



CHAPTER II

BACKGROUNDS AND LITERATURE REVIEW

THEORETICAL BACKGROUNDS

2.1 Air Pollution

2.1.1 Current ambient air quality and trends in Bangkok

Monitoring of ambient air quality in Bangkok was carried out by Pollution Control Department (PCD) in both general background areas and roadside areas (Wangwongwatana *et al.*, 2006) as follows:

2.1.1.1 General ambient air quality monitoring

Continuous general ambient air monitoring stations are placed in residential, commercial, industrial and mixed areas of Bangkok which far away from main road 50 meters. Monitoring locations are carefully selected to ensure that monitoring stations are not directly influenced by any particular major sources so that the quality of the general ambient air in Bangkok is monitored and impacts to general population can then be evaluated. Originally, there were 6 continuous monitoring stations installed in 1983. The air pollutants being measured were only limited to CO, TSP, and Pb. They were subsequently renovated and upgraded and a few new stations were installed in October of 1996 bringing the total number of stations for general ambient air quality monitoring in Bangkok to 10 stations. Every station monitors CO, TSP, PM₁₀, Pb, SO₂, NO_x, and O₃. Hydrocarbons (HC) are also monitored in some monitoring stations. The new stations are equipped with 10-metre meteorological masts measuring wind speed, wind direction, temperature, humidity, and solar radiation.

2.1.1.2 Roadside street-level ambient air quality monitoring

Since, there are a lot of Thai people living and working in shop houses, which are in close proximity to the street, it is also necessary to monitor the quality of air at street level where these people are exposed to air pollution. Roadside ambient air quality monitoring stations are placed far away from the street 3-5 meters. Roadside ambient air quality monitoring in Bangkok is carried out in two different ways as follows:

a) Long-term continuous roadside ambient air quality monitoring

In 1991, four permanent on-line and real-time continuous roadside ambient air quality-monitoring stations were operated in Bangkok in the areas experiencing traffic congestion. Each station has its own electronic display board to continuously display instantaneous concentrations of CO, PM₁₀, and noise levels to the public. Simultaneously, data are transmitted via a dedicated telephone line and are logged into a central processing computer at PCD.

In October of 1996, PCD installed three new on-line roadside ambient air quality monitoring stations continuously measuring CO, TSP, PM₁₀, Pb, SO₂, NO_x, O₃ and HC. These new stations are equipped with 10-metre meteorological masts measuring wind speed and direction, temperature, humidity and solar radiation.

b) Short-term temporary roadside ambient air quality monitoring

In addition to permanent roadside monitoring stations, temporary monitoring of CO, TSP and Pb is carried out annually approximately 15 of the most congested streets in Bangkok at roadsides for a period of 2 to 4 consecutive weeks at each street. Data are collected manually every day.

Results of ambient air quality monitoring for more than 10 years indicate that the greatest concerned air pollutants in Bangkok are SPM, especially PM₁₀ and CO.

They are mostly emitted by transport sector. The World Bank estimated transport sector emissions of these two air pollutants in 1991 to be 76 and 1,065 thousand tons per year, respectively. The report also concluded that air pollution in Bangkok, due to high concentrations of SPM, is among the highest priority problems.

Table 2.1 and Table 2.2 summarized ambient air quality in Bangkok in 2004 in the general background areas and at roadside sites, respectively. As indicated, the principal concern is along the major roads in Bangkok where pollutant concentrations and frequency of exceeding of the ambient air quality standards (Table 2.3) for TSP, PM₁₀, and CO are high enough to result in significant adverse health impacts on the local population.

Table 2.1 Ambient air quality in the general areas of Bangkok in 2004

Pollutants	Range	Concentrations			Frequency of Exceeding Standard (%)
		95 percentile	Average	Standard	
TSP (24-hr), mg/m ³	0.02 – 0.32	0.21	0.11	0.33	0/436 (0)
PM ₁₀ (24-hr), µg/m ³	19.3 – 219.3	116.4	59.8	120	82/1,873 (4.4)
Pb (1-month), µg/m ³	0.02 – 0.34	0.22	0.09	1.5	0/107 (0)
CO (1-hr), ppm	0 – 8.3	2.0	0.7	30	0/71,616 (0)
CO (8-hr), ppm	0 – 5.2	1.8	0.7	9	0/74,282 (0)
O ₃ (1-hr), ppb	0 – 173.0	53.0	15.7	100	102/58,081 (0.18)
SO ₂ (1-hr), ppb	0 – 103.0	14.0	5.0	300	0/70,886 (0)
SO ₂ (24-hr), ppb	0 – 19.7	10.3	5.0	120	0/2,884 (0)
NO ₂ (1-hr), ppb	0 – 170.0	58.0	24.3	170	0/69,752 (0)

Source: PCD, 2005

Table 2.2 Ambient air quality at the roadside sites of Bangkok in 2004

Pollutants	Concentrations				Frequency of Exceeding Standard (%)
	Range	95 percentile	Average	Standard	
TSP (24-hr), mg/m ³	0.01 – 0.77	0.38	0.18	0.33	53/631 (8.4)
PM ₁₀ (24-hr), µg/m ³	21.5 – 224.8	135.2	78.5	120	243/2,282 (10.6)
Pb (1-month), µg/m ³	0.02 – 0.31	0.22	0.10	1.5	0/104 (0)
CO (1-hr), ppm	0 – 15.1	4.5	1.7	30	0/55,940 (0)
CO (8-hr), ppm	0 – 10.6	4.1	1.7	9	44/56,647 (0.08)
O ₃ (1-hr), ppb	0 – 143.0	45.0	12.4	100	12/24,977 (0.05)
SO ₂ (1-hr), ppb	0 – 66.0	17.0	7.0	300	0/24,615 (0)
SO ₂ (24-hr), ppb	0.4 – 23.6	12.8	7.0	120	0/1,069 (0)
NO ₂ (1-hr), ppb	0 – 172.0	77.0	34.7	170	1/24,895 (0.004)

Source: PCD, 2005

Table 2.3 Ambient air standards of Thailand (1995)

Pollutants	1-hr Average		8-hr Average		24-hr Average		1-yr Average		Measurement Method
	mg/m ³	ppm	mg/m ³	ppm	mg/m ³	ppm	mg/m ³	ppm	
1. PM ₁₀	-	-	-	-	0.12	-	0.05	-	Gravimetric-High Volume
2. TSP	-	-	-	-	0.33	-	0.10	-	Gravimetric-High Volume
3. CO	34.2	30	10.26	9	-	-	-	-	Non-Dispersive Infrared Detection
4. SO ₂	0.78	0.30	-	-	0.30	0.12	0.10	0.04	UV-Fluorescence
5. NO ₂	0.32	0.17	-	-	-	-	-	-	Chemiluminescence
6. O ₃	0.20	0.10	-	-	-	-	-	-	Chemiluminescence

Source: Notification of National Environmental Board No. 10 (1992) under the Enhancement and Conservation of National Environmental Quality Act B.E. 2535 (1992) published in the Royal Government Gazette No. 112 Part 52 dated May 25, B.E. 2538 (1995)

It was reported that 60 percent of TSP by weight in Bangkok is PM₁₀. Monitoring of PM₁₀ began in Bangkok in 1992. In 2004, 24-hour average concentrations of roadside ambient PM₁₀ in Bangkok ranged from 21.5-224.8 µg/m³ with the annual average of 78.5 µg/m³. There were 243 out of 2,282 observations representing 10.6 percent of the total observations having concentrations exceeding the standard of 120 µg /m³. The annual average concentration of 78.5 µg/m³ also exceeded the standard of 50 µg/m³. Figure 2.1 showed the increasing trend of roadside ambient PM₁₀ concentrations in Bangkok from 1999 to 2004.

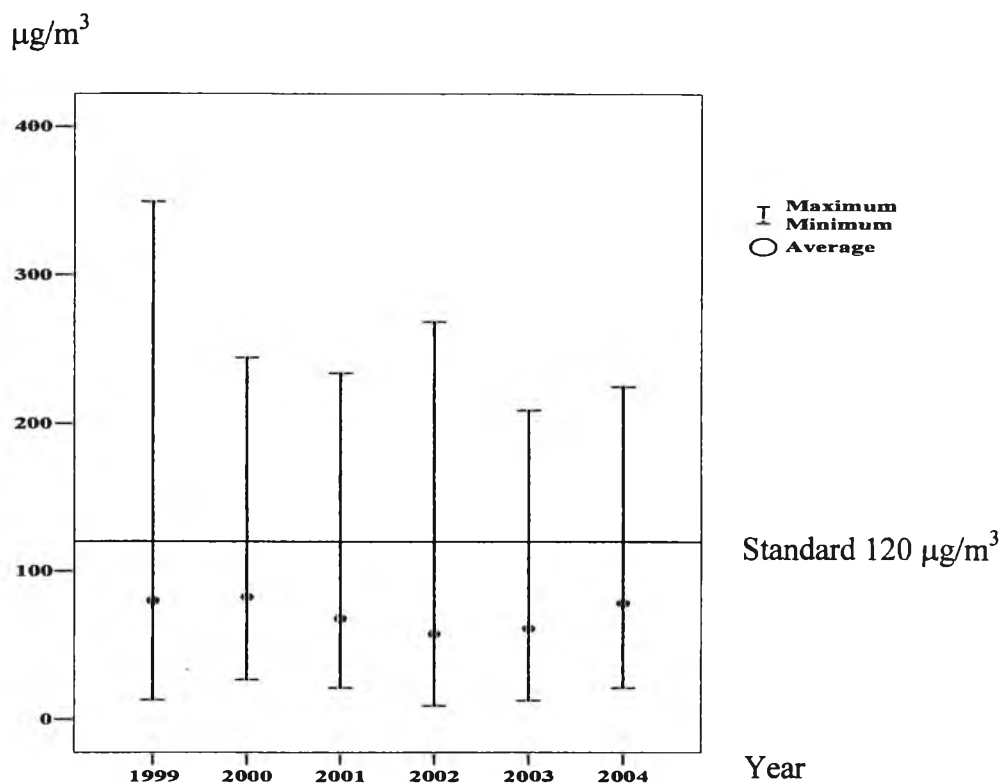


Figure 2.1 Roadside PM₁₀ concentrations in Bangkok, 1999-2004 (PCD, 2005)

