these self-reported numbers tend to be significantly lower than actual incidence.

Because of the limited data currently available for social and economic impact of COPD in Arizona, these indicators will be addressed on a national level.

**Burden of COPD in the United States**

COPD is a major cause of morbidity and mortality in the United States. An estimated 11.2 million adults have ever reported a physician diagnosis of COPD. A recent survey found that 24 million U.S. adults have some evidence of impaired lung function, indicating under diagnosis of COPD.

The economic toll on the U.S. is quite heavy. According to estimates made by the National Heart Lung and Blood Institute in 2004, the annual cost to the nation for COPD was $37.2 billion, including $20.9 billion in direct healthcare expenditures, $7.4 billion in indirect morbidity costs, and $8.9 billion in indirect mortality costs. In 2000, COPD was responsible for 8 million physician office and hospital outpatient visits, and 726,000 hospitalizations in the U.S.

**Figure 2** Age-Adjusted Mortality Rates for Chronic Lower Respiratory Diseases by Race/Ethnic Group, Arizona, 2003

![Age-Adjusted Mortality Rates for Chronic Lower Respiratory Diseases by Race/Ethnic Group, Arizona, 2003](image-url)
NUMBER OF DEATHS PER 100,000 POPULATION
(age-adjusted to 2000 standard)

ARIZONA RATE = 45.4
The impact of COPD to the working population is underestimated, due to the assumption that it is a disease of the elderly. According to Centers for Disease Control data published in 2002, 70% of COPD patients were below the age of 65. The disability caused by COPD is significant and is documented in a survey released by the American Lung Association in 2001, “Confronting C.O.P.D. in America.” It revealed that 51% of COPD patients say their condition limits their ability to work, limits them in normal physical exertion (70%), household chores (56%), social activities (53%), sleeping (50%) and family activities (46%).

Beyond the debilitation of the patient, the burden on the families of COPD patients is tremendous, particularly in the later stages of the disease, when mild exertion is difficult and the patient ceases to be ambulatory. This leads to more lost wages, and numerous lifestyle accommodations required on the part of the caregivers. The burden of COPD in the United States appears to be generally increasing across many demographic groups. Data is limited since it is self-reported, and does not include the large number of undiagnosed cases of COPD, or hospitalizations where COPD is not listed as the admitting diagnosis.

### Risk Factors for COPD

#### Tobacco Use

The primary risk factor for COPD is tobacco smoking, which accounts for an estimated 80 to 90% of the deaths caused by COPD. Male smokers are nearly 12 times as likely to die from COPD and female smokers 13 times as likely to die as the nonsmoking population. Smokers have higher death rates for chronic bronchitis and emphysema, as well as a higher prevalence of lung function abnormalities, respiratory symptoms, and all forms of chronic obstructive airway disease. Differences between smokers and nonsmokers increase in direct proportion to quantity of smoking. Age of starting, total pack-years, and current smoking status are predictive of COPD mortality. Approximately 15% of smokers develop clinically significant COPD.

Also significant in terms of risk for COPD and tobacco use is the finding that smoking during pregnancy is associated with low birth weight, and infants with low birth weight appear to have a greater risk of developing COPD.

Tobacco smoking is not the only cause of COPD, and in some parts of the world it may not even be the major cause due to the role of indoor air pollution and occupational exposure. The prevailing notion that COPD is largely self-inflicted is counterproductive for future research and disease interventions. It is crucial to take into account the fact that not all smokers develop clinically significant COPD. This points to additional factors that determine each individual’s susceptibility.
**Secondhand Smoke**

Smoking and probably passive smoke exposure in childhood compromises lung growth leading to diminished maximal lung function in young adulthood. These deficiencies may portend airway hyperreactivity in adult life. Passive smoke exposure is a risk factor for symptoms of cough and sputum production, and may account for some of the COPD that develops in nonsmokers. The World Health Organization (WHO) estimates that passive smoking is associated with a 10 to 43% increase in the risk of COPD in adults.

**Ambient Air Pollution**

The role of outdoor air pollution in causing COPD is unclear, and the relative effect of short-term, high peak exposures and long-term, low-level exposures is yet to be resolved. There is some evidence that particles found in polluted air will increase the total burden of inhaled particles, which may contribute to total risk. There are studies that have shown comparative increases in chronic respiratory symptoms and pulmonary function abnormalities among subjects living in communities with significant outdoor air pollution.

Indoor air pollution from biomass fuel has been established as a risk for the development of COPD. This exposure is seen in environments where biomass fuel is used for cooking and heating in poorly vented dwellings, resulting in high levels of indoor particulate matter.

**Occupational Inhalants**

The impact of occupational exposures in the development of COPD is not yet fully known. However, it has been established that occupational exposures to dust, chemicals and some vapors, irritants and fumes are important contributors, especially when the exposures are sufficiently intense or prolonged. Miners, firefighters, metal workers, grain handlers, cotton workers, paper mill workers, agricultural workers, construction workers, and others employed in occupations associated with prolonged exposure to dusts, fumes, or gases have been seen to develop significant airflow obstruction, reduced lung function and respiratory symptoms.

A study of 3,380 British coal miners showed that there was clinically significant respiratory dysfunction due to inhalation of coal dust, leading to COPD, which was independent of the contribution of smoking to incidence of disease among the subjects. Grain dust exposure has also been determined to be a risk for both smokers and nonsmokers.

These occupational exposures can cause COPD independently of tobacco smoking and increase the risk in the presence of concurrent tobacco smoking by increasing airway hyperresponsiveness. A recent study found that in the United States, 19% of COPD overall could be attributed to occupational hazard with 31% of COPD cases among never smokers being work related.
**Alpha1-antitrypsin Deficiency (AAT)**

This is the only genetic abnormality that is known to lead to COPD. Severe AAT deficiency leads to premature emphysema, often with chronic bronchitis and occasionally with bronchiectasis. It is estimated that this condition is responsible for less than 5% of the emphysema cases in the U.S. Alpha-1 related emphysema is caused by an inherited lack of a protective protein called alpha-1 antitrypsin (AAT). In normal and healthy individuals, AAT protects the lungs from a natural enzyme called neutrophil elastase. Neutrophil elastase is an enzyme that normally serves a useful purpose in lung tissue – it digests damaged or aging cells and bacteria in order to provide for healing. However, once it is done digesting those proteins, it does not stop, and attacks the lung tissue. Alpha-1-antitrypsin, in sufficient amounts, will trap and destroy the neutrophil elastase before it has a chance to begin damaging the delicate lung tissue. If allowed to progress, this form of emphysema becomes chronic and lung tissue continues to be destroyed; eventually it is fatal if the progress is not slowed down or halted.

