CHAPTER II

REVIEW OF LITERATURE
CHAPTER II

REVIEW OF LITERATURE

2.1.1 Indoor Air Pollution Exposure Concern

2.1.2 Respiratory Health Impacts of Indoor Air Pollution on Women

2.1.2.1 Asthma

2.1.2.2 COPD (Chronic Obstructive Pulmonary Disease)

2.1.2.3 Respiratory Tract Infection

2.1.3 Respiratory Health Impact Assessment

2.1.3.1 Respiratory Health Questionnaire

2.1.3.2 Spirometry

2.1.3.3 Breath Exhaled Carbon Monoxide Monitor
2.1.1 INDOOR AIR POLLUTION EXPOSURE CONCERN

2.1.1 (a) Sources of Fuel:

About 1.5 million years ago, our ancestors Homo erectus learned to light fire from wood to keep wild animals away. Over the years, the human race has used several biological sources of fuel to produce fire, for not only getting rid of enemies but also for cooking and heating. These sources have included wood, charcoal, dried twigs and grass, crop residues, and animal dung cakes, which collectively are called biomass fuels.54

2.1.1 (b) Burning of Fuels:

The air pollution is caused by both natural and man-made sources. Major man-made sources of indoor environments are tobacco smoke and combustion of biomass fuels for cooking and heating which are the most significant sources. In addition, mold can also be a significant source of biological pollutants indoors.73 Indoor air pollution is one of the leading causes of morbidity and mortality. Burning of biomass fuels is ranked as one of the top ten risk factors for the global burden of disease.56 However the estimate of biomass use every day is greater than 2 billion kg worldwide18 in open fires and insufficient stoves in poorly ventilated rooms which are making biomass smoke one of the most important sources of indoor air pollution globally. Although biomass fuels are cheap and easily available, they are insufficient not only because they are low warming but also because they produce many pollutant products. For example, wood smoke is a complex mixture of numerous volatile and particulate substances derived from wood polymers and resins. More than 200 chemical and compound groups have been identified, and > 90% of these are in the inhalable size
A significant number of these biomass smoke constituents are known to be toxic or have irritant effects on the respiratory tract and include particulate matter that are < 10 microns in aerodynamic diameter (PM$_{10}$), carbon monoxide (CO), nitrogen dioxide, sulfur dioxide, aldehydes (eg formaldehyde), polycyclic aromatic hydrocarbons (eg benzopyrene), volatile organic compounds, chlorinated dioxins, and free radicals. Among these, PM$_{10}$ has the most significant adverse health impacts. In homes that use biomass fuel, the mean 24-h PM$_{10}$ levels have been shown to reach 300 to 3,000 µg/m$^3$ and sometimes can be as high as 30,000 µg/m$^3$. Daytime respirable particulate measurements in homes using biomass fuel in China, Kenya, Mexico, Guatemala, Brazil, and India show average PM$_{10}$ levels of around 1,000 µg/m$^3$ but may easily reach levels up to 3,000 µg/m$^3$. The United States Environmental Protection Agency (EPA) safety standard for 24-h average PM$_{10}$ exposure is 150 µg/m$^3$. The levels encountered in homes that use biomass fuel are therefore around 10 to 70 times above ambient levels observed in some of the most polluted cities of the world. Moreover, the mean CO concentrations in homes that use biomass fuel are typically in the range of 2 to 50 ppm but can be as high as 500 ppm during cooking, which is significantly greater than the EPA 8-h safety standard for CO (< 9 ppm).

2.1.1 (c) Exposure Time-Span:

In developing countries, women living in rural areas and urban slums spend several hours every day cooking food using biomass fuel. They are therefore exposed to extremely high levels of indoor air pollutants every day for several years. However, several studies indicated that biomass fuel can emit more main indoor air pollutants than other types of cooking fuels as shown in Table 4.
Table 4: Indoor air pollutants emission by different types of cooking fuels

<table>
<thead>
<tr>
<th>Type of cooking fuel</th>
<th>Indoor air pollutants emission (μg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PM\textsubscript{10}</td>
</tr>
<tr>
<td>LPG</td>
<td>73</td>
</tr>
<tr>
<td>Kerosene</td>
<td>203</td>
</tr>
<tr>
<td>Wood</td>
<td>500</td>
</tr>
<tr>
<td>Dung</td>
<td>732</td>
</tr>
</tbody>
</table>

2.1.1 (d) Respiratory Health Effects on Women:

Summarily, the facts that mentioned above imply that mankind still has to face adverse respiratory health effects due to combustion of cooking fuel, particularly, in women who spend more time in the kitchen. The true burden of respiratory illness and its association with indoor environmental factors, especially amongst women in India and Thailand are not known. Thus it is essential to concentrate on studying respiratory health effects on women from indoor smoke by different types of cooking fuels in India and Thailand.