2.1.2 Physico-Chemical Properties of PM₁₀

PM₁₀ make up a large proportion of particles that can be drawn deep through the alveoli in lungs. Larger particles tend to be trapped in the nose, mouth or throat. Size is not an absolute criterion as thin flakes or fibers longer than 10 micrometers may be part of a PM₁₀ sample because of their aerodynamic properties. Chemical properties of PM₁₀ vary depending on sources and chemical adsorbed. It is important to note that PM₁₀ is not one particular substance but a classification of dust by size rather than chemical properties (Australian Government, 2006).



2.1.3 Emissions and Composition of PM₁₀

The major sources of particulate matter in Bangkok are motor vehicles, roads and construction dust, industries and power plants (PCD, 2002).

2.1.3.1 Mobile Source

From vehicular registration statistics, it was found that in the year 2004 the number of vehicles registered in Bangkok was 6.3 million (Figure 2.2), increasing of 52% from 1999. The increase of vehicles in Bangkok is not proportionate to the increase of roads and has caused traffic congestion and delay in transportation. These large numbers of vehicles and traffic congestion have put severe impact on air quality of Bangkok.

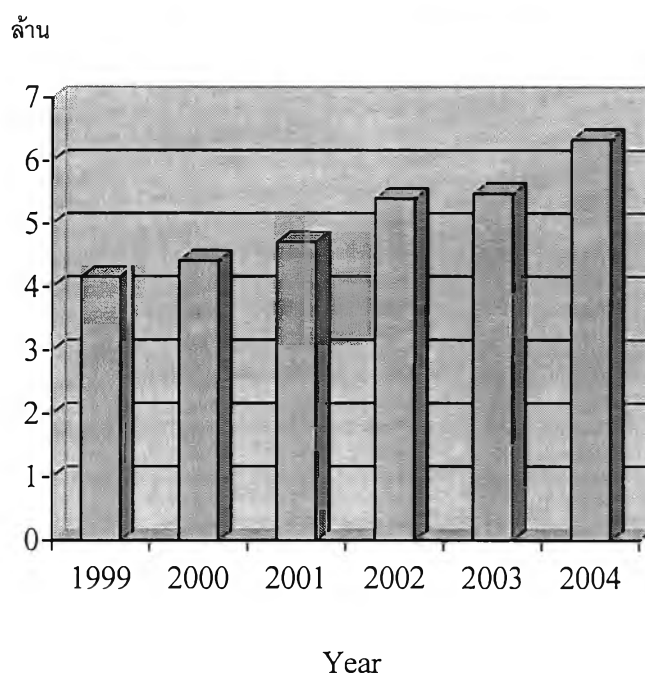


Figure 2.2 Number of motor vehicles registered in Bangkok under Motor Vehicle Act and Land Transport Act, 1999-2004.

Source: Land Transport Department, Ministry of Transport and Communications, 2004

2.1.3.2 Stationary Source

Crematoriums in Bangkok cause both significant nuisance and air quality problems because of incomplete combustion. The majority (65%) burn wood chips and charcoal, while the rest primarily burns diesel fuel. Only a few use liquefied petroleum gas (LPG). The construction of buildings and infrastructures lead to high level of dust pollution. Lack of proper planning and zoning of housing areas has aggravated the seriousness of air pollution.

Table 2.4 showed source contributions to PM₁₀ ambient concentrations in Bangkok. The table clearly showed that reentrained dust is the single most important contributor to PM₁₀ levels.

Table 2.4 Source contributions to PM₁₀ ambient concentrations

Source category	Annual average ambient concentration*			24-Hour average ambient concentration*		
	(µg/m ³)			(µg/m ³)		
	1996	2000	2005	1996	2000	2005
Background						
Reentrainment	18	17	14	52	48	39
Mobile sources	14	13	10	37	35	27
Construction	7	7	8	16	17	18
Industrial boilers	7	7	7	15	15	15
Power plants	<1	<1	<1	<1	<1	<1
Total	46	44	38	119	115	99
Hot-spots						
Reentrainment	39	37	30	118	109	89
Mobile sources	29	27	21	85	81	62
Construction	10	11	12	31	33	36
Industrial boilers	7	7	7	15	15	15
Power plants	<1	<1	<1	<1	<1	<1
Total	85	82	70	248	238	201
			*Note: Annual PM ₁₀ standard = 50			*Note: 24-Hour PM ₁₀ standard = 120

Source: Radian International, 1998

Because of the large number of sources, particulate matter may contain hundreds of different chemical elements. Fine particles may contain substantial quantities of sulfate, ammonium, nitrate, elemental carbon and condensed organic compounds. Carcinogenic compounds and heavy metals are also concentrated in these particles.

The Clean Air Act designates 188 substances, known to have harmful health effects, as hazardous air pollutants (HAPs). Table 2.5 showed lists of HAPs as defined in EPA's ambient air pollution monitoring database.

Table 2.5 Hazardous Air Pollutants

Hazardous air pollutants	Alternative names and notes
Arsenic (PM ₁₀)	Inorganic arsenic compounds (including arsine) in PM ₁₀ sample
Arsenic (PM _{2.5})	Inorganic arsenic compounds (including arsine) in PM _{2.5} sample
Beryllium (PM ₁₀)	Beryllium compounds in PM ₁₀ sample
Beryllium (PM _{2.5})	Beryllium compounds in PM _{2.5} sample
Cadmium (PM ₁₀)	Cadmium compounds in PM ₁₀ sample
Cadmium (PM _{2.5})	Cadmium compounds in PM _{2.5} sample
Chromium (PM ₁₀)	Chromium compounds in PM ₁₀ sample
Chromium (PM _{2.5})	Chromium compounds in PM _{2.5} sample
Lead (PM ₁₀)	Lead compounds in PM ₁₀ sample
Lead (PM _{2.5})	Lead compounds in PM _{2.5} sample
Manganese (PM ₁₀)	Manganese compounds in PM ₁₀ sample
Manganese (PM _{2.5})	Manganese compounds in PM _{2.5} sample
Mercury (PM ₁₀)	Mercury compounds in PM ₁₀ sample
Mercury (PM _{2.5})	Mercury compounds in PM _{2.5} sample
Nickel (PM ₁₀)	Nickel compounds in PM ₁₀ sample
Nickel (PM _{2.5})	Nickel compounds in PM _{2.5} sample

Source: <http://www.EPA AirData - About AQS Hazardous Air Pollutants.htm>

Radian International (1998) reported the compositions of emission sources in Bangkok (Table 2.6). The table showed that the compositions of variety emission sources consist of the heavy metal as hazardous air pollutants.

Table 2.6 Compositions of emission sources in Bangkok area (% mass \pm uncertainty)