It is estimated that there are 100,000 Americans today who were born with Alpha-1 deficiency. Alpha-1 related emphysema might afflict a majority of these individuals. However, AAT deficiency is often under diagnosed or misdiagnosed. As many as 3% of individuals with chronic obstructive pulmonary disease (COPD) may have undiagnosed Alpha-1 deficiency. Worldwide, it is estimated that 116 million people (25 million Americans) are carriers of the disease. WHO recommends that all individuals with COPD, and adults and adolescents with asthma (an estimated 20 million Americans) be tested for Alpha-1. A simple blood test can determine whether a person has low levels of the protective protein AAT. Also, a DNA-based cheek swab test has been recently developed to aid in diagnosis.

The onset of Alpha-1 related emphysema symptoms often appear between ages 32 and 41 years but may appear later. The early age at which the disease is present and the fact that the disease most frequently appears in the lower, rather than the upper, lung regions helps distinguish Alpha-1-related emphysema from other types of emphysema. Evidence shows that smoking significantly increases the risk and severity of emphysema in AAT deficient individuals and may decrease their life span by as much as 10 years.

**Hyperresponsive Airways (Asthma)**

Asthma, nonspecific airway hyperresponsive-ness and atopy (the genetic tendency to develop the classic allergic diseases – atopic dermatitis, allergic rhinitis or hay fever, and asthma) may possibly play a role in COPD. This relationship was originally proposed in 1960 by Orie and colleagues who were in the Netherlands, and thereby given the name “the Dutch hypothesis.” Evidence is steadily accumulating that this hyperresponsiveness is predictive of an accelerated rate of decline of lung function in smokers. How these trends are related to the
development of COPD is still unknown. Airway hyperresponsiveness may also develop after exposure to tobacco smoke or other environmental insults and thus may be a result of smoking-related airway disease.

**Infections**

There is an association of severe childhood infections with reduced lung function and increased respiratory symptoms in adulthood. This may be explained by an increased diagnosis of severe infections among children with underlying airway hyperresponsiveness, which as discussed above may itself be a risk factor. Also, viral infections may be related to another risk factor such as birth weight. The rate of infections in childhood is also directly related to the amount of exposure to secondhand smoke. Among adults, it has been established that HIV infection accelerates the onset of smoking-induced emphysema.³²

**Demographic Status**

Sex: Although the rates of COPD deaths have historically been higher in males than females, beginning in 2000, women in the United States have exceeded men in the number of deaths attributed to COPD. In 2002, over 61,000 females died compared to 59,000 males.³³ This has been attributed to the increasing use of tobacco among females, and some studies have suggested that women are more susceptible than men to the effects of tobacco smoke.³⁴

Race: COPD is, as of now, the only lung disease in which the white population has higher age-adjusted death rates than blacks. In 2002, the age-adjusted death rate in whites (44 per 100,000) was 1.6 times greater than the rate in blacks (28 per 100,000). The highest prevalence rates were in the over age 65 population for whites, and in the 45 to 64 year old age group for blacks. Black women had the lowest age-adjusted death rates with 19 per 100,000. Hispanics (male and female aggregated) had an age-adjusted mortality rate of 19 per 100,000, which is markedly lower than the other ethnic groups.³⁵

Socioeconomic Status: There is evidence that the risk of developing COPD is inversely related to socioeconomic status. One correlation that has been documented is the relationship to malnutrition. Because lower serum levels of the major antioxidant vitamins exist, the development of COPD may be more prevalent among those who do not have proper nutrition available. Oxidant injury is believed to be one of the mechanisms of alveolar and airway damage, so a diet deficient in antioxidants could be a risk factor. There is also an association with reduced food intake and levels of antioxidant vitamins in smokers as compared with nonsmokers linking the risk of smoking and COPD. Prevalence rates of tobacco use are also indirectly related to socioeconomic status throughout the world, as is the incidence of exposure to other inhaled pollutants such as biomass fuels.³⁶ The relationship between social position and respiratory mortality may also be attributed to housing conditions, exposure to occupational pollutants, and childhood exposure to indoor air pollution.
According to the U.S. Department of Health and Human Services, National Health Interview Survey of 2002, Table 3 substantiates the link between socioeconomic status and the incidence of COPD.

**Diagnosis and Staging**

Diagnosis of COPD should be considered in any patient who has any combination of the following:

- symptoms of cough
- sputum production
- dyspnea
- history of exposure to risk factors for the disease (especially smoking).

The diagnosis requires spirometry (a test of pulmonary function); post-bronchodilator (after the administration of an inhaled bronchodilator) FEV1/FVC <0.7 (a value comprised of the ratio of the forced expiratory volume in one second of expiration over the forced vital capacity) confirms the presence of airflow limitation that is not fully reversible.

Spirometry should be obtained in all persons with the following history:

- exposure to cigarettes and/or environmental or occupational pollutants

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Emphysema</th>
<th>Chronic Bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Education:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than H.S. diploma</td>
<td>3.5 (0.29)</td>
<td>6.3 (0.47)</td>
</tr>
<tr>
<td>H.S. diploma or G.E.D.</td>
<td>1.8 (0.16)</td>
<td>5.4 (0.29)</td>
</tr>
<tr>
<td>Some College</td>
<td>1.5 (0.16)</td>
<td>5.1 (0.28)</td>
</tr>
<tr>
<td>Bachelor's degree or higher</td>
<td>0.8 (0.13)</td>
<td>2.7 (0.23)</td>
</tr>
<tr>
<td><strong>Family Income:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than $20,000</td>
<td>3.1 (0.23)</td>
<td>6.9 (0.47)</td>
</tr>
<tr>
<td>$20,000-$34,999</td>
<td>1.7 (0.22)</td>
<td>5.6 (0.40)</td>
</tr>
<tr>
<td>$35,000-$54,999</td>
<td>1.5 (0.21)</td>
<td>4.4 (0.34)</td>
</tr>
<tr>
<td>$55,000-$74,999</td>
<td>1.2 (0.26)</td>
<td>4.9 (0.58)</td>
</tr>
<tr>
<td>$75,000 or more</td>
<td>0.7 (0.23)</td>
<td>2.9 (0.35)</td>
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<tr>
<td><strong>Poverty Status:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor</td>
<td>3.5 (0.36)</td>
<td>8.3 (0.64)</td>
</tr>
<tr>
<td>Near Poor</td>
<td>2.4 (0.25)</td>
<td>5.9 (0.41)</td>
</tr>
<tr>
<td>Not Poor</td>
<td>1.2 (0.10)</td>
<td>4.0 (0.18)</td>
</tr>
</tbody>
</table>
• family history of chronic respiratory illness
• presence of cough, sputum production or dyspnea.