2.1.2 RESPIRATORY HEALTH IMPACTS OF INDOOR AIR POLLUTION ON WOMEN

2.1.2 (a) Toxic Pollution and Women:

Respiratory health is influenced by a wide range of physical, social and environmental factors. In addition to the production of toxic pollution, the supply and use of household energy in conditions of poverty and scarcity affects health - particularly of women and young children. Association of exposure with chronic bronchitis and chronic obstructive lung disease is quite well established, particularly among women.
2.1.2 (b) Impact on Three Diseases:

Results of previous epidemiological studies of indoor smoke summarized that women had particular respiratory impact due to indoor smoke three diseases; chronic respiratory disease (asthma, COPD), lung cancer, and respiratory tract infection. However, lung cancer which causes from coal but it is not mainly used for cooking in India and not used in Thailand therefore the present researcher has reviewed literature chronic respiratory disease and respiratory tract infection except lung cancer.

2.1.2.1 Asthma

2.1.2.1 (a) Definition

Asthma is an inflammatory disorder of the airways, which causes attacks of wheezing, shortness of breath, chest tightness, and coughing.\textsuperscript{82}

2.1.2.1 (b) Signs and symptoms

Common symptoms of asthma in a steady-state include: nighttime coughing, shortness of breath with exertion but no dyspnea at rest, a chronic 'throat-clearing' type cough, and complaints of a tight feeling in the chest. Severity often correlates to an increase in symptoms. Symptoms can worsen gradually and rather insidiously, up to the point of an acute exacerbation of asthma.\textsuperscript{82}

2.1.2.1 (c) Causes

Asthma is caused by inflammation in the airways. When an asthma attack occurs, the
muscles surrounding the airways become tight and the lining of the air passages swell. This reduces the amount of air that can pass by, and can lead to wheezing sounds. In sensitive individuals, asthma symptoms can be triggered by breathing in allergy-causing substances (called allergens or triggers). Common asthma triggers include:

(i) Animals (pet hair or dander),
(ii) Dust,
(iii) Changes in weather (most often cold weather),
(iv) Chemicals in the air or in food,
(v) Exercise,
(vi) Mold,
(vii) Pollen,
(viii) Respiratory infections, such as the common cold,
(ix) Strong emotions (stress), and
(x) Wood and tobacco smoke.

Many people with asthma have an individual or family history of allergies, such as hay fever (allergic rhinitis) or eczema. Others have no history of allergies.

2.1.2.1 (d) Diagnosis

Asthma is defined simply as reversible airway obstruction. The basic measurement is peak flow rates and the following diagnostic criteria are used by the British Thoracic Society:

(i) 20% difference on at least three days in a week for at least two weeks;
(ii) 20% improvement of peak flow following treatment, for example:

- 10 minutes of inhaled β-agonist (e.g., salbutamol);
- Six weeks of inhaled corticosteroid (e.g., beclometasone);
- 14 days of 30 mg prednisolone.

(iii) 20% decrease in peak flow following exposure to a trigger (e.g., exercise).

As research summaries for small airways disease indicates, small airways disease is defined by a reduction in forced expiratory flow at 25-75% of the pulmonary volume (FEF_{25-75\%}) and normal spirometry [normal forced expiratory volume at 1 second (FEV₁), forced vital capacity (FVC), and FEV₁/FVC ratio], may be a marker for early allergic or inflammatory involvement of the small airways in subjects with allergic diseases and no asthma. In addition, sometimes the only abnormality is a reduction in FEF_{25-75\%}. Isolated reduction in FEF_{25-75\%} is considered an early detector of very mild obstruction. It can also be a normal variant.

The FEF_{25-75\%} also reflects the severity of pulmonary impairment in obstruction. Even though FEF_{25-75\%} is used to define small airways obstruction but in case of investigating for prevalence or health protection and promotion from risk factor of obstructive airways disease, for instance, detecting of early COPD, smoking cessation, is more effective. While using spirometry to define and classify small airways diseases, Table 5 is established for classification of small airways obstruction severity which proceeded to show that FVC value is equal or greater than 81% of predicted, but FEF_{25-75\%} < 65\%. 
Table 5: Classification of small airways obstruction severity

<table>
<thead>
<tr>
<th>%predicted FEF_{25-75%}</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>66%</td>
<td>normal</td>
</tr>
<tr>
<td>50 - 65%</td>
<td>mild small obstruction</td>
</tr>
<tr>
<td>35 - 49%</td>
<td>moderate small obstruction</td>
</tr>
<tr>
<td>&lt; 35%</td>
<td>severe small obstruction</td>
</tr>
</tbody>
</table>