	Heavy duty truck	Light duty truck	Motorcycle	Soil	Road dust	Steel mill	Power plant
EC	18.181 \pm 2.243	61.758 \pm 13.423	3.440 \pm 5.220	1.199 \pm 0.752	1.145 \pm 0.478	4.379 \pm 0.716	0.000 \pm 0.300
OC	79.647 \pm 2.326	15.616 \pm 4.537	34.327 \pm 11.950	8.117 \pm 0.590	11.909 \pm 2.531	5.130 \pm 0.957	0.000 \pm 0.300
NH ₄ ⁺	0.000 \pm 0.3004	0.000 \pm 0.300	0.000 \pm 0.300	0.001 \pm 0.000	0.002 \pm 0.002	0.052 \pm 0.073	0.018 \pm 0.005
Cl ⁻	0.000 \pm 0.300	0.000 \pm 0.300	0.000 \pm 0.300	0.118 \pm 0.072	0.127 \pm 0.047	0.442 \pm 0.625	0.045 \pm 0.014
NO ₃ ⁻	0.000 \pm 0.300	0.000 \pm 0.300	0.000 \pm 0.300	0.014 \pm 0.007	0.015 \pm 0.017	0.209 \pm 0.296	0.000 \pm 0.300
SO ₄ ²⁻	0.000 \pm 0.300	0.000 \pm 0.300	0.000 \pm 0.300	0.690 \pm 0.451	0.228 \pm 0.084	0.746 \pm 1.055	35.300 \pm 10.590
Al	0.098 \pm 0.044	0.028 \pm 0.017	0.022 \pm 0.012	9.370 \pm 1.330	7.877 \pm 0.998	0.470 \pm 0.197	0.025 \pm 0.008
Si	0.660 \pm 0.208	0.431 \pm 0.241	0.124 \pm 0.113	22.950 \pm 2.850	20.133 \pm 0.946	1.923 \pm 0.598	0.104 \pm 0.031
P	0.091 \pm 0.077	0.018 \pm 0.011	0.002 \pm 0.003	0.037 \pm 0.016	0.067 \pm 0.037	0.002 \pm 0.002	0.000 \pm 0.300
S	0.289 \pm 0.314	0.365 \pm 0.168	0.113 \pm 0.076	0.478 \pm 0.190	0.423 \pm 0.122	0.198 \pm 0.101	2.920 \pm 0.876
Cl	0.024 \pm 0.018	0.014 \pm 0.008	0.016 \pm 0.013	0.166 \pm 0.104	0.264 \pm 0.131	0.815 \pm 0.179	0.000 \pm 0.300
K	0.106 \pm 0.027	0.001 \pm 0.002	0.009 \pm 0.007	1.500 \pm 0.060	1.827 \pm 0.327	0.453 \pm 0.008	0.010 \pm 0.003
Ca	0.289 \pm 0.062	0.055 \pm 0.027	0.087 \pm 0.078	17.450 \pm 1.250	20.200 \pm 2.337	3.133 \pm 0.342	0.062 \pm 0.019
Ti	0.026 \pm 0.023	0.002 \pm 0.002	0.003 \pm 0.003	0.295 \pm 0.057	0.267 \pm 0.034	0.080 \pm 0.019	0.000 \pm 0.000
V	0.004 \pm 0.003	0.002 \pm 0.001	0.002 \pm 0.005	0.012 \pm 0.003	0.012 \pm 0.004	0.007 \pm 0.001	1.070 \pm 0.321
Cr	0.000 \pm 0.300	0.001 \pm 0.000	0.002 \pm 0.005	0.011 \pm 0.01	0.013 \pm 0.003	0.043 \pm 0.018	0.010 \pm 0.003
Mn	0.001 \pm 0.001	0.001 \pm 0.001	0.001 \pm 0.001	0.078 \pm 0.022	0.075 \pm 0.017	0.606 \pm 0.284	0.003 \pm 0.001
Fe	0.349 \pm 0.181	0.066 \pm 0.044	0.017 \pm 0.018	3.685 \pm 0.685	3.520 \pm 0.406	15.700 \pm 3.034	0.123 \pm 0.037
Cu	0.014 \pm 0.005	0.004 \pm 0.002	0.003 \pm 0.003	0.016 \pm 0.004	0.030 \pm 0.018	0.106 \pm 0.030	0.002 \pm 0.001
Ni	0.003 \pm 0.002	0.001 \pm 0.001	0.001 \pm 0.002	0.003 \pm 0.000	0.003 \pm 0.000	0.033 \pm 0.010	0.548 \pm 0.164
Zn	0.086 \pm 0.098	0.039 \pm 0.009	0.022 \pm 0.020	0.036 \pm 0.011	0.092 \pm 0.050	3.913 \pm 2.295	0.003 \pm 0.001
As	0.000 \pm 0.300	0.001 \pm 0.001	0.001 \pm 0.002	0.003 \pm 0.001	0.001 \pm 0.002	0.000 \pm 0.300	0.004 \pm 0.001
Br	0.005 \pm 0.002	0.000 \pm 0.001	0.001 \pm 0.003	0.002 \pm 0.001	0.001 \pm 0.000	0.011 \pm 0.004	0.000 \pm 0.300
Ba	0.102 \pm 0.116	0.000 \pm 0.300	0.068 \pm 0.133	0.037 \pm 0.005	0.036 \pm 0.017	0.033 \pm 0.012	0.015 \pm 0.004
Pb	0.027 \pm 0.010	0.003 \pm 0.003	0.011 \pm 0.012	0.012 \pm 0.005	0.024 \pm 0.012	0.448 \pm 0.244	0.001 \pm 0.000

Source: Radian International, 1998

Polycyclic Aromatic Hydrocarbons (PAHs) are a group of organic compounds made up of two or more fused benzene rings in linear, angular or cluster arrangements. Different arrangements of the rings have resulted in the identification of over 200 different compounds. Some of PAHs are carcinogenic compounds, particularly, benzo(a)pyrene (BaP) is a very strongly carcinogenic (IARC, 1983). Thongsanit, *et al.* (2003) reported the highest and lowest concentrations of PAHs in Bangkok were 195 ng/m³ at Chulalongkorn University and 6 ng/m³ at Bangkok University. Benzo(e)pyrene, indeno(123cd)pyrene, and benzo(ghi) perylene were the major compounds with average concentrations of 8, 10, and 13 ng/m³, respectively. 97% of PAHs were found in the small particulate size range of ~ 0.95 μm.

2.2 Respiratory System

2.2.1 Structure and Function of the Lung

Respiratory system consists of nose, nasal cavity, pharynx, larynx, trachea, bronchus, bronchioles, and lungs. There may be divided into upper and lower respiratory tract (CCOHS, 2006) (Figure 2.3).

Lungs lie within thorax, protected by rib cage. Ribs offer support to intercostals muscles and diaphragm. It is the action of these muscles that enlarges the chest during normal breathing. The main function of lungs is rapid gas exchange. This is accomplished by a well-coordinated interaction of lungs with central nervous system, diaphragm and chest wall musculature, and circulatory system (Godwin, 2006).

Gas exchange occurs in alveolus where thin laminar blood flow and inspired air are separated only by a thin tissue layer. Gas exchange takes 0.25 seconds or 1/3 of the total transit time of a red cell. The entire blood volume of the body passes through the lungs each minute in the resting state, which is 5 liters per minute. The total surface area of the lung is about 80 meters square, equivalent to the size of a tennis court (Godwin, 2006).

Only about 10% of the lung is occupied by solid tissue, whereas the remainder is filled with air and blood. Supporting structures of the lung must be delicate to allow gas exchange, yet strong enough to maintain architectural integrity, that is sustain alveolar structure. The functional structure of the lung can be divided into (1) the conducting airways (dead air space), and (2) the gas exchange portions. The two plumbing systems are: airways for ventilation, and the circulatory system for perfusion. Both are under low pressure (Godwin, 2006).

Total lung weight is about 300-400 g. Upper and middle lobes are anterior, while the lower lobes are posterior. Development of each lobe results in division into 19 bronchopulmonary segments which are relatively constant and which often have pathophysiologic correlates, i.e. secondary tuberculosis is seen in the apical segments (Godwin, 2006).

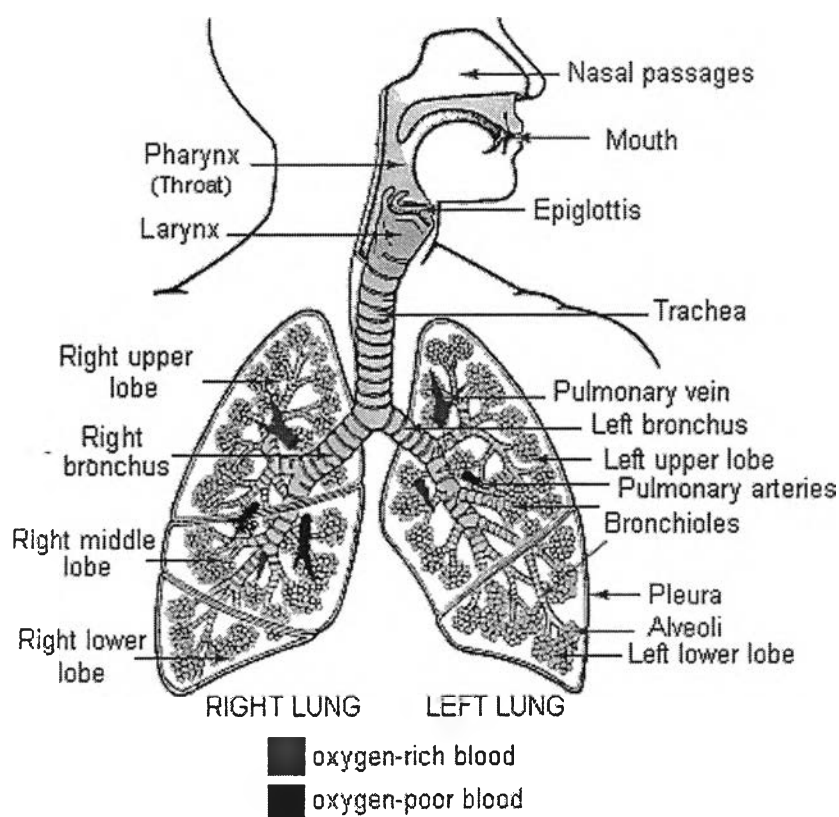


Figure 2.3 Passage of air and the respiratory structures

Source: <http://www.sirinet.net/~igjohnso/respiratory.html>

2.2.2 Development of the lungs

1. The bronchi grow and branch during the glandular period, which last until approximate the 17th fetal weeks.
2. Bronchi and bronchioles expand and branch during the canalicular period. Respiration becomes possible towards the end of this period around the 25th fetal weeks.
3. The number of terminal sacs increases during the initial part of the alveolar period. Alveolar sacs continue to be formed during early childhood (up to year 8) and mature into alveoli.

2.2.3 Defense Mechanisms of the Lung

Protection against particles is at four levels (Godwin, 2006):

1. Upper airway filter

Filtering mechanisms in the nasal cavity trap and eliminate so that larger particles over 15 μm in diameter hit the surface and are carried in the mucus to the pharynx and swallowed. If the particles are irritated or cause an allergic reaction, two reflexes are sneezing and coughing.