**Spirometric Classification**

Spirometric classification has proved useful in predicting health status, utilization of healthcare resources, and development of exacerbation and mortality in COPD. It is intended to be applicable to populations and not to substitute clinical judgment in the evaluation of the severity of disease in individual patients.38

Spirometry is as important for the diagnosis of COPD as blood pressure measurements are for the diagnosis of hypertension. Testing with spirometry determines the presence and severity of the airway obstruction in COPD, by measuring how effectively, and how quickly, the lungs can be emptied. It is easy to administer, takes only a few minutes to complete, and is noninvasive. It can be conducted in the primary care physicians’ office with a spirometer, a relatively inexpensive device. Patients take a deep breath and exhale into the spirometer as hard and fast as they can for a minimum of six seconds. The spirometer is connected to a computer that records the volume of air exhaled in one second (FEV1) and the total amount of air exhaled in a forced maneuver (FVC). The FEV1/FVC ratio is the primary measurement in identifying an obstructive impairment of the airways, and is also used to monitor the progression of COPD.39

It is accepted that a single measurement of FEV1 incompletely represents the complex clinical consequences of COPD because: 1) many patients

<table>
<thead>
<tr>
<th>Severity</th>
<th>Postbronchodilator FEV1/FVC</th>
<th>FEV1 % predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>At risk</td>
<td>&gt;0.7</td>
<td>≥ 80</td>
</tr>
<tr>
<td>Patients who:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>smoke or have</td>
<td></td>
<td></td>
</tr>
<tr>
<td>exposure to</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pollutants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>have cough,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sputum or dyspnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>have family history of respiratory disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild COPD</td>
<td>≤0.7</td>
<td>≥ 80</td>
</tr>
<tr>
<td>Moderate COPD</td>
<td>≤0.7</td>
<td>50-80</td>
</tr>
<tr>
<td>Severe COPD</td>
<td>≤0.7</td>
<td>30-50</td>
</tr>
<tr>
<td>Very severe COPD</td>
<td>≤0.7</td>
<td>&lt;30</td>
</tr>
</tbody>
</table>

FEV1: forced expiratory volume in one second; FVC: forced vital capacity.
are practically asymptomatic; 2) persistent cough and sputum production often precede the development of airflow limitation and, in others, the first symptom may be the development of dyspnea with previously tolerated activities; and 3) in the clinical course of the disease, systemic consequences, such as weight loss and peripheral muscle wasting and dysfunction, may develop.

Due to these and other factors, a staging system that could offer a composite picture of disease severity is highly desirable, although it is currently unavailable. However, spirometric classification is useful in predicting outcomes such as health status and mortality, and should be evaluated.

In addition to the FEV1, the Body Mass Index (BMI) and level of dyspnea have proved useful in predicting outcomes such as survival and it is recommended that they be evaluated.

BMI is easily obtained by dividing the weight (in kg) over the height (in m²). Values <21 kg/m² are associated with increased mortality.

Functional dyspnea can be assessed by the Medical Research Council dyspnea scale:

0: not troubled with breathlessness except with strenuous exercise.
1: troubled by shortness of breath when hurrying or walking up a slight hill.
2: walks slower than people of the same age due to breathlessness or has to stop for breath when walking at own pace on the level.
3: stops for breath after walking ~ 100m or after a few minutes on the level.
4: too breathless to leave the house or breathless when dressing or undressing.40

**Treatment/Management**

An effective treatment program for COPD includes these four components of care: (1) assess and monitor disease, (2) reduce risk factors, (3) manage stable COPD by both pharmacologic and non-pharmacologic interventions, and (4) manage acute exacerbations. Spirometry is a key tool in the assessment and monitoring of disease as is continuous appraisal by healthcare providers of patients’ symptoms and health status. The other three components are addressed in the following sections.

**Tobacco Smoking Cessation**

Since smoking is the predominant risk factor for COPD, and the majority of patients are smokers when they are diagnosed, the first line of treatment for the disease is smoking cessation. Cessation of smoking is the single most effective and cost effective intervention to retard the progression of COPD, and improve the quality of life and activity level of the patient.

Cigarette smoking is an addiction and a chronic relapsing disorder regarded as a primary disorder by the United States Department of Health and Human Services Guidelines and by the WHO. Therefore, treating tobacco use and dependence should be regarded as a primary and specific intervention.
Introduction

Preventing the development and progression of COPD can be regarded as one of the secondary effects prevented by treating the primary disorder, because although cigarette smoking is the single most important cause of COPD, it is also a major risk factor for many other diseases including atherosclerotic vascular disease, cancer, peptic ulcer and osteoporosis.

Smokers experience an accelerated rate of decline in lung function. Individual susceptibility, however, varies greatly and depends on a complex interaction of many genetic and environmental factors. It is often stated that 15% of smokers will develop COPD. This dramatically underestimates the impact of smoking because the majority of smokers will develop loss of lung function, and reduced lung function, at any level, is predictive of increased mortality.

Many smokers with undiagnosed COPD have symptoms. It is necessary to identify and properly diagnose individuals earlier in the course of the disease when physiological limitation and symptoms are milder. Quitting smoking can slow the progressive loss of lung function and can reduce symptoms at any point in time. Yet, the beneficial impact of smoking cessation on the natural history of COPD is greatest the earlier cessation is achieved. Adolescents who quit smoking will have increased lung growth.41

Pharmacologic Interventions

Effective medications for COPD are available and all patients who are symptomatic merit a trial of drug treatment. Therapy with currently available medications can reduce or abolish symptoms, increase exercise capacity, reduce the number and severity of exacerbations, and improve health status. Unfortunately, at present no drug treatment has been shown to modify the rate of decline of lung function.

The inhaled route is preferred when both inhaled and oral formulations are available. Smaller doses of active treatment can be delivered directly with equal or greater efficacy and with fewer side effects when administered by inhalation.

Patients must be educated in the correct use of whatever inhalation device is employed. Significant numbers of patients cannot effectively coordinate their breathing with a metered dose inhaler (MDI) but can use a breath-activated inhaler, a dry powder inhaler (DPI) device or a spacer chamber. The latter may be useful when inhaled corticosteroids are administered, as it reduces the oropharyngeal deposition and subsequent local side effects associated with these drugs.

Medications of different classes have been found to be useful in treating COPD and can be used in combination. The overall approach to managing stable COPD involves a stepwise increase in treatment, depending on the severity of the disease.