From: Pulmonary Function Testing. A Practical Approach^{88}

2.1.2.1 (e) Implication of indoor air pollution on asthma

International variations in the prevalence of asthma,^{89} together with recent increases in many countries, have focused attention on the role of air pollution. The complex influence of air pollution on the development of asthma is a matter of controversy. (i) While some assert that air pollution, including environmental tobacco smoke, may be a factor sensitizing genetically susceptible individuals to allergens in early life,^{90} (ii) a recent systematic review does not support this view in so far as environmental tobacco smoke is concerned.^{91} (iii) There is more consistent evidence that air pollution and environmental tobacco smoke trigger asthma in sensitized individuals.^{92} (iv) In developing countries, studies on biomass smoke in relation to asthma in children and adults have yielded mixed findings.^{93-107} (v) A questionnaire survey of children aged 9–12 years in Turkey, which included spirometry, found that coal users had more day/night cough (p < 0.05) and that those using wood-burning stoves had the lowest values of FVC, FEV1, PEFR (peak expiratory flow rate) and FEF_{25\%} (forced expiratory flow rate at 25% of lung volume);^{93} however, there was no adjustment for confounding. (vi) A matched case-control study of people aged 11–17 years in rural Nepal found an adjusted odds ratio of 2.3 (1.2–4.8) for asthma among those using wood fires or stoves compared to gas or kerosene (Schei, personal communication). (vii) In Jordan a cross-
Sectional study of lung function in children aged 11–13 years found significantly reduced FVC, FEV₁, PEFR and FEF₂₅₋₇₅% for exposure to wood/kerosene stoves and environmental tobacco smoke, but no adjustment was made for confounding.⁹⁴ (viii) A case-control study of schoolchildren in Nairobi found increased exposure to wood smoke in asthmatics.⁹⁵ (ix) Several studies, however, have reported no association. (x) A case-control study of children aged between 1 month and 5 years who were hospitalized with asthma in Kuala Lumpur found that the use of kerosene or wood stoves was not independently associated with asthma, but that there was an association between mosquito coil smoke and this disease.⁹⁶ (xi) Noorhassim found no association between asthma diagnosed by doctors or reported wheeze and biomass smoke in a cross-sectional study of 1007 children aged 1–12 years in Malaysia.⁹⁷ (xii) A study in urban Maputo found no association after adjustment between fuel type and either wheeze or peak flow.⁹⁸ (xiii) Qureshi found no association in rural Pakistan, although the number of people with asthma was small.⁹⁹ (xiv) Preliminary findings of another cross-sectional study of 1,058 children aged 4–6 years in rural Guatemala, in which the methods of the International Study of Asthma and Allergy in Childhood (ISAAC) were used, suggest a possible protective effect.

(xv) A study of nearly 29,000 adults in rural China reported that the adjusted odds ratios for wheezing and asthma for a group with occupational exposure to wood or hay smoke were 1.36 (1.14–1.61) and 1.27 (1.02–1.58) respectively.¹⁰⁰ Since 93% of the sample used wood or hay for cooking the relationship with asthma was studied among the 39% of women and 21% of men exposed occupationally. Similarly elevated odds ratios were reported for those using coal for cooking. (xvi) Mixed findings have also been reported from developed countries, several studies having found positive associations¹⁰¹ (xvii) and some having found no association, as with children aged 5–9 years in Seattle.¹⁰²
There is evidence that biomass smoke is associated with reduced risk, reflecting a possible protective effect. (xviii) Von Mutius found the risk of hay fever, atopy and bronchial reactivity to be reduced in rural German children aged 9–11 years whose homes were heated by coal or wood.¹⁰³ (xix) Similar evidence has been reported from urban Australia.¹⁰⁴

Overall, the evidence on exposure to biomass smoke and asthma in developing countries is limited and inconsistent. Although asthma is less common among rural populations where biomass fuels are used most, it should not be assumed that smoke exposure is protective in these settings.

(xx) For asthmatic attacks, Desai and colleagues,¹⁰⁵ taking into account three studies, ⁹⁶,¹⁰⁰,¹⁰⁶ have estimated that exposure to solid fuel smoke exacerbates asthma with a relative risk of 1.6 (95% CI, 1.0–2.5%) for children between 5 and 14 years and of 1.2 (95% CI, 1.0 – 1.5) for persons older than 15 years. The relationship between indoor air pollution and the development of asthma is even more controversial. (xxi) Mishra and colleagues¹⁰⁷ determined that elderly men and women living in households using biomass fuels have a significantly higher prevalence of asthma than those living in households using cleaner fuels, with an OR of 1.59 (95% CI, 1.30–1.94); the adjusted effect was higher among women (OR, 1.83; 95% CI, 1.32–2.53) than among men (OR, 1.46; 95% CI, 1.14–1.88).