2. Lower airway filter

This level lined by mucociliary epithelium acts as a low resistance filter which removes nearly all of the particles down to about 5 μm in diameter. The particles are carried in the mucus back to the larynx, join the particles from the upper airways and are swallowed or expectorated.

3. Macrophage clearance to the airways

Particles less than 0.5 μm in diameter getting beyond the mucociliary system onto the lining of the alveolar ducts and alveoli and may be retained. Macrophages move out from the wall and engulf the particles, moving back in when fully loaded.

4. Macrophage segregation and clearance via the lymphatics

When the overload of the particles is very heavy then the macrophages damages or cell die. The other macrophages attempt to carry the particles to the hilar lymph nodes either via the lymphatics in the interlobular septa and under the pleura or those along the blood vessels (Figure 2.4).

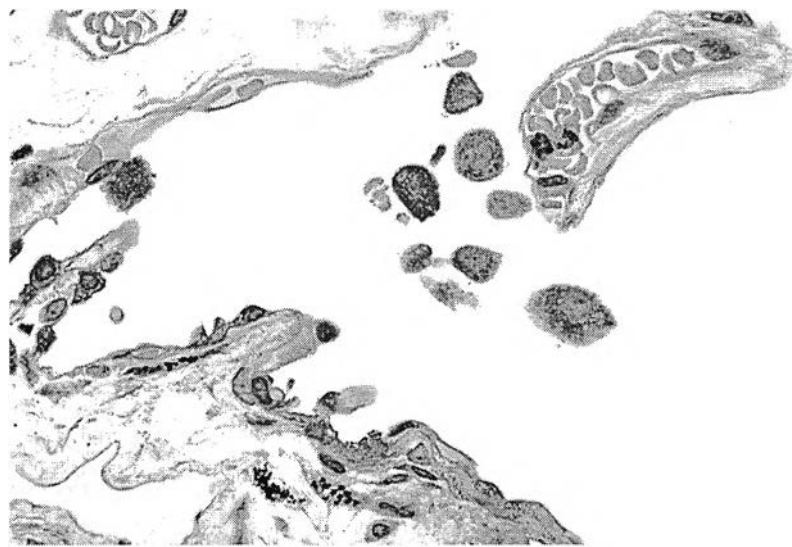


Figure 2.4 Alveolar macrophages of human lung

Source: <http://www.bu.edu/histology/p/1390600a.htm>

Figure 2.5 showed range of the particles size. Particles may be placed into several categories such as dust, ash, fume, smoke, and secondary particles. Dust particles are defined as particles of mechanical or biological origin including soil, sea spray, spores, pollen, and bacteria. Smoke describes carbonaceous residues from incomplete combustion processes. Secondary particles are formed in the atmosphere due to reaction of gases. Mists or fogs are liquid droplets in the atmosphere and fumes result from gaseous materials which nucleate in the atmosphere to form larger aggregates.

It has been difficult to separate the effects of the particles from cigarette smoking and environment pollutants. However, the severity of the damage depends on the combined effect of the toxicity and the level of exposure.

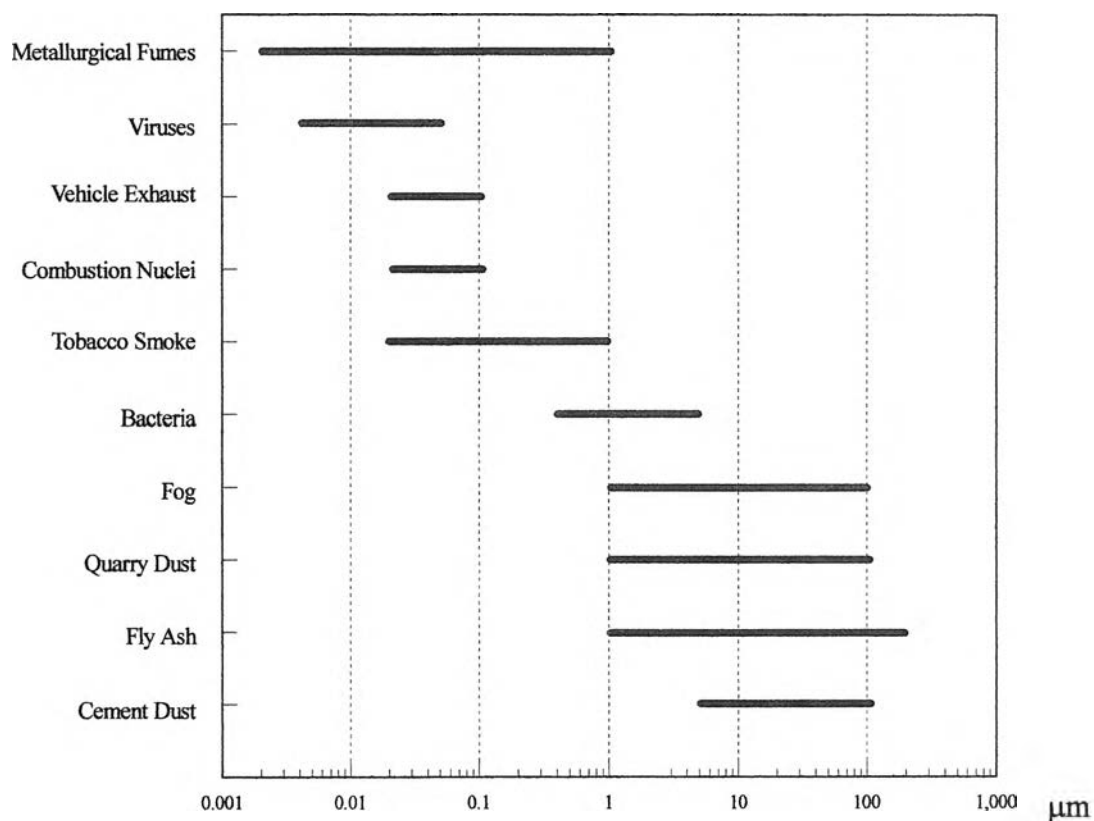


Figure 2.5 Range of particle size (EPA, 1991)

2.2.4 Lung Dosimetry

Four processes that the particulates deposited in the lungs (CCOHS, 2006):

1. Interception: particle is deposited when it travels so close to a surface of the airway passages that an edge of the particle touches the surface.
2. Impaction: particles having an aerodynamic diameter of 5 to 30 μm are deposited in the nasopharyngeal region.
3. Sedimentation: small particles with an aerodynamic diameter of about 1 to 5 μm are deposited in the tracheobronchial region.
4. Diffusion: small particles with an aerodynamic diameter of about 0.5 μm are deposited in the alveolar region.

2.2.5 Spirometry

Spirometry is the most commonly used in pulmonary function tests (PFTs), which is an important tool in the diagnosis, assessment, and management of respiratory diseases. It remains the most reliable tests for cross-sectional surveys. The reliability of spirometry depends on quality of the equipment, standardized methodology, particularly regarding how quickly the subject increases flow at the beginning of the expiration; sustained effort throughout the expiration; duration of the expiratory maneuver; and repeatability (ATS, 1995). Steps of spirometry standardization were shown in Figure 2.6.

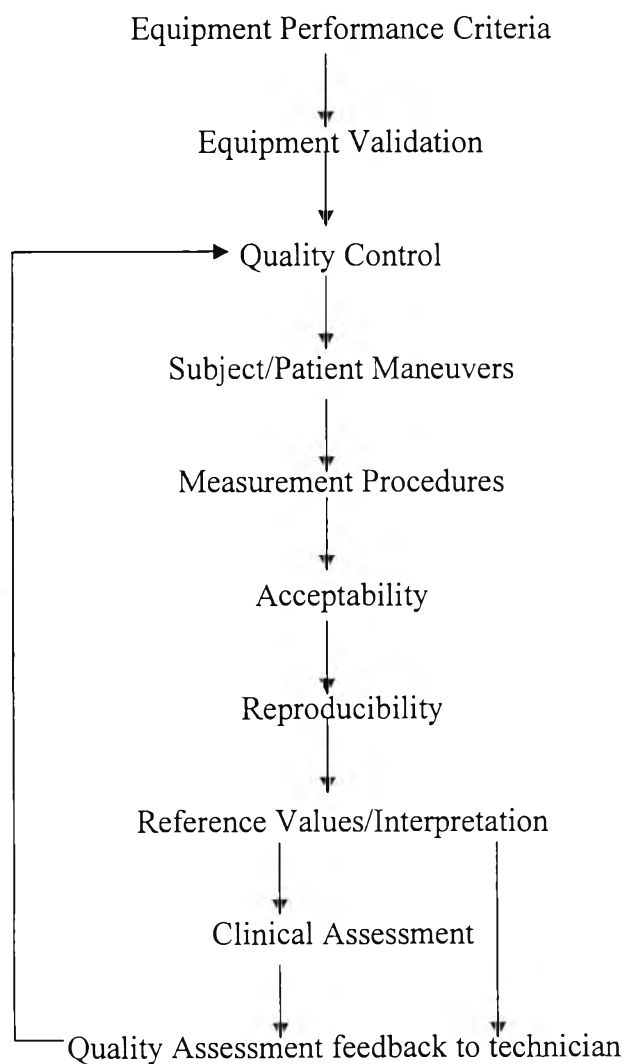


Figure 2.6 Spirometry standardization steps (ATS, 1995)

2.2.5.1 Equipment Performance Criteria

Instrumentation recommendations should be followed to provide accurate spirometric data and information that are comparable from laboratory to laboratory and from one time period to another. The accuracy of a spirometry system depends on the resolution (i.e., the minimal detectable volume or flow) and linearity of the entire system, from volume or flow transducer to recorder, display, or processor. The equipment recommendations for diagnostic spirometry are summarized in Table 2.7 (ATS, 1995).