Bronchodilators are a class of medications that relax the muscles around the bronchi to allow easier breathing. They are typically indicated for the relief
of bronchospasm, which are contractions of the smooth muscle in the walls of the bronchi and bronchioles that cause the airways to constrict or narrow. Anticholinergic bronchodilators fall into this class of COPD medications, as do short-acting beta2-agonists, long-acting beta2-agonists, methylxanthines (e.g., theophylline), and a combination of an anticholinergic bronchodilator and a short-acting beta2-agonist.

All major guidelines for COPD management recommend beginning treatment with aerosol bronchodilators, which are inhaled directly into the lungs and have few side effects.

In response to irritants such as cigarette smoke, the body produces a chemical “messenger” called acetylcholine that induces the airways to constrict. Anticholinergic bronchodilators are the only medications that act by blocking acetylcholine, thereby relaxing the muscle tissue and keeping the airways open. Anticholinergic medications work via part of the parasympathetic nervous system, which controls airway size. In addition to helping COPD patients take fuller breaths, maintenance use of anticholinergic medication may also help lower the incidence of acute exacerbations in COPD patients.

Anticholinergics are most often administered through metered-dose inhalers, or “puffers,” as they are commonly called. The effects of the medication generally last from four to six hours, so physicians typically prescribe use four times a day. Inhaled anticholinergics are minimally absorbed, resulting in relatively few side effects. Some common side effects of ipratropium bromide, an inhaled anticholinergic therapy, include cough and nervousness.

Anticholinergic bronchodilators, as a class, are the number one prescribed bronchodilator used in the treatment of COPD. Currently, the leading anticholinergic medication prescribed by physicians is ipratropium bromide. It is sold alone under the brand name ATROVENT, Inhalation Aerosol or in combination with albuterol sulfate under the brand name COMBIVENT, Inhalation Aerosol.

Beta2-agonists work via part of the nervous system that controls muscle tissue around the airways. They work by stimulating receptors in the sympathetic nervous system, leading to dilation of air passages. Two types of beta2-agonists are available: short-acting beta-agonists and long-acting beta-agonists.

Short-acting beta2-agonists are recommended for patients with COPD who experience intermittent symptoms. They are also used as a “rescue” medication to fend off an impending attack of shortness of breath. Short-acting beta2-agonists are typically prescribed along with anticholinergics to open up the airways of COPD patients with continuing symptoms. The short-acting beta2-agonist most commonly prescribed by physicians is albuterol. In clinical studies, the most common side effects of albuterol included tremor, nausea, tachycardia, palpitations and nervousness.

Long-acting beta-agonists are bronchodilators that are taken twice a day and, like short-acting beta-agonists, work via part of the nervous system that controls muscle tissue around the airways.
Long-acting beta-agonists are often prescribed for nighttime breathing problems because they provide up to 12 hours of relief. Patients using long-acting beta-agonists need to be reminded to continue using their short-acting beta-agonist for “rescue” therapy, because long-acting beta-agonists do not work as quickly and are indicated for use only twice a day. The most common side effects seen with use of long-acting beta-agonists by patients with COPD include headache, upper respiratory tract infection, nasopharyngitis and cough.

Combination bronchodilators are the combination of an anticholinergic and short-acting beta2-agonist, and work via the part of the nervous system that controls airway size, as well as the part that controls muscle tissue around the airways. Increased efficacy is seen with this combination agent over the individual components, without an increase in side effects. The most common side effects include bronchitis, upper respiratory tract infection and headache.

Another bronchodilator used in the treatment of COPD is theophylline, which is taken orally. Theophylline affects many parts of the body, including muscle tissue and the heart. It works by opening up the airways, increasing muscle endurance, and decreasing muscle fatigue. At one time, theophylline was the most widely prescribed COPD medication, but it has lost favor because of side effects. However, theophylline may have benefits that go beyond bronchodilation, and it is still an important part of COPD management.

Theophylline is taken orally once or twice a day, so it may be particularly valuable for noncompliant patients who cannot optimally use aerosol therapy. The dosage should be adjusted to reach a therapeutic serum level, so blood levels should be monitored. However, some patients experience side effects even at low serum levels. The most common side effects seen are nausea, vomiting, headache and insomnia.

Currently, inhaled corticosteroids are not indicated for the treatment of COPD. They are the cornerstone of asthma therapy, but have a limited role in the maintenance of lung function in patients with COPD. Only about 10% of patients with COPD show a significant improvement in lung function when treated with corticosteroids. The reason is that different mediators cause inflammation in asthma and COPD. The mediators that cause inflammation in COPD have only limited responsiveness to corticosteroids, while those mediators responsible for inflammation in asthma are dramatically affected by inhaled corticosteroids.

Surveys of clinicians' prescribing habits, however, have shown little difference in the use of inhaled corticosteroids for asthma patients and for COPD patients. Guidelines for the treatment of COPD suggest that because inhaled corticosteroids play only a minor role in the maintenance treatment of COPD and may produce systemic side effects, they should be reserved for patients whose symptoms are not optimally controlled with
bronchodilators. This subgroup of patients should receive inhaled or oral corticosteroids for a trial period. If a significant objective clinical response is not achieved, corticosteroids should be discontinued. When a benefit is observed with oral corticosteroids, the dose should be tapered to the lowest possible dose. At that point, a trial of an inhaled corticosteroid should be initiated. The most common side effects of inhaled corticosteroids include upper respiratory infection, headache and pharyngitis.

Antibiotics may be given to patients with COPD for acute bacterial infections of the respiratory tract, including sinusitis, acute bronchitis and some types of pneumonia. Antibiotics are also used to treat exacerbations when symptoms of infection are present, such as fever, increased cough and sputum changes.42

COPD patients are at high risk for respiratory tract infections; therefore prophylaxis via vaccination is recommended for this population. Vaccination can reduce the incidence and severity of bronchial infections with which COPD morbidity and mortality is closely associated. Annual vaccine against influenza, with vaccine formulation and potency revised yearly is essential. Amantadine and similar drugs can be used for prophylaxis during high-risk periods for influenza for patients who either have not been vaccinated in time or for patients who have had prior allergic reactions to the vaccine or are allergic to eggs.

Although there is debate about its benefits, the pneumococcal vaccine is also currently recommended for all COPD patients. This vaccine is not given annually, and there is concern about the rate of decline in immune response and immune reactions in immunocompetent patients.43

Long term oxygen therapy (LTOT) is used to treat patients with chronic hypoxemia (low levels of oxygen being carried by the blood). It can improve survival, exercise tolerance, sleep efficiency, and cognitive performance in these patients. The use of LTOT can be continuous, with activity only, or during sleep, depending on the needs of the patient. There are established guidelines for prescribing oxygen for patients. These guidelines are based on the values of arterial blood gases or pulse oximetry testing at rest and with activity (if tolerated by the patient). All prescriptions for oxygen should include the source of oxygen (gas, liquid or concentrator), the method of delivery, duration of use, and specific flow rates for rest, exercise and sleep.44

**Non-pharmacological Interventions**

These include pulmonary rehabilitation services, which may require long-term adherence by the patient from diagnosis throughout the course of their disease, and surgical options, which are not widely used, but have had some beneficial outcomes in select patients.