2.1.2.3 COPD (Chronic obstructive pulmonary disease)

COPD is characterized by progressive airflow obstruction and destruction of lung parenchyma, and is caused by chronic exposure of genetically susceptible individuals to environmental factors. Tobacco smoking is established as a major risk factor, but
emerging evidence suggests that other risk factors are important, especially in developing countries.\textsuperscript{108}

\subsection{2.1.2.3 (a) Definition}

COPD is one of the most common lung diseases. It makes it difficult to breathe. There are two main forms of COPD:

(i) Chronic bronchitis, defined by a long-term cough with mucus, and

(ii) Emphysema, defined by destruction of the lungs over time.

Most people with COPD have a combination of both conditions.\textsuperscript{109}

\subsection{2.1.2.3 (b) Signs and symptoms}

One of the most common symptoms of COPD is shortness of breath (dyspnea). In the advanced stages of COPD, dyspnea can become so bad that it occurs during rest and is constantly present. Other symptoms of COPD are a persistent cough, sputum or mucus production, wheezing, chest tightness, and tiredness.\textsuperscript{109,110}

Common signs are:\textsuperscript{109}

(i) Tachypnea, a rapid breathing rate,

(ii) Wheezing sounds or crackles in the lungs heard through a stethoscope,

(iii) Breathing out taking a longer time than breathing in,

(iv) Enlargement of the chest, particularly the front-to-back distance (hyperinflation),

(v) Active use of muscles in the neck to help with breathing,

(vi) Breathing through pursed lips, and
(vii) Increased anteroposterior to lateral ratio of the chest (i.e. barrel chest).

2.1.2.3 (c) Causes

Smoking is the leading cause of COPD. The more a person smokes, the more likely that person will develop COPD although some people smoke for years and never get COPD. In rare cases, nonsmokers who lack a protein called alpha-1 antitrypsin can develop emphysema.\(^\text{109}\)

Other risk factors for COPD are:

(i) Exposure to certain gases or fumes in the workplace,

(ii) Exposure to heavy amounts of secondhand smoke and pollution, and

(iii) Frequent use of cooking gas without proper ventilation.\(^\text{109}\)

2.1.2.3 (d) Diagnosis

The diagnosis of COPD should be considered in anyone who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease such as regular tobacco smoking.\(^\text{111,112}\) No single symptom or sign can adequately confirm or exclude the diagnosis of COPD\(^\text{113}\) although COPD is uncommon under the age of 40 years. The diagnosis of COPD requires lung function tests.

2.1.2.2 (e) Implication of indoor air pollution on COPD

COPD is one of the most important causes of global burden of disease in people older than 40 years and is increasing.\(^\text{114}\) (i) In developed countries, most COPD cases are
related to cigarette smoking. In developing countries, COPD is also a prevalent condition. (ii) In Latin America, the prevalence of COPD varies from 7.8 to 19.7% in the urban population aged 40 years and older. In these countries, a significant fraction of COPD, which could reach 50%, especially in women, occurs in never-smokers, and could be attributed predominantly to biomass (wood) burned in open stoves for cooking (and heating in the colder, higher altitudes).

(iii) A large number of mainly cross-sectional and case-control studies have found association of exposure to solid fuel smoke with COPD, chronic bronchitis, chronic airway disease, and airflow obstruction, especially in women. (iv) The overall risk of COPD in women exposed to indoor air pollution from domestic solid fuel use, especially wood, estimated by Smith and coworkers, was consistently higher (OR, 3.2; 95% CI, 2.3–4.8) than in men (OR, 1.8; 95% CI, 1.0–3.2), who were likely less exposed. (v) Two recent probabilistic, population-based studies ratified a clear association between the exposure to smoke from biomass fuels and COPD defined by a post-bronchodilator FEV₁ to FVC ratio less than 70%. (vi) One of these studies, which included 5,539 people, demonstrated that cooking 10 years or more with a wood stove was an independent risk factor for COPD after adjusting by age, sex, active and passive smoking, education level, history of tuberculosis, and exposure to charcoal or dust at work (OR, 1.50; 95% CI, 1.36–2.36; P < 0.001). This finding could be partially explained by the persistent high levels of pollutants in living and sleeping areas at homes where biomass fuels are used.