Table 2.7 Minimal recommendations for diagnostic spirometry

Test	Range/Accuracy (BTPS)	Flow range (L/s)	Time (s)	Resistance and back pressure	Test signal
VC	0.5 to 8 L \pm 3% of reading or \pm 0.050 L, whichever is greater	Zero to 14	30		3-L Cal syringe
FVC	0.5 to 8 L \pm 3% of reading or \pm 0.050 L, whichever is greater	Zero to 14	15	Less than 1.5 cm H ₂ O/L/s	24 standard waveforms 3-L Cal syringe
FEV ₁	0.5 to 8 L \pm 3% of reading or \pm 0.050 L, whichever is greater	Zero to 14	1	Less than 1.5 cm H ₂ O/L/s	24 standard waveforms
Time zero	The time point from which all FEV ₁ measurements are taken			Back extrapolation	
PEF	Accuracy: \pm 10% of reading or \pm 0.400 L/s, whichever is greater Precision: \pm 5% of reading or \pm 0.200 L/s	Zero to 14		Same as FEV ₁	26 flow standard waveforms
FEF _{25-75%}	7.0 L/s \pm 5% of reading or \pm 0.200 L/s, whichever is greater	\pm 14	15	Same as FEV ₁	24 standard waveforms
V	\pm 14 L/s \pm 5% of reading or \pm 0.200 L/s, whichever is greater	Zero to 14	15	Same as FEV ₁	Proof from manufacturer
MVV	250 L/min at TV of 2 L within \pm 10% of reading or \pm 15 L/min, whichever is greater	\pm 14 \pm 3%	12 to 15	Pressure less than \pm 10 cm H ₂ O at 2-L TV at 2.0 Hz	Sine wave pump

Source: ATS, 1995

2.2.5.2 Equipment Validation

The accuracy validation limits (tolerance for simulator systems is included in these limits) for volume are: volume (Forced vital capacity: FVC, FEV₁) \pm 3.5% of reading or \pm 0.070 L, whichever is greater; and average flow (FEF_{25-75%}) \pm 5.5% of reading or \pm 0.250 L/s, whichever is greater. The error range is expanded from the earlier ATS spirometry recommendation to allow for errors associated with mechanical syringes. The precision validation limits are: volume (FVC, FEV₁) 3.5% (range percent) or 0.100 L, whichever is greater; and flow (FEF_{25-75%}) 5.5% or 0.250 L/s, whichever is greater. Mechanical syringes used for validation must be accurate within \pm 0.025 L for FVC and FEV₁ and \pm 0.100 L/s for FEF_{25-75%} (ATS, 1995).

2.2.5.3 Quality Control

It is important to ensure that the laboratory is consistently meeting appropriate standards (ATS, 1995).

1. Technician's role in quality control

In any quality control program, an important element is a procedures manual containing calibration procedures, test performance procedures, calculations, criteria, reference values source, and action to be taken when panic values are observed. Technician should maintain records of continuing education and the results of evaluation and feedback provided by the medical director. This feedback should include, at a minimum: (1) information concerning the nature and extent of unacceptable FVC maneuvers and non-reproducible tests; (2) correction action the technician can take to improve the quality and number of acceptable maneuvers; and (3) recognition for superior performance by the technician in obtaining good maneuvers from challenging subjects.



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2. Hygiene and infection control

The major goal of infection control is to prevent infection transmission to subjects and staff during pulmonary function testing.

3. Equipment quality control

Attention to good equipment quality control and calibration is an important part of good laboratory practice (Table 2.8).

Table 2.8 Equipment quality control summary

Test	Minimum interval	Action
Volume	Daily	3-L syringe check
Leak	Daily	3 cm H ₂ O constant pressure for 1 min
Linearity	Quarterly	1-L increments with a calibrating syringe
	Weekly (flow spirometers)	measured over entire volume range (flow spirometers simulate several different flow ranges)
Time	Quarterly	Mechanical recorder check with stopwatch
Software	New versions	Log installation date and perform test using known subject

Source: ATS, 1995

2.2.5.4 Subject Instruction and Maneuver Performance

Technician should demonstrate the appropriate technique (ATS, 1995) as follows:

1. Check spirometer calibration
2. Explain test
3. Prepare subject by ask about smoking, recent illness, medication use, etc.
4. Instruct and demonstrate test to subject
 - Correct posture with head elevated
 - Inhale completely

- Position mouthpiece (open circuit)
 - Exhale with maximal force
5. Perform maneuver
- Have subject assume correct posture
 - Attach nose clip
 - Inhale completely; the inhalation should be rapid but not forced
 - Place mouthpiece in mouth and close lips around mouthpiece
 - Exhale maximally as soon as lips are sealed around mouthpiece
 - Repeat instructions as necessary, coaching vigorously
 - Repeat for a minimum of three maneuvers; no more than eight are usually required
 - Check test reproducibility and perform more maneuvers as necessary

2.2.5.5 Measurement Procedures

Spirometric variables should be measured from a series of at least three acceptable forced expiratory curves. The largest VC should be reported from all acceptable curves, including the forced maneuvers (FVC). The largest FVC and the largest FEV₁ (BTPS) should be recorded after examining the data from all of the acceptable curves, even if they do not come from the same curve. Other measures, such as the FEF_{25-75%} and the instantaneous expiratory flows, should be obtained from the single curve that meets the acceptability criteria and gives the largest sum of FVC plus FEV₁ (best test) (ATS, 1995).

2.2.5.6 Acceptability

Acceptability criteria for FVC measurements, individual spirometry are acceptable if they are free from artifacts such as cough or glottis closure during the first second of exhalation, early termination or cutoff, variable effort, leak, and obstructed mouthpiece. Have good starts through extrapolated volume less than 5% of FVC or 0.15 L, whichever is greater or time-to-PEF (Peak expiratory flow) of less

than 120 ms (optional unit further information is available). Have a satisfactory exhalation by 6 s of exhalation and/or a plateau in the volume-time curve or reasonable duration or a plateau in the volume-time-curve of if the subject cannot or should not continue to exhale (ATS, 1995).

2.2.5.7 Reproducibility

Reproducibility criteria for FVC measurements, after three acceptable spiromograms have been obtained, apply the following tests: Are the two largest FVC within 0.2 L of each other? Are the two largest FEV₁ within 0.2 L of each other? If both of these criteria are met, the test session may be concluded. If both of these criteria are not met, continue testing until both of the criteria are met with analysis of additional acceptable spiromograms or a total of eight tests have been performed or the subject cannot or should not continue and save at a minimum the three best maneuvers (ATS, 1995).

2.2.5.8 Reference Values/Interpretation

The procedures for interpretation and reference values may legitimately vary from laboratory to laboratory depending upon geographic location and the characteristics of the population being tested (ATS, 1995).

The sources of variation in lung function such as:

1. Technical: instrument, subject, posture, observer, procedure, temperature, altitude, etc.
2. Personal factors, including size, age, gender, physical activity, muscularity, race, etc.
3. Environmental factors, including tobacco smoke (personal and environment), occupation, residence (urban or rural), air pollution (home, environmental), and socioeconomic status.

Figure 2.7 showed lung volume and ventilatory capacity. Figure 2.8 showed normal flow volume loop. The parameters of spirometry are:

1. FVC: Maximal volume of air exhaled with maximally forced effort (perform as rapidly and completely as possible) from a position of maximal inspiration, i.e., vital capacity performed with a maximally forced expiratory effort, expressed in liters.
2. FEV₁ (Forced expiratory volume in one second): The volume of air exhaled during the first second of the performance of the FVC, expressed in liters.
3. FEF_{25-75%}: Mean forced expiratory flow during the middle half of the FVC.

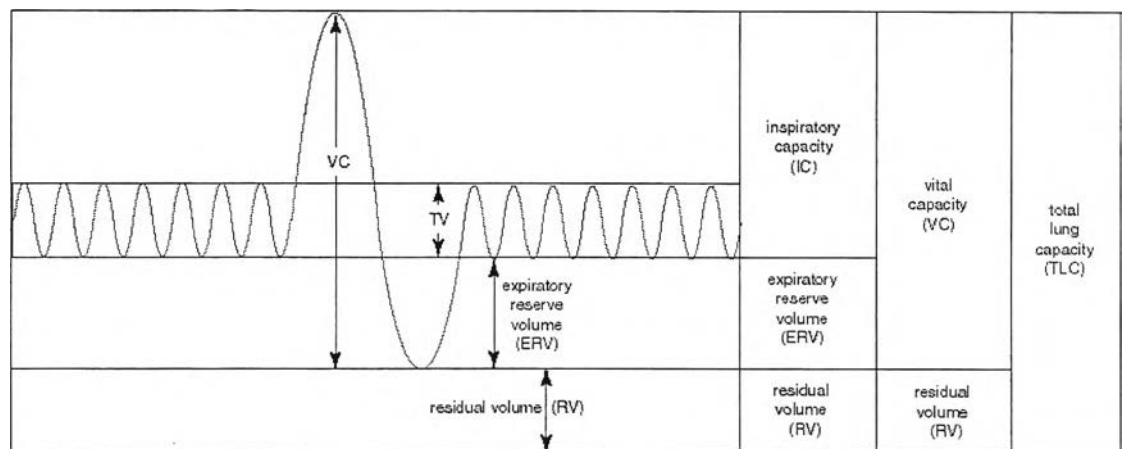


Figure 2.7 Lung volume, shown by block diagrams (*right*) and by a spirographic tracing (*left*). TV = tidal volume.