Pulmonary rehabilitation is a multidisciplinary program of care that is individually tailored and designed to optimize the COPD patient’s physical and social performance and autonomy.
Comprehensive pulmonary rehabilitation generally includes exercise training (endurance and strength), education (promoting disease awareness and self-management), psychosocial and behavioral intervention (support groups and referral to behavioral health services), nutritional therapy and outcome assessment. Smoking cessation intervention is an obviously important component of the pulmonary rehabilitation process for smokers.

Pulmonary rehabilitation is usually coordinated by an experienced healthcare professional, such as a registered nurse, physical therapist or respiratory therapist. Also involved is a multidisciplinary team that varies between programs, but often includes physicians, nurses, respiratory therapists, physical therapists, occupational therapists, psychologists, dieticians and social workers.

Pulmonary rehabilitation results in improvements in multiple outcome areas of considerable importance to the patient, including dyspnea, exercise ability, health status and healthcare utilization. These positive effects occur despite the fact that it has a minimal effect on static pulmonary function measurements. This reflects the fact that much of the morbidity from COPD results from secondary conditions, which are often treatable if recognized. Examples of these treatable conditions are cardiac deconditioning, peripheral muscle dysfunction, and a reduction in total and lean body mass anxiety, and poor coping skills.45

Surgical interventions for COPD are limited to those few selected patients who might benefit from the procedure, are healthy enough to withstand the trauma of the surgery, and who will be compliant with and able to tolerate aftercare. These surgeries include bullectomy, lung volume reduction surgery, and lung transplantation, which may result in improved spirometry, lung volume, exercise capacity, dyspnea, health-related quality of life and possibly survival in highly selected patients.

- **Bullectomy**: This is a procedure in which a large bulla, which does not contribute to gas exchange, is removed to alleviate local symptoms such as hemoptysis, infection, or chest pain, and to allow re-expansion of the compressed lung region. It can be done thoracoscopically.

- **Lung Volume Reduction Surgery**: This is a surgery in which parts of the lung are resected to reduce hyperinflation. This results in making respiratory muscles more effective by improving their mechanical efficiency. This procedure also increases the elastic recoil pressure of the lung and thus improves expiratory flow rates.

- **Lung Transplantation**: This is a highly limited procedure due to cost, availability of donor organs, and selectivity of appropriate candidates. There is also considerable question about the long-term survival benefits, although it has been shown to increase functional capacity and improve quality of life.46
Exacerbations

An exacerbation of COPD is an event that changes the natural course of the disease so that symptoms become more severe, and a change in management of the disease is warranted. The causes of exacerbations may be infectious or non-infectious, and in some instances, the reason for worsening of symptoms remains unknown. Severity of exacerbations varies, and prognosis often depends on the stage of advancement of the disease and the existence of comorbid conditions. Common comorbid conditions associated with poor prognosis in exacerbations are congestive heart failure, coronary artery disease, diabetes mellitus, renal and liver failure. Most often, exacerbations can be managed by primary care physicians on an outpatient basis. Corticosteroids and antibiotics are used widely in the management of COPD exacerbations. It is essential that patients understand how to monitor their symptoms and detect when there is a change, so that they can immediately seek medical attention.

In some instances, exacerbations can be so severe that hospitalization is required, even including non-invasive or invasive mechanical ventilation. There are times when a patient cannot be removed from ventilation after an exacerbation and becomes ventilator dependent. Some patients who are dependent on mechanical or assisted ventilation can be discharged from the hospital on ventilators and can continue to be managed at home. This requires coordinated support from caregivers and health professionals, but is less costly than hospitalization or extended care facilities, and can provide an improved quality of life for patient and family.

When COPD patients experience an exacerbation, causing them to interact with healthcare professionals and institutions, it can be seen as an opportunity for evaluation, education, and follow-up. Unfortunately, approximately one third of patients with acute exacerbations have recurrent symptoms within 14 days, and 17% relapse and require hospitalization. Identifying those patients at risk for relapse and providing adequate follow-up can reduce these rates. Efficacy of disease treatment and management of the COPD patient with an exacerbation should be measured not just by recovery from exacerbations, but also by exacerbation-free intervals, resource utilization, and improved quality of life.
Objectives

Epidemiology and Research

Objectives

1. Establish a surveillance system to accurately track the mortality and morbidity of COPD in Arizona, and also measure the impact on the economy of the state.

2. Support research into COPD etiology and clinical management, as well as healthcare policies and outcomes particularly as the activities relate to state issues.

Rationale

It is essential that COPD prevalence be assessed and monitored as a foundation for managing the disease. Disease surveillance is difficult and expensive, but in regard to COPD, accurate epidemiological data is particularly elusive. Prevalence and morbidity data, when available, greatly underestimate the total burden of COPD because the diagnosis is usually delayed until it is clinically apparent and moderately advanced.

The term Chronic Obstructive Pulmonary Disease is imprecise and variably defined, and is not often used in health surveys. The diseases under this umbrella term may or may not include asthma, and chronic bronchitis is often confused with acute bronchitis. Other chronic obstructive lung diseases such as obliterative bronchiolitis and cystic fibrosis are excluded since they have known etiology or specific pathology. Other nomenclature for COPD as defined in this paper include Chronic Obstructive Lung Disease, or Chronic Lower Respiratory Disease, which could include diseases other than emphysema and chronic bronchitis.

Mortality data for COPD is also underestimated because it is more likely to be cited as a contributory rather than as an underlying cause of death if it is cited at all. This is also a problem with hospitalization data.

Research progress in the field of COPD has been slow and mostly focused on the association of COPD with cigarette smoking. There has not yet been significant research leading to a reduction in COPD prevalence or morbidity, to the development of any therapy proven to modify the disease process itself, or to an adequate understanding of how risk factors other than cigarette smoking may contribute to COPD.
Understanding why only certain smokers develop COPD not only illuminates the mechanisms of the development of the disease, but also might allow for the targeting of intensive smoking interventions to individuals at highest risk while enhancing the effectiveness of these interventions. There are also variations in the rate of decline in lung function among individuals with COPD that suggests intrinsic or environmental factors influencing disease course, which may differ from those determining susceptibility to disease. Recommended objectives for future research include description of the disease process, disease pathogenesis, advanced therapeutic modalities and clinical studies to validate or revise current clinical practices.51

**Objective**

1. Establish a surveillance system to accurately track the mortality and morbidity of COPD in Arizona, and also measure the impact on the economy of the state.