(vii) An increased risk for COPD in people exposed to wood and charcoal smoke (OR, 4.5; 95% CI, 1.4–14.2) was found in Spain, and it would be important to confirm this association in other developed countries.
(viii) The report of respiratory symptoms, especially phlegm and cough, is consistently higher in women cooking with biomass fuels in comparison with those using cleaner fuels (charcoal, gas, kerosene).\textsuperscript{98,128,131} This finding has been associated with the PM\textsubscript{10} concentrations, which often exceed 2,000 $\mu$g/m$^3$.\textsuperscript{131} For example, wood users (mean PM\textsubscript{10}, 1,200 $\mu$g/m$^3$) had significantly more cough than charcoal (PM\textsubscript{10}, 540 $\mu$g/m$^3$), liquefied petroleum gas (LPG), and electricity users (PM\textsubscript{10}, 200–380 $\mu$g/m$^3$).\textsuperscript{100} The use of biomass fuels, mainly wood, has been also associated with an impairment of pulmonary function. Mild to moderate reductions of FEV\textsubscript{1}/FVC, FEV\textsubscript{1}, FEV\textsubscript{1}%, PEF, and FEF\textsubscript{25–75}% have been associated with the exposure to indoor biomass burning in cross-sectional studies.\textsuperscript{127,131} (ix) Other studies, mainly hospital-based case-control studies, confirm that people exposed to biomass smoke have a high risk for developing airflow obstruction with significant reduction of FEV\textsubscript{1} and FEV\textsubscript{1}/FVC.\textsuperscript{118,129,134} (x) The exposure-response curves for COPD related to indoor biomass smoke exposure have not been established, but in a case-control study,\textsuperscript{134} the risk for chronic bronchitis and chronic airway disease increased linearly with the exposure estimated as hour-years (average hours a day cooking with a wood stove multiplied by years of cooking), and the risk of airflow obstruction increased briskly above 200 hour-years.\textsuperscript{137}  

2.1.2.2 (f) Comparison between COPD related to smoking and to biomass smoke exposure

Wood smoke-attributable COPD presents clinically with minimal emphysema as a chronic obstructive disease with persistent cough, phlegm and dyspnea.\textsuperscript{138,139} The women with wood smoke-attributable COPD tend to be older, shorter, and have a greater body mass index than those with cigarette smoking-attributable COPD;\textsuperscript{117,138}
However, most clinical characteristics, quality of life, and mortality were similar in both groups once severity of airflow obstruction was taken into account.\textsuperscript{117} Lung morphology in necropsies from women with COPD only exposed to tobacco smoke and from those only exposed to biomass smoke\textsuperscript{138} shows varying severities of similar alterations. For example, anthracosis and scarring were more frequent and emphysema milder in wood smoke–attributable COPD compared with smokers. Widespread mucosal swelling and anthracotic plaques of the airways have also been described in women exposed to biomass smoke.\textsuperscript{140}

(i) Adults aged over 45 years had a high prevalence of respiratory symptoms and disease, similar in men and in women, and 20% of men and 10% of women had an FEV\textsubscript{1}/FVC (forced expiratory volume in one second/forced vital capacity) below 60%.\textsuperscript{141} The clinical presentation was as chronic obstructive pulmonary disease with, in a few patients, local lung fibrosis and bronchiectasis (localized destruction and infection of the lung),\textsuperscript{142} and disease was attributed to indoor air pollution and repeated infections. Most patients were smokers of home-grown tobacco, inhaled in a similar way to cigars, but no association with smoking was found for airflow obstruction or mortality.\textsuperscript{143} (ii) Numerous studies, including ones with cross sectional and case-control designs, have reported on the association between exposure to biomass smoke and chronic bronchitis or chronic obstructive pulmonary disease.\textsuperscript{26,30,94,95,114,116,118-124,131,135,144-151} (iii) In Nepal, the prevalence of chronic bronchitis was similar in men and women (18.9%); this would not have been expected if cigarette smoking, being commoner in men, had been the main cause.\textsuperscript{30,120} (iv) The prevalence of chronic bronchitis was also greater in women in Ladakh, where few women smoke,\textsuperscript{26} (v) and in Pakistan.\textsuperscript{99} (vi) Exposure to biomass smoke has been reported as more frequent in
people with airflow obstruction in hospital-based case-control studies and some community studies. In hospital-based studies, obstruction was often severe and the association with exposure was strong, adjusted odds ratios being in the range 1.8–9.7. One community study reported an adjusted odds ratio of 2.5, but in spirometric studies the reported differences in lung function associated with exposure to wood smoke have usually been relatively small, probably reflecting the selection of much more severe cases in hospital studies. In rural Mexico the use of biomass was associated with a 4% decrease in FEV1/FVC, while an increase in the kitchen particle concentration of 1000 mg/m³ was associated with a reduction of 2% in FEV1. (x) In India, patients using biomass had lower FVC than those using kerosene, gas and mixed fuels. Pandey reported an exposure-response relationship with FEV1 and FVC which decreased as the reported hours of exposure increased; it was not statistically significant in non-smokers. (xii) Experience with cigarette smokers suggests that fewer than 15% of people exposed to wood smoke are likely to develop chronic obstructive pulmonary disease or chronic bronchitis, although this may depend on the level of exposure. Exposure was usually estimated from questionnaires as present or absent, as hours spent close to a wood stove, or as hours multiplied by years of exposure. (xiii) The studies measuring particle levels in kitchens confirmed very high concentrations; a time-budget assessment was also made in one of these studies. (xiv) Norboo reported the use of kitchen and exhaled personal carbon monoxide levels. Chronic bronchitis has generally been determined by questionnaire, while spirometry has been employed to determine airflow obstruction and chronic
obstructive pulmonary disease. (xv) In many of the studies there has been scant attention to quality control.