Source: http://en.wikipedia.org/wiki/Lung_volumes

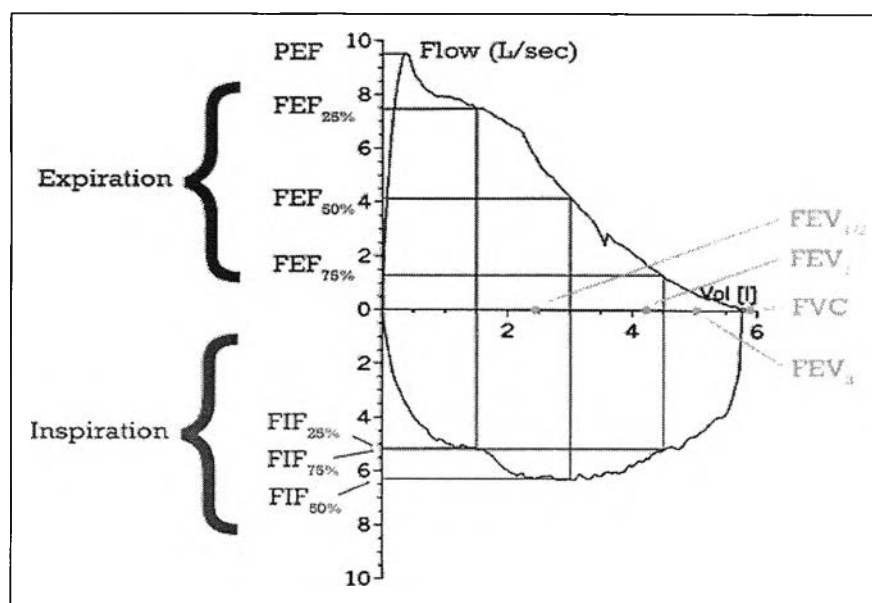


Figure 2.8 Normal flow volume loop

Source: <http://en.wikipedia.org/wiki/Spirometry>

Indications and uses of spirometry (ATS, 1995) as follows:

1. Diagnostic

1.1 To evaluate symptoms, signs, or abnormal laboratory tests

- Symptoms: dyspnea, wheezing, orthopnea, cough, phlegm production, chest pain
- Signs: diminished breath sounds, overinflation, expiratory slowing, cyanosis, chest deformity, unexplained crackles
- Abnormal laboratory tests: hypoxemia, hypercapnia, polycythemia, abnormal chest radiographs

1.2 To measure the effect of disease on pulmonary function

1.3 To screen individuals at risk of having pulmonary diseases such as smokers, individuals in occupations with exposure to injurious substances, and some routine physical examinations

1.4 To assess preoperative risk

1.5 To assess prognosis (lung transplant, etc.)

1.6 To assess health status before enrollment in strenuous physical activity programs

2. Monitoring

2.1 To assess therapeutic interventions

- Bronchodilator therapy
- Steroid treatment for asthma, interstitial lung disease, etc.
- Management of congestive heart failure
- Other (antibiotics in cystic fibrosis, etc.)

2.2 To describe the course of diseases affecting lung function

- Pulmonary diseases such as obstructive airway diseases and interstitial lung diseases
- Cardiac diseases such as congestive heart failure
- Neuromuscular diseases such as Guillain-Barre Syndrome

2.3 To monitor persons in occupations with exposure to injurious agents

2.4 To monitor for adverse reactions to drugs with known pulmonary toxicity

3. Disability/impairment evaluations

3.1 To assess patients as part of a rehabilitation program such as medical, industrial, and vocational

3.2 To assess risks as part of an insurance evaluation

3.3 To assess individuals for legal reasons such as social security or other government compensation programs, personal injury lawsuits, and others

4. Public health

4.1 Epidemiologic surveys

- Comparison of health status of populations living in different environments
- Validation of subjective complaints in occupational/ environmental settings

4.2 Derivation of reference equations

2.3 Health Effects

Both coarse and fine particles are of health concern because they can penetrate into the sensitive regions of the respiratory tract. Fine particles are of greatest concern because they are linked to the most serious effects. They can be deeply inhaled into the lungs where they can be absorbed into the bloodstream or remain embedded for long periods of time. They can cause persistent coughs, phlegm, wheezing, and physical discomfort.

Several recently published community health studies indicate that significant respiratory and cardiovascular-related problems are associated with exposure to particle levels well below the existing particulate matter standards (Aekplakorn *et al.*, 2003b; Duanping *et al.*, 2004; Karita *et al.*, 2001; Karita *et al.*, 2004; Ostro *et al.*, 1999; and Tamura *et al.*, 2003). These negative effects include premature death, hospital admissions from respiratory causes, and increased respiratory symptoms. Short-term exposure to coarse particulate matter can lead to coughing, minor throat irritation and a reduction in lung function. Long-term exposure to particulate matter may increase the rate of respiratory and cardiovascular illness and reduce life span.

For environmental and occupational lung disease, the changes which occur in the lung are: (1) the variety of potentially pathologic agents, (2) the diversity of possible pathologic responses in the lung, and (3) the numerous occupations in which exposure can occur. Occupational lung diseases occur as a result of inhalation of noxious particles, fumes, and gases. A person inhales about 10,000 liters of air per day and that this air is frequently polluted.

Injury by types of particles, fumes, or gases (Godwin, 2006) is:

1. Organic dusts tend to produce allergic reactions.
2. Smoke and noxious fumes damage the conducting airways and/or cause acute diffuse alveolar damage (DAD).

3. Inorganic dusts, if small enough, penetrate into the periphery of the lungs and are associated with chronic disease resulting in fibrosis and occasionally neoplasia.

Factors that contribute to lung disease (Godwin, 2006) are:

1. Size of particle: larger particles get trapped higher in the airways and are more easily eliminated. Smaller particles can get further out into the acini.
2. Solubility of gases: soluble gases tend to cause injury in the upper airways and tend to be more irritating, insoluble gases will get to the peripheral lung and cause edema.
3. Duration of exposure: usually the longer the exposure the more severe the disease.

Types of reactions in upper airways and lower air spaces (Godwin, 2006) are:

1. Acute

- 1.1 In upper airways soluble gases (e.g. chloride and ammonia) will cause direct injury to the epithelium, which will result in an acute inflammatory reaction followed by scarring with prolonged exposure. Hypersensitivity reactions (asthma), immediate and delayed, also occur in the upper airways, e.g. Farmer's Lung and Humidifiers Lung.

- 1.2 In the distal airspaces, acute reactions follow exposure to insoluble gases, some metals, and fumes. This manifests as acute edema or acute diffuse alveolar damage.

2. Sub acute

- 2.1 Hypersensitivity pneumonia

- 2.2 Alveolar filling processes and accelerated fibrosis

3. Chronic

Include the development of fibrosis, granuloma formation, carcinoma, and bronchitis. Also leads to increased incidence of infections, such as tuberculosis. These changes can occur anywhere in the respiratory tract.

Types of pathologic responses or changes in the lung (Godwin, 2006) are:

1. Bronchial Asthma, which is a state of hyperirritability of the conducting airways. There is spasm of the bronchial smooth muscle, which causes narrowing of the airways and increased mucus production and edema of the bronchial mucosa. There consists of two types:

1.1 Atopic/allergic asthma, which includes occupational asthma as a subset. People who develop allergic-type asthma have a hyperirritable conducting airway system and develop a combination of immediate type I-IgE and delayed type III-IgG immune reactions. These two factors make them susceptible to asthmatic attacks. Many environmental agents can cause occupational asthma.

1.2 Non-atopic asthma usually related to viral infection.

2. Small Airways Disease

Pathologically manifests as thickening of the walls of the respiratory and terminal bronchioles. Fibrous and muscular thickening that causes constriction of the lumen. Associated with smoking; also, some people feel that it may be associated with asbestos exposure.

3. Restrictive lung disease

Over 150 different disease processes cause infiltrative or restrictive lung disease. In general, most of these processes affect the alveolar wall and as such can be contrasted with obstructive lung disease, which primarily affects the conducting airways. Injury often involves not only the

components of the interstitium, but also the alveolar epithelium and capillary endothelium. The functional changes reflect a restriction of airflow manifest by reduced expansion of the lung parenchyma, decreased total lung capacity, decreased vital capacity, decreased residual volume, and decreased lung compliance.

There are many differences between children and adults in the ways that they respond to air pollution (Kleinman, 2006). For example, children take in more air per unit body weight at a given level of exertion than do adults. When a child is exercising at maximum levels, they may take in 20 to 50 percent more air and more air pollution than would an adult in comparable activity. Children also spend more time outside than adults. The average adult, except for those who work mostly outdoors, spends most of their time indoors at home and work. Children are often outdoors during periods when air pollution is at its highest. The typical adult spends 85 to 95 percent of their time indoors, while children may spend less than 80 percent of their time indoors. Children may also exert themselves harder than adults when playing outside. The most important difference between adults and children is that children are growing and developing. Along with their increased body size, children's lungs and immune system are growing and changing.