**Strategies**

- Develop surveys to collect data not available through existing data sources.

- Use existing and new data sources to assess (by demographic, geographic, and socioeconomic variables):
  
  a. COPD disease severity (e.g. hospitalization rates, mortality rates, emergency department visit rates).
  
  b. COPD management and treatment patterns (e.g. prescription utilization patterns).

- Cost of COPD (e.g. direct and indirect costs including AHCCCS, medical care billing and pharmacy costs).

- Prevalence of exacerbations.

- Disparities in access to healthcare for COPD in target populations.

- Quality of life for people with COPD and their families.

- Establish a standardized case definition of COPD, and establish criteria for the measurement of COPD morbidity and mortality.

- Collect accurate, timely data on prevalence, morbidity, mortality, and cost statistics for COPD.

- Create an infrastructure for emergency department and hospitalization data to be obtained from all medical facilities statewide including federal and tribal and Bureau of Indian Affairs institutions.

- Collect data and do analysis on the effect of COPD on work ability, efficiency, and job choice, as well as the effect of occupational exposures on COPD risk.

- Ensure adequate resources to develop and/or maintain new and/or existing surveillance systems.

- Disseminate reports based on acquired data to community stakeholders.
Objective

2. Support research into COPD etiology and clinical management, as well as healthcare policies and outcomes particularly as the activities relate to state issues.

Strategies

- Improve data sources to increase understanding of COPD risk factors and evaluate effectiveness of interventions.
- Encourage basic and clinical research to develop new and effective disease modifying therapies that will decrease the loss of lung function, restore lung function, and lengthen lifespan.
- Identify intermediate end-points and biomarkers for the effective and efficient translation of basic research findings to clinically relevant outcomes.
- Increase research related to occupational and environmental causes and contributors to COPD.
- Foster research that addresses the risk and resiliency of smokers in the development of COPD and the variation in susceptibility to COPD and disease progression that distinguishes the 10 to 20% of smokers of one pack per day or more who eventually develop clinically significant airflow limitation from other tobacco smokers who never develop airway dysfunction.
- Encourage research funding to develop and evaluate the role of chronic disease management programs for COPD.
- Perform research studies to determine the necessary elements and best timing for pulmonary rehabilitation, including cost/benefit analyses.
- Increase participation in clinical trials through education of patients.

Treatment and Management

Objectives

3. Improve early detection and diagnosis of COPD.
4. Promote better care for patients with COPD in Arizona according to established guidelines.
5. Educate healthcare providers to manage patients with COPD to increase longevity and quality of life and reduce exacerbations of the disease.
7. Improve access to pulmonary rehabilitation programs for Arizonans with COPD in order to prevent and forestall premature morbidity and mortality.

Rationale

Currently in the U.S., mortality due to chronic obstructive pulmonary disease is ranked number four and is projected to rise to number three by 2020. The number of patients with physician diagnosed COPD is estimated at 10 million, the number of people with abnormal lung function is estimated at 24 million. The underdiagnosis of lung disease in the U.S. may approach 14 million.52
Healthy People 2010 describes the following opportunities to help meet its objectives for improving health for individuals with COPD:

“Primary care physicians are in a key position to provide optimal care to patients with COPD and to provide counseling during clinical or health center visits to patients who smoke. Effective tests are available to screen patients for COPD, and primary care physicians need to be trained in the latest methods to detect and treat the disease.”

Since COPD can remain asymptomatic for 20 to 30 years before clinical symptoms appear, and by the time it is diagnosed, 75% of lung function has been lost, it is essential that early diagnosis and intervention occur in those people at risk for COPD. Early diagnosis coupled with smoking cessation can improve lung function and retard the rate of decline and deterioration of quality of life. Preliminary studies have indicated that knowledge of abnormal lung function can be a powerful motivator in smoking cessation and that smokers who are tested who have normal lung function may be motivated by relief that smoking has not yet damaged their lungs. Not only COPD, but also risk of lung cancer, heart attack and stroke is predicted by abnormal spirometry results. The spirometer is a tool for all four of these most common causes of death in the U.S. and the world. The National Lung Health Education Program is promoting patients to “Test Your Lungs, Know Your Numbers,” so that spirometric values are as well known as blood type, blood pressure and cholesterol levels, and they are working to educate providers and demystify spirometry, to foster utilization by healthcare professionals.53

**Objective**

3. Improve early detection and diagnosis of COPD.

**Strategies**

- Spirometry capability should be available to all healthcare professionals.
- Develop a practical and feasible list of indications for spirometry and an easily understandable algorithm for interpretation and action.
- Encourage primary care providers to perform an office spirometry test for all patients over 45 years old who report smoking tobacco, or anyone of any age who has one of the cardinal symptoms of COPD: chronic cough, excess mucus (sputum) production, dyspnea on mild exertion out of proportion to age, or wheezing.
- Develop a consensus for severity assessment criteria, and promote consistent utilization of criteria by healthcare providers.

**Objective**

4. Promote better care for patients with COPD in Arizona according to established guidelines.

**Strategies**

- Establish a community standard for COPD including consensus on clinical guidelines to be adopted (i.e. American Thoracic Society, “Standards for the Diagnosis and Care of Patients with Chronic Obstructive Pulmonary Disease”).
- Assess current healthcare provider knowledge of established standard clinical guidelines.
Objectives

5. Educate healthcare providers to manage patients with COPD to increase longevity and quality of life and reduce exacerbations of the disease.

Strategies

- Implement physician education programs that impact behavior change such as reminder systems, standing orders, clinical pathways or protocols, opinion leaders and physician champions, as well as self-monitoring and feedback.
- Conduct provider education utilizing interactive techniques on diagnosis and management of COPD.
- Create an incentive program to motivate healthcare providers to participate in educational sessions. Develop interdisciplinary models of COPD care.
- Work with healthcare providers to develop models for chronic disease management that are applicable to COPD.
- Develop or adopt a COPD education program to certify health educators in Arizona.
- Partner with non-profit agencies and community organizations to facilitate provider education.
- Promote the use of COPD management plans as part of comprehensive COPD education programs for providers.

Objective


Strategies

- Encourage providers to promote healthy living practices to patients such as tobacco cessation, good nutrition, physical activities and regular vaccinations against influenza and pneumonia.
- Create incentives for healthcare professionals to counsel their patients in healthy lifestyle choices, and COPD self-management.
- Educate providers to develop and utilize COPD disease management plans to enable their patients to avoid exacerbations of their disease and/or minimize the severity of these exacerbations.
- Coordinate efforts with health plans, medical providers, government and non-government agencies, families, and other stakeholders to promote, develop, use and disseminate COPD disease management plans for all patients with COPD.