2.1.2.3 Respiratory Tract Infection

On the basis of the various existing (i) epidemiological studies, respiratory tract infection which affect from indoor smoke is acute respiratory infections (ARI), acute lower respiratory infections (ALRI), pneumonia, and pulmonary tuberculosis. A number of studies have shown that biomass smoke is an important cause of indoor pollution (ii) and is one of the predisposing factors in Acute Respiratory Infection (ARI) and rhinitis. (iii) The highest exposure is most likely experienced by women, infants and young children. (iv) In the short term, indoor air pollution can cause irritated or dry mucous membranes in the eyes, nose, respiratory tract and throat. It may also cause dizziness, fatigue, fever, forgetfulness, headaches, irritability, lethargy and nausea. Often, the health effects of indoor air pollution are attributed to colds and flu but they can build into asthma, hypersensitivity pneumonitis, pneumonia and pulmonary tuberculosis. (v) Exposure to biomass smoke has been strongly associated with ARI in preschool age children. (vi) Studies in developed countries show an association between biomass fuel and indoor air pollution and respiratory allergy in children. (vii) ARI, ALRI and pneumonia due to indoor smoke are most important cause of death in children under five years that was not reported in adulthood. This study aims to explore respiratory health effects due to indoor smoke in female adult, so the respiratory diseases of infant and childhood need to be excluded in the literature review.
As the criteria of spirometry which has precaution on the subject with communicable diseases such as acute respiratory tract infection, pulmonary TB, the researcher is required to exclude pulmonary TB out of this study also.

2.1.3 RESPIRATORY HEALTH IMPACT ASSESSMENT

2.1.3 (a) Tools of Assessment: Questionnaire, Spirometry and Breath Exhaled CO Monitor:

The three research objective tools are respiratory health questionnaire, spirometry and exhaled breath CO monitor, which will be used to assess the respiratory health impacts; asthma, COPD and lung oxidative stress in this study.

2.1.3.1 Respiratory Health Questionnaire

The questionnaire in this study is mixed from validated respiratory health questionnaire that has used nine items of ISAAC (International Study of Asthma and Allergic in Childhood) questionnaire for asthma investigating and nine items of BOLD (The burden of obstructive lung disease) questionnaire which has been developed from pre-existing validated questionnaires that have already been used in multinational studies. It is obtained from the information about respiratory symptoms (cough, sputum, wheezing, shortness of breath), exposure to potential risk factors, occupation, respiratory diagnoses (asthma, emphysema, COPD, chronic bronchitis, etc.), co-morbidities, health care utilization, medication use, activity limitation, and health status. It includes sections taken from the 1978 ATS/DLD Respiratory Symptom Questionnaire and the questionnaires used in the European Community Respiratory Health Study and the US
Lung Health Study and the SF-12 to assess overall health status. This study tool also includes items on standard socioeconomic-demographic variables.

2.1.3.2 Spirometry

2.1.3.2 (a) Definition

Spirometry (meaning *the measuring of breath*) is a dependent effort test and the most common of the Pulmonary Function Tests (PFT), measuring lung function, specifically the measurement of the amount (volume) and speed (flow) of air that can be inhaled and exhaled. Spirometry is an important tool used for generating pneumotachographs which are helpful in assessing conditions such as asthma, pulmonary fibrosis, cystic fibrosis, and COPD.\(^\text{160}\) This test is performed using a device called a spirometer, which comes in several different varieties. Most spirometers display the following graphs, called spirograms:

(i) A *volume-time curve*, showing volume (liters) along the Y-axis and time (seconds) along the X-axis (fig 7A).