2.4 Epidemiology

Epidemiology is the study of the determinants, occurrence, and distribution of health and disease in a defined population. Infection is the replication of organisms in host tissue, which may cause disease. A carrier is an individual with no overt disease who harbors infectious organisms. Dissemination is the spread of the organism in the environment (Brachman, 2006).

There are two types of epidemiological study: experiments and observational studies. In experiments (clinical trials, intervention studies, and randomized controlled trials) the investigator controls which treatment subjects receive, usually by random allocation. For harmful interventions (e.g. cigarette smoking) randomization is

unethical so it is necessary to observe the results of natural treatment allocations in observational studies. Thus observational studies are used to examine how exposure to risk factors influences the probability of developing disease. Observational studies are likely to be influenced by selection bias; randomization in controlled trials is designed to overcome selection bias. There are three types of observational study; cross-sectional (prevalence); cohort (longitudinal); case-control. There are several types of risk factor such as genetic, environment, social, and life style (PHS, 2006).

2.4.1 Cross-sectional Studies

Cross-sectional studies are widely used in epidemiological studies. It is used to estimate the prevalence of diseases or the prevalence of exposure to risk factors or both. The following steps have to be completed:

1. Define target population: usually defined by age, gender and place of residence.
2. Take sample: this requires a population register as a sampling frame.
3. Devise case-definition: define fixed criteria by which the condition is identified.
4. Complete case-ascertainment: apply the case-definition to the sample, obtaining a high response rate.
5. Analysis: estimate the prevalence rate.

For example, cigarette smoking is associated with the development of lung cancer. This did not necessarily prove that smoking caused lung cancer because some criteria used to decide whether an association is causal (PHS, 2006):

1. Strength: strong associations likely to be causal.
2. Consistency: the observation repeatedly confirmed in different persons, in different places, at different times.
3. Temporality: cause before effect.

4. Dose response relationship: increasing risk of disease with increasing exposure to risk factor.
5. Biological plausibility: fits in with biological knowledge about mechanism.
6. Coherence: fits in with other evidence e.g. secular trends in lung cancer mortality.
7. Analogy: cause and effect already established for similar exposure and disease.
8. Specificity: one cause, one effect.
9. Experiment: e.g. removing cause reduces effect.

A bias is a factor that leads to results, which show a systematic departure from the truth. Random error affects all observations equally and can be overcome by increasing the sample size. Bias results from factors, which have different effects on diseased and undiseased or on, exposed and unexposed. There are two main sources of bias: selection bias and measurement bias.

In cross-sectional studies, data on exposure and health outcome are collected at the same time. The association between exposure and prevalence of the health outcome is studied. These studies may use the current exposure situation and sometimes, also past exposure. In the latter case, the same methods as used in retrospective studies may be applied. Direct measurements of current exposure are often convenient to perform and, in fact, are an advantage of the cross-sectional study design. A major disadvantage of concurrent assessment of exposure and health is the inability to establish the temporal sequence of exposure and health outcome. This is particularly true for situations where a disease may cause a change in the exposure pattern, for example asthmatics tend to give up smoking due to the disease.

2.4.2 Respiratory Questionnaires

The Committee on Standards for Epidemiology Surveys in Chronic Respiratory Disease of the ATS devised the ATS-DLD-78-C version of the children's

questionnaire, which is tentatively recommended as a standard questionnaire for studies of children in epidemiologic investigations designed to assess respiratory symptoms of children by questioning responsible adults. Representatives from other groups, such as the American College of Chest Physicians (ACCP) and the National Institute of Occupational Safety and Health (NIOSH), were invited to send representatives. Others were also consulted during the contract period. The questionnaire to be developed would be for completion by the parent or guardian of children. The questionnaire is divided into 3 components are demographic data, respiratory symptoms, and family history (Ferris, 1978).

Historical factors that were considered important in the development of chronic respiratory diseases were included in the questionnaire. A number of these factors are considered as risk factors in themselves; a number are confounding variables that conceivably could be associated with other factors being tested as well as with the development of disease. Rationales for the children's questionnaire (Ferris, 1978) are:

Page 2-5 (Q 1-13): Demographic data

The chronologic residence history provides information on both places of residence and the number of years at the current address. Both may be useful indices of environmental exposure. Risk of exposure to infectious agents varies with contact with other children. Therefore, knowledge about exposure to other children, particularly for younger children, and the numbers of persons in a household may be useful variables. Exposure to animal hair and feathers may be related to an allergic diathesis.

Page 5-8 (Q 14-16, 20-22): Cough and Congestion and/or Phlegm

These questions are designed to determine the presence of cough and/or phlegm and to obtain a measure of its chronic.

Page 6-7 (Q17-19): Wheezing

Wheezing independent of the diagnosis of asthma appears to be an important predictor of poorer pulmonary function.

Page 8-10 (Q23-33): Other Illnesses

A history of eczema before age 2 is commonly associated with subsequent development of asthma. This question is, therefore, more useful in predicting asthma in younger (less than 8 years old) than in older children, who are likely to have already developed their asthma and in whom preferential recall would be more strongly exhibited. Similarly, the significance of middle ear infections is very different for different aged children. The question on external ear infections is included to lessen the confusion about what is considered a middle ear infection. The question regarding leaving the hospital with the mother is designed to obtain information on prematurity and other neonatal events.

Page 10-12 (Q34-46): Family History

Social class, as defined by occupational class and educational level, cigarette smoking in the household, and familial history of respiratory disease are all considered as risk factors for developing respiratory disease.

2.5 Statistical Analysis

2.5.1 One-way Analysis of Variance (ANOVA)

One-way ANOVA is a technique for assessing how several nominal independent variables affect a continuous dependent variable. The least-significant-difference (LSD) method is an application of the Bonferroni correction for multiple testing. An approximate way to circumvent the problem of distorted significance levels when making several tests involves reducing the significance level used for each individual test sufficiently to fix the overall significance level (i.e., the probability of falsely rejecting at least one of the null hypotheses being tested) at some

desired value. General data configuration for one-way ANOVA (Kleinbaum *et al.*, 1998) as example:

Population	Sample size	Observations	Total	Sample Mean
1	n_1	$Y_{11}, Y_{12}, Y_{13}, \dots, Y_{1n_1}$	$T_1 = Y_{1.}$	$\bar{Y}_{1.} = T_1 / n_1$
2	n_2	$Y_{21}, Y_{22}, Y_{23}, \dots, Y_{2n_2}$	$T_2 = Y_{2.}$	$\bar{Y}_{2.} = T_2 / n_2$
3	n_3	$Y_{31}, Y_{32}, Y_{33}, \dots, Y_{3n_3}$	$T_3 = Y_{3.}$	$\bar{Y}_{3.} = T_3 / n_3$
\vdots	\vdots	\vdots	\vdots	\vdots
k	n_k	$Y_{k1}, Y_{k2}, Y_{k3}, \dots, Y_{kn_k}$	$T_k = Y_{k.}$	$\bar{Y}_{k.} = T_k / n_k$
	k		$G = Y_{..}$	$\bar{Y} = G / n$
	$n = \sum_{i=1}^k n_i$			

The number of n_i observations selected from each i th population. Double-subscript notation (Y_{ij}) is used to distinguish one observation from another. The first subscript for a given observation denotes the population number, and the second distinguishes that observation from the others in that sample. The totals for each sample (from each population) are denoted alternatively by T_i or $Y_{i.}$ for the i th sample, where the \cdot denotes that summing over all values of j . The grand total over all sample is denoted as $Y_{..} = G$. The sample means are alternatively denoted by $\bar{Y}_{i.}$ or T_i / n_i for the i th sample; these statistics are particularly important because they represent the estimates of the population means of interest. Finally, the grand mean over all samples is $\bar{Y} = G / n$ (Kleinbaum *et al.*, 1998).

The null hypothesis of equal population means ($H_0: \mu_1 = \mu_2 = \dots = \mu_k$) was test by using an F test. The test statistic is calculated (Kleinbaum *et al.*, 1998) as follows:

$$F = \frac{\text{MST}}{\text{MSE}}$$

$$\text{Where: MST} = \frac{\sum_{i=1}^k (T_i^2 / n_i) - G^2 / n}{k - 1}$$

$$\text{and MSE} = \frac{\sum_{i=1}^k \sum_{j=1}^{n_i} Y_{ij}^2 - \sum_{i=1}^k (T_i^2 / n_i)}{n - k}$$

The critical region for this test involves only upper percentage points of the F distribution, since only large values of the F statistic (usually values much greater than 1) will provide significant evidence for rejecting H_0 (Kleinbaum *et al.*, 1998).

2.5.2 Yates' Correction Chi-Square Test

The observed frequencies in a contingency table are discrete and thereby give rise to a discrete statistic, X^2 , which is approximated by the X^2 distribution which is continuous. It was proposed a procedure for correcting for this in the case of 2×2 tables. Crosstabulation tables were used for display the relationship between the pairs of selected categorical variables (Wayne, 1978).