Objective

7. Improve access to pulmonary rehabilitation programs for Arizonans with COPD in order to prevent and forestall premature morbidity and mortality.
**Strategies**

- Increase awareness of the effectiveness of pulmonary rehabilitation among patients with COPD, physicians (both primary care and specialists), and policy makers, including AHCCCS (Arizona Healthcare Cost Containment System).

- Address issue of adequate reimbursement for pulmonary rehabilitation services.

**Patient Education and Quality of Life Issues**

**Objectives**

8. Improve self-management knowledge and behavior in people with COPD, their families and other caregivers.

9. Provide social support for patients, families, and caregivers impacted by COPD.

**Rationale**

Education about COPD and methods for treatment and management are critical in enabling patients to understand the disease process and provide them with the knowledge they need to make informed decisions. An effective disease management program that balances nutrition, exercise and behavior modification can help patients cope with symptoms, avoid exacerbations and comorbid conditions and focus on a positive program to prolong quality of life, keeping them active and productive for as long as possible. The families of COPD patients absorb a tremendous burden, particularly in the later stages of the disease, when mild exertion is difficult and the patient ceases to be ambulatory. Support and education for the patient and family can improve quality of life, but also reduces the direct and indirect costs to our society.

**Objective**

8. Improve self-management knowledge and behavior in people with COPD, their families and other caregivers.

**Strategies**

- Develop or promote models for chronic disease management applicable to COPD.

- Institutionalize the use of COPD disease management plans modeled after asthma management plans.

- Encourage improved patient/provider relationships to enhance COPD education and self-management skills.

- Develop or identify a clearinghouse of COPD related information and resources which are available on the Internet.

- Work with community organizations to make COPD education materials available statewide.

**Objective**

9. Provide social support for patients, families, and caregivers impacted by COPD.

**Strategies**

- Establish and sustain COPD support groups for patients, families and caregivers.
• Evaluate home care and monitoring in relation to utilization of healthcare resources including physician offices, emergency departments, skilled nursing facilities, and acute care facilities.

• Include COPD management in workplace wellness and disease management programs.

Prevention

Objectives

10. Promote healthy living practices, which provide the most effective method of preventing COPD (tobacco abstinence, periodic health checks, avoidance of unhealthy work environments).

11. Reduce exposure to environmental and occupational risk factors to prevent the onset and progression of COPD.

Rationale

Since 80 to 90% of COPD cases occur in tobacco users, obviously abstinence from tobacco use would vastly decrease morbidity and mortality for the disease. In addition, early detection of airway hyperreactivity by healthcare providers during routine health checks can be a tool in motivating tobacco users to quit, thereby preventing the development of COPD and the subsequent progression of the disease.

Tobacco addiction is complex and the fact that the devastating health effects caused by tobacco use and secondhand smoke exposure on average take decades to appear greatly contributes to the limited perceived risk of tobacco use. A long-term comprehensive tobacco control program which focuses on preventing the initiation of tobacco use among youth and young adults, promoting quitting, and reducing exposure to secondhand smoke is essential to reduce the disability and death related to COPD.

Environmental exposure to certain inhalants is another cause of COPD. Management of controllable exposure such as workplace regulations of occupational inhalants and policies protecting the public from environmental tobacco smoke can reduce potential risk. When environmental exposure is coupled with tobacco use, the total load of inhaled irritants increases the risk of developing the disease exponentially.

Objective

10. Promote healthy living practices, which provide the most effective method of preventing COPD (tobacco abstinence, periodic health checks, avoidance of unhealthy work environments).

Strategies

• Encourage campaigns to emphasize the impact of smoking cessation.

• Develop creative approaches to alert young people to the potential of COPD and its causes, especially tobacco use and secondhand smoke.

• Increase resources devoted to COPD prevention in the workplace, targeting interventions, and evaluating the effectiveness of those interventions.
• Encourage or require third party payers to support treatments for smoking cessation as a component of COPD treatment.

**Objective**

11. Reduce exposure to environmental and occupational risk factors to prevent the onset and progression of COPD.

**Strategies**

- Partner with state and federal regulatory agencies to manage environmental and occupational risk factors.
- Educate patients and caregivers about those environmental and occupational inhalants that exacerbate their disease and the avoidance of these agents.
- Promote public awareness of environmental factors that cause and contribute to COPD.
- Disseminate educational materials on occupational risk factors and triggers to employers, employees, healthcare providers and health insurance plans.
- Promote clean indoor air quality management plans for all public buildings.
- Promote awareness of the effects of secondhand smoke on COPD patients.

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**Disparity**

**Objective**

12. Identify and eliminate disparities in COPD prevention, diagnosis, and management throughout the state.

**Rationale**

The definition of a disparate population group in relationship to COPD encompasses those at highest risk to develop COPD, as well as those with COPD who do not have ready access to diagnosis, treatment and management, thereby causing them to bear a disproportionate burden of morbidity and mortality. Since we have established that the incidence of COPD is highly related to tobacco use, there is value in defining those groups at highest risk for COPD similarly to those at highest risk for tobacco related health disparities. Therefore, the ADHS/TEPP strategic plan identification of populations at highest risk for tobacco related health disparities is useful as stated: “Disparities (i.e., measurable differences) derive from ingrained patterns of resource allocation (e.g., services, funding, staffing, equipment, community support, etc.) and prioritization that contribute to modes of inequity and social injustice. Among the various Arizona communities, tobacco-related disparities may be characterized by race (a class or kind of people unified by community of interests, habits, or characteristics – African American, Asian, Caucasian, Hispanic), ethnicity (of or relating to large groups of people classed according to common racial, national, tribal, religious,
linguistic, or cultural origin or background – African American, specific to Asian region, multi-Latin American Descent, multi-European Descent), gender (male, female, possibly transgender), age, income/class (what one makes does not always reflect community status), health status, education, sexual orientation, or geographic location, among others.”

As discussed previously in the plan, there is evidence that the risk of developing COPD is inversely related to socioeconomic status. This tracks the documented trend of prevalence rates of tobacco use, which is also indirectly related to socioeconomic status. The relationship between social position and respiratory disease may also be attributed to housing conditions, poor nutrition, exposure to occupational pollutants, and childhood exposure to indoor air pollution.

Arizonans of lower socioeconomic status are least likely to have access to early detection of and intervention with COPD and tend to have erratic relationships with healthcare providers, making disease management and follow-up inconsistent if it is available at all. They also are least likely to take advantage of state funded tobacco cessation programs.