(ii) A *flow-volume loop*, which graphically depicts the rate of airflow on the Y-axis and the total volume inspired or expired on the X-axis (fig 7B).\(^\text{160}\)

\begin{itemize}
  \item A.
  \item B.
\end{itemize}

*Figure 7: A. Volume-time curve, B. Flow-volume loop.\(^\text{160}\)*
2.1.3.2 (b) Procedure

The most important step before the start of spirometry is to calibrate the machine, using one litre or three litre calibration syringe. All diagnostic spirometers must be capable of being calibrated and this should be done regularly (preferable daily). The most common error noted in the spirometers if not calibrated regularly is that it gives false over-reading of values - FEV₁ and FVC showing values between 200-500% higher than predicted normal are not uncommon. After spirometry calibrating, the participant’s age, gender, and race are recorded, and height and weight are measured before the procedure begins. The participant should not have eaten heavily within three hours of the test and should be instructed to wear loose-fitting clothing over the chest and abdominal area. The participant is explained and demonstrated the breathing maneuvers. Then practice breathing into the mouthpiece until the participant is able to duplicate the maneuvers successfully on three consecutive attempts. Generally, the participant is asked to take the deepest breath they can, and then exhale into the sensor as hard as possible, for as long as possible. It is sometimes directly followed by a rapid inhalation (inspiration), in particular when assessing possible upper airway obstruction. The test will be preceded by a period of quiet breathing in and out from the sensor (tidal volume), or the rapid breath in (forced inspiratory part) will come before the forced exhalation. During the test, soft nose clips have to be used to prevent air escaping through the nose like a filter mouthpieces must be used to prevent the spread of microorganisms. The spirometry test needs at least 3 acceptable and 3 repeatable reading for quality assurance. Acceptability and repeatability criteria are as follows:

Acceptability criteria:

(i) No inadequate inspiratory effort-only seen on flow-volume loop.
(ii) No slow hesitated start-best seen to Volume/Time Graph.

(iii) No cough-best seen on Flow/Volume Graph.

(iv) No poor effort-see the shape of the expiratory loop on Flow/Volume Loop.

(v) No early termination-FET should be preferably 6 seconds or more. Less than 6 seconds may be permitted if the expiratory loop has reached a plateau on the Volume/Time curve for at least one second.

(vi) No glottic closure or obstruction of mouthpiece due to tongue.

Repeatability criteria:

(i) The two highest values of the FVC and FEV₁ taken from acceptable forced expiratory maneuvers must show minimal variability. Difference between the two best FVC and FEV₁ maneuvers should be within 200 ml. If more than 200 ml the values are repeatable and hence not reliable.

Despite, up to a maximum of 8 blows is recommended, because the subject is unlikely to give a better blow if she has not been able to do it 8 times, and also, it is very tiring to make subject blows for 8 times. The test should be taken at a later date.

2.1.3.2 (c) Precautions

Spirometry is contraindicated in participants whose condition will be aggravated by forced breathing, including:\textsuperscript{161}

(i) Hemoptysis (spitting up blood from the lungs or bronchial tubes),

(ii) Pneumothorax (free air or gas in the pleural cavity),

(iii) Recent heart attack,

(iv) Unstable angina,

(v) Aneurysm (cranial, thoracic, or abdominal),
(vi) Thrombotic condition (such as clotting within a blood vessel),

(vii) Recent thoracic or abdominal surgery,

(viii) Nausea or vomiting, and

(ix) Respiratory tract infection such as acute upper respiratory infection (common cold, flu) and lower respiratory infection (pneumonia, pulmonary tuberculosis).

2.1.3.2 (d) Parameters

The common pulmonary function values measured with spirometry are:

(i) **VC-Vital Capacity** - The amount of air that can be forcibly exhaled from the lungs after a full inhalation.

(ii) **FVC-Forced Vital Capacity** - The amount of air which can be forcibly exhaled from the lungs after taking the deepest breath possible.

(iii) **FEV₁-Forced Expiratory Volume in One Second** - The amount of air which can be forcibly exhaled from the lungs in the first second of a forced exhalation.

(iv) **FEV₁/FVC-Percent (FEV₁%)** - The ratio of FEV₁ to FVC is exhaled from the lungs during the first second of forced exhalation.

(v) **PEFR-Peak Expiratory Flow Rate** - The maximum flow rate of expired air is measured.

(vi) **Forced expiratory flow 25% to 75%** - The amount of air flow halfway through an exhale (FVC).

(vii) **FET-Forced Expiratory Time** - A measure of the length of the expiration in seconds.¹⁶²
2.1.3.2 (e) Type of spirometer

There are essentially two different types of spirometers

I - Volumetric Spirometers
   I-i Water bell
   I-ii Bellows wedge

II - Flow measuring Spirometers
   II-i Fleisch-pneumotach
   II-ii Lilly (screen) pneumotach
   II-iii Turbine
   II-iv Pitot tube
   II-v Hot-wire anemometer
   II-vi Ultrasound$^{163}$

Flow measuring spirometers or flow-sensor based spirometers utilize a sensor that measure flow as a primary signal and calculate volume by electronic (analogue) or numerical (digital) integration of the flow signal. The most commonly used flow sensors detect and measure flow from a pressure drop across a known, constant resistance, cooling of one or more heated wires, or by electronically counting the rotation of a turbine blade. Especially, the ultrasonic flow-sensor based spirometer which is lighter, portable, accurate, reliable and provide a print-out with all the spirometry indices desired.$^{164}$ Hence it has been used for collecting data in the present study. It has two versions; EasyOne®, Switzerland has been used in India and Microlab II®, UK has been used in Thailand (fig 8A, 8B).
Carbon monoxide (CO) is a product of heme degradation by HO. Two isoforms of HO have been described: the constitutive HO-2, which is highly expressed in the brain and testes; and the inducible HO-1, which is ubiquitous. The latter is activated by a variety of pro-inflammatory cytokines, nitric oxide, H$_2$O$_2$, endotoxin, and oxidants, and is an important part of a protective response to oxidative stress. Breath exhaled CO (eCO) is a useful marker of underlying oxidative stress in the lungs. eCO concentration is measured by breath exhaled CO monitor that is sensitive to CO concentrations from 1 to 500 ppm (by volume).

### 2.1.3.3 (a) Definition

Breath exhaled CO monitoring is performed using breath exhaled CO monitor that measures breath CO levels in parts per million (ppm).

### 2.1.3.3 (b) Physiology

Following inhalation, CO displaces oxygen in the erythrocyte to form carboxyhemoglobin (COHb). In this form, CO has a half-life of about 5 to 6 hours.
and may remain in the blood for up to 24 hours depending on a number of factors, such as gender, physical activity, and ventilation rate.\textsuperscript{176,177} While some exposure to CO may occur in normal day-to-day life, due to environmental pollution, passive smoking, and occupational exposure, the most likely cause of high levels of exposure is smoking.\textsuperscript{178}

2.1.3.3 (e) Procedure

The measurement of breath exhaled CO levels may provide an immediate, noninvasive method of assessing smoking status. Furthermore, the development of relatively inexpensive portable breath exhaled CO monitors enable eCO levels to be assessed in a wide variety of clinical settings.\textsuperscript{171,179} Breath exhaled CO monitor is based on the conversion of CO to CO\textsubscript{2} over a catalytically active electrode. On breath holding, the CO in the blood forms an equilibrium with the CO in the alveolar air; therefore, there is a high degree of correlation between eCO levels and COHb concentration. This enables the breath exhaled CO monitor to accurately estimate the blood COHb concentration from the eCO level. The breath exhaled CO monitor was calibrated weekly using a mixture of 50 ppm CO in air.\textsuperscript{180}

To standardize the breath being analyzed by the breath exhaled CO monitor, the subjects will be asked to exhale completely, inhale fully, and then hold their breath for 15 seconds. If the subjects will be unable to hold their breath for 15 seconds, they will be asked to hold it for as long as possible and the length of time are recorded. Following the breath hold, the subjects will be asked to exhale slowly into the breath exhaled CO monitor and will be encouraged to exhale fully in order to sample the alveolar air.\textsuperscript{180} In addition, exhaled CO breath test used measures the concentration of CO in the lung alveolar – a good proxy for exposure to indoor air pollution that in the absence of high
exposure to indoor air pollution, concentrations in the normal range of 0 to 3 ppm. For defining smoking status, concentrations in the range of 0 to 6 ppm would indicate that the person is a non-smoker, while 7 to 10 ppm, 11 to 20 ppm, and greater than 20 ppm would indicate that the person is a light, regular or heavy smoker, respectively.\textsuperscript{181,182} Exhaled breath CO monitor can measure and investigate major oxidative stress in the lungs which cause by exposure to indoor air pollution, since breath exhaled CO is biomarker of lung oxidative stress.

In this study the researcher has used Vitalograph \textsuperscript{®} (UK), the pocket sized breath exhaled CO monitor (fig 9), which is measured at a three times point.\textsuperscript{173}

\textbf{Figure 9:} Hand held, pocket-sized Breath CO monitor (Vitalograph\textsuperscript{®}, UK).\textsuperscript{173}

The second Chapter brings out the fact that (i) Asthma (ii) Chronic Obstructive Pulmonary Disease (COPD) and (iii) Respiratory Tract Infection are the Respiratory Health Impacts on women due to Indoor Air Pollution. Biomass fuel burning results in Indoor Air Pollution that causes these health threats. Each health hazard is explained in terms of definition, signs and symptoms, causes and diagnosis. The case studies have been reviewed and twenty-one case studies on Asthma, ten and fifteen (comparative) on COPD and on respiratory Tract Infection seven case studies have been recorded. The three objective tools of assessment such as Questionnaire, Spirometry and Breath Exhaled CO Monitor are discussed in details to present the procedure of assessment of health impact of burning biomass fuel for cooking purposes.