Identifying differences in how racial and cultural groups in our state view and interact with healthcare systems will also be important in establishing programs and methods to influence members of these groups who are at risk or afflicted with chronic diseases. Although currently COPD prevalence is greatest among the white population, the large numbers of non-whites who are at risk and who are afflicted with lung disease, as well as the many who may be undiagnosed, make this essential in planning interventions.

**Strategies**

- Identify specific populations with increased rates of COPD and/or with limited access to COPD resources.
- Explore constructs contributing to the burden of COPD in identified disparate populations.
- Collaborate with community partners to identify and promote available COPD-related resources and education to rural and border communities.
- Develop, promote and disseminate COPD-related resources that are culturally sensitive.
- Develop, promote, and disseminate COPD-related resources that meet the needs of all Arizona residents taking into account socioeconomic status, race, education level, language and age.
- Working in conjunction with the Arizona Healthcare Cost Containment System, develop a system to aid uninsured residents with COPD to obtain health insurance coverage.
- Identify emergency assistance programs and organizations providing free or discounted services, medications, and/or medical equipment statewide.
- Assist healthcare providers and community healthcare organizations to provide COPD education to all patients diagnosed in Arizona.
Collaborative Efforts

Objective
13. Foster communication, collaboration and networking opportunities among patients, caregivers, healthcare professionals, public health officials, and other stakeholders.

Rationale
Partnerships are essential in any successful disease control program. For COPD, few established coalitions and workgroups exist. It is essential that these be created statewide to begin to address the seriousness of COPD as a public health issue. Collaborative efforts among medical organizations, government agencies, patient groups, and policy makers can provide a concerted effort to reduce the prevalence and mortality of the disease. By sustaining and expanding a strong network, stakeholders can promote unified, consistent messages to raise awareness of COPD.

By bringing together healthcare professionals, patient organizations, governmental agencies, and other stakeholders, efforts can be made to define barriers, create and implement programs for prevention and early detection, promote better care for patients, reach undiagnosed patients with information, and improve the quality of life for those impacted by the disease.

A well-established coalition can also stimulate scientific research to better understand COPD, seek new treatments for the disease, as well as prevention strategies. There also would be opportunities to define and evaluate programmatic best practices, promote established clinical guidelines, and create a platform for community advocacy and the mobilization and coordination of efforts to combat the disease.

Strategies
• Foster the establishment of community-based healthcare coalitions for COPD, including the participation of physicians and other healthcare professionals.
• Develop public-private partnerships with organizations and government agencies that include, or could be expanded to include, projects related to COPD.
• Bring together COPD stakeholders to continually refine, alter, promote and monitor the implementation of the COPD state plan.

Advocacy

Objective
14. Advocate and support policies to reduce the prevalence of tobacco use and secondhand smoke exposure among Arizonans.

Rationale
Policies that support and promote tobacco abstinence and clean air quality ordinances are important in the overall reduction of COPD morbidity and improvement of quality of life of persons with the disease. Increased awareness of COPD as an important health problem by decision
makers can set the stage for such measures. The relationship between tobacco use and the incidence of COPD justifies public policy intervention due to the high direct medical costs and indirect disability costs associated with the disease.

Voluntary worksite policies and local ordinances promoting smoke-free environments have spurred interest in a statewide law that would protect all Arizonans from the dangers of environmental tobacco smoke while stressing importance of tobacco abstinence. Additionally, providing a consistent minimal level regulation for smoking in public places would alleviate confusion and competition among neighboring communities.

**Strategies**

- Educate policy makers about the burden of COPD in Arizona and the risk relationship of tobacco use and secondhand smoke exposure.
- Public policies that promote clean indoor air quality.
- Statewide smoke-free initiative to prohibit smoking in all enclosed public places.

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**Public Awareness**

**Objective**

15. Increase awareness of the medical community, public health officials and the general public that COPD is a serious public health problem in Arizona.

**Rationale**

COPD has generally been overlooked by the public and health community, especially in relationship to its significant impact on mortality and morbidity worldwide. Some of the reasons for this may be incomplete information about the disease and its prevalence. It is also a disease that remains undetected until it is moderately advanced, its symptoms being attributed to aging or poor physical conditioning, with a long drawn out course towards decline and death as compared to more dramatic and sudden life-threatening illnesses. The fact that smoking tobacco causes 80 to 90% of COPD cases has caused COPD to be viewed as self-inflicted, and thereby less important than other chronic diseases, especially in regard to resources allocated to provide surveillance, conduct research, and improve lifestyle for patients and families.

Difficulties in determining the human and economic burden of COPD have previously been discussed within this plan, as well as confusion over the definition of the disease. Another challenge to positive patient outcomes is the prevalence of later stage diagnosis.

In order to raise awareness of COPD, a consistent, unified message needs to be developed
and promoted through educational programs, workshops, and special events. The increasing burden of COPD will not be arrested until knowledge and public awareness of the disease is promoted, thereby increasing the demand for accurate surveillance and improved public health interventions. In order to do this, committed organizations and individuals must be brought together to exchange information and plan mobilization to raise the profile of this forgotten disease and bring attention to these critical issues.

Strategies

- Develop a COPD awareness campaign with a clear, consistent message about COPD, tailored to different constituencies.

- Target the awareness campaign to a variety of groups, including health professionals, decision makers, providers, payers, lay public, and patients.

- Promote awareness of occupational and environmental conditions that present risk factors for the development and exacerbation of COPD cases.

Future Directions

The ultimate goal of creating a comprehensive COPD disease control plan should be to coordinate existing services and available resources, identify gaps in services, and provide a guide for future endeavors. In order to achieve this goal, future steps for this comprehensive COPD disease control plan for the State of Arizona should include stakeholder participation in assessing and modifying the plan as workgroups see fit. Stakeholders may include among others, medical and public health professionals, patients and caregivers, employers, health insurance providers, and representatives from the AHCCCS program. This comprehensive plan is a framework, and in order to refine and reach toward implementation, a formal collaboration across multiple organizations and disciplines needs to be formed to set priorities of action, mobilize support to implement priorities, and put in place an evaluation system to monitor progress and periodically reassess priorities. In addition, the plan should be updated and revised on a continuous basis to ensure the plan is addressing relevant issues and meeting the needs of persons with COPD in Arizona.
References


19 National Pharmaceutical Council, Inc.. *Disease Management for Chronic Obstructive Pulmonary Disease, September 2003.*


“Chronic Obstructive Pulmonary Disease is the only major disease that is rising in prevalence and mortality while all other major causes of death are declining.”