A 2×2 Contingency Table			
Second Criterion of Classification	First Criterion of Classification		
	1	2	Total
1	a	b	$a + b$
2	c	d	$c + d$
Total	$a + c$	$b + d$	n

The correction consists of subtracting half the total number of observations from the absolute value of the quantity $ad - bc$ before squaring.

$$X_{\text{corrected}}^2 = \frac{n(|ad - bc| - .5n)^2}{(a + c)(b + d)(a + b)(c + d)}$$

For example as follows:

Junior high school student			
Dyspnea and wheezing	HR	Area C	Total
1	10	1	11
2	63	102	165
Total	73	103	176
<i>p</i> -value		0.002	

$$\begin{aligned}
 \chi^2_{\text{corrected}} &= \frac{176(|10 \times 102 - 1 \times 63| - .5 \times 176)^2}{(10 + 63)(1 + 102)(10 + 1)(63 + 102)} \\
 &= 9.739
 \end{aligned}$$

2.5.3. Binary Logistic Regression Technique

Logistic regression is a mathematical modeling approach that can be used to describe the relationship of several predictor variables X_1, X_2, \dots, X_k to a dichotomous dependent variable Y , where Y is typically coded as 1 or 0 for its two possible categories. To determine how one or more independent variables are related to the probability of the occurrence of one of two possible outcomes. Logistic regression coefficients can be used to estimate odds ratios for each of the independent variables in the model. The formula for the logistic model can be written in a form that describes the probability of occurrence of one of the two possible outcomes of Y , as follows (Kleinbaum *et al.*, 1998):

$$\text{pr}(Y = 1) = \frac{1}{1 + \exp [- (\beta_0 + \sum_{j=1}^k \beta_j X_j)]} \quad (2.1)$$

Where: $\text{pr}(Y)$ = the probability of a positive response, X_j = independent variables

Predict available of model by predicted value and real value by cut value = .5 as probabilities cutting score: P (Event) less than 0.5 \rightarrow $y = 0$ or no event, if P (Event) greater than or equal to 0.5 \rightarrow $y = 1$ or event.

The most important reason for the popularity of the logistic model is that the right-hand side of the preceding expression ensures that the predicted value of Y will always lie between 0 and 1. Using the logit form of the logistic model defined by the expression

$$\text{logit}[\text{pr}(Y = 1)] = \beta_0 + \sum_{j=1}^k \beta_j X_j \quad \text{_____} (2.2)$$

Where: Y = the dependent variable, β_0 = constant, β_j = coefficients

The dependent variable should be dichotomous. Independent variables can be interval level or categorical; if categorical, they should be a dummy or indicator coded. A dummy, or indicator variable is any variable in a regression equation that takes on a finite number of values so that different categories of a nominal variable can be identified. The term dummy reflects the fact that the values taken on by such variables (usually value like 0 and 1) do not indicate meaningful measurements but rather the categories of interest. Example of dummy variable includes the following:

$$\begin{aligned} X_1 &= \begin{cases} 1 & \text{if residence is in high-polluted roadside area (HR)} \\ 0 & \text{otherwise} \end{cases} \\ X_2 &= \begin{cases} 1 & \text{if residence is in high-polluted general area (HG)} \\ 0 & \text{otherwise} \end{cases} \\ X_3 &= \begin{cases} 1 & \text{if residence is in moderate-polluted roadside area (MR)} \\ 0 & \text{otherwise} \end{cases} \\ X_4 &= \begin{cases} 1 & \text{if residence is in control area (C)} \\ 0 & \text{otherwise} \end{cases} \end{aligned}$$

The variable X_1 , X_2 , X_3 , and X_4 indicate the nominal variable geographical residence. The two categories of geographical residence are described by the following combination of the four variables X_1 , X_2 , X_3 , and X_4 :

Residence is in HR area: $X_1 = 1, X_2 = 0, X_3 = 0, X_4 = 0$

Residence is in HG area: $X_2 = 1, X_1 = 0, X_3 = 0, X_4 = 0$

Residence is in MR area: $X_3 = 1, X_1 = 0, X_2 = 0, X_4 = 0$

Residence is in C area: $X_4 = 1, X_1 = 0, X_2 = 0, X_3 = 0$

Logistic regression model regresses the dichotomous outcome variable (Any of respiratory symptoms) on the predictors questionnaire responder, age, gender, residential years, home size, family members, parental smoking habits, use of air conditioners, having domestic pets, and residential areas j , for $j = 1, 2, 3, 4$. The logit form of the model being fit is given as

$$\text{logit}[\text{pr}(Y = 1)] = \beta_0 + \beta_1 (\text{questionnaire responder}) + \beta_2 (\text{age}) + \beta_3 (\text{gender}) + \beta_4 (\text{residential years}) + \beta_5 (\text{home size}) + \beta_6 (\text{family members}) + \beta_7 (\text{parental smoking habits}) + \beta_8 (\text{use of air conditioners}) + \beta_9 (\text{having domestic pets}) + \beta_{10} (\text{residential area 1: HR}) + \beta_{10} (\text{residential area 2: HG}) + \beta_{10} (\text{residential area 3: MR}) + \beta_{10} (\text{residential area 4: C})$$

The odds ratio (OR) is a widely used measure of effect in epidemiological studies. It is a ratio of event odds. It means a measure that compares two or more groups in predicting the outcome (dependent) variable. It defines odds of an event as the ratio of the probability that the event occur (e.g. any of respiratory symptoms) divided by the probability that the event does not occur (e.g. no any of respiratory symptoms). Thus, the odds for event D are given by the formula:

$$\text{odds}(D) = \frac{\text{pr}(D)}{\text{pr}(\text{not } D)} = \frac{\text{pr}(D)}{1 - \text{pr}(D)}$$

Any odds ratio, by definition, is a ratio of two odds:

$$OR_{A \text{ vs. } B} = \frac{\text{odds}(D_A)}{\text{odds}(D_B)} = \frac{\text{pr}(D_A)}{1 - \text{pr}(D_A)} \div \frac{\text{pr}(D_B)}{1 - \text{pr}(D_B)}$$

Generally, when computing an odds ratio, it can define the two groups (or individuals) that are to be compared in terms of two different specifications of the set of predictors X_1, X_2, \dots, X_k . It can do this by letting $X_A = (X_{A1}, X_{A2}, \dots, X_{Ak})$ and $X_B = (X_{B1}, X_{B2}, \dots, X_{Bk})$ denote the collection of X 's for groups (or individuals) A and B , respectively. It can estimate an odds ratio: the general formula for an odds ratio comparing two specifications of the set of predictors X_A and X_B is

$$OR_{X_A \text{ vs. } X_B} = e^{\sum_{j=1}^k (X_{Aj} - X_{Bj}) \beta_j} \quad \text{—————(2.3)}$$

For example, if $k = 10$, X_1 is questionnaire responder (mother=1, father or others=0), X_2 is gender (male=1, female=0), X_3 is age (continuous), X_4 is residential years (continuous), X_5 is home size (continuous), X_6 is family members (continuous), X_7 is parental smoking habits (mother or father or both smokes=1, neither smokes=0), X_8 is use of air conditioners (yes=1, no=0), X_9 is having domestic pets (yes=1, no=0), X_{10} is residential areas (HR = 1, HG = 2, MR = 3, C = 4), then X_A and X_B are two specifications of these ten variables, so that $X_A = (1, 1, 10, 2, 2, 4, 1, 1, 1, 1)$ and $X_B = (0, 1, 10, 2, 2, 4, 1, 1, 1, 1)$, as described earlier, then

$$OR_{X_A \text{ vs. } X_B} = e^{(1-0)\beta_1 + (1-1)\beta_2 + (10-10)\beta_3 + (2-2)\beta_4 + (2-2)\beta_5 + (4-4)\beta_6 + (1-1)\beta_7 + (1-1)\beta_8 + (1-1)\beta_9 + (1-1)\beta_{10}}$$

$$= e^{\beta_1} \quad \text{—————(2.4)}$$

A 95% confidence interval for e^{β_1} can be obtained by computing

$$\exp [\hat{\beta}_1 \pm 1.96 (\text{SE})] \quad \text{—————(2.5)}$$

Where: β_j = the estimated coefficient, SE = the estimated standard error

Maximum likelihood estimates and associated standard errors of the regression coefficients in a logistic model are typically obtained by using computer packages for logistic regression. These statistics can then be used to obtain numerical values for estimated adjusted odds ratios, to test hypotheses, and to obtain confidence intervals for population odds ratios based on standard maximum likelihood techniques.

When OR greater than 1.0, it means increased risk; OR greater than 2 usually important. If OR equal to 1.0 means no risk and OR less than 1.0 means decreased risk. Checking observations did not occur by chance, usually determined by a confidence interval such as 95% CI indicating that the true value lies in the range with a 95% probability.

LITERATURE REVIEW

1. Other Air Pollutants

A large city with congested areas like Bangkok has serious environmental problems from air pollution. Particulate matter is a major air pollution problem. The major sources of PM₁₀ are road dust, steam boiler, vehicle engine combustion, construction, and power plant. In 1993, the level of lead in the air was reduced significantly after the introduction of unleaded gasoline. During the period from 1995 to 2000, carboxyhemoglobin levels were above 3 percent in the period 1995-1997 (Figure 2.9). After that, during the period of economic turmoil starting in July 1997, carboxyhemoglobin levels decreased to 1.5 percent in 1998 and 1.14 percent in 2000; methemoglobin levels also decreased from 1.14 percent to become 0.2 percent. Sulfhemoglobin was not present during these six years of monitoring (Saenghirunvattana *et al.*, 2000).

Level (%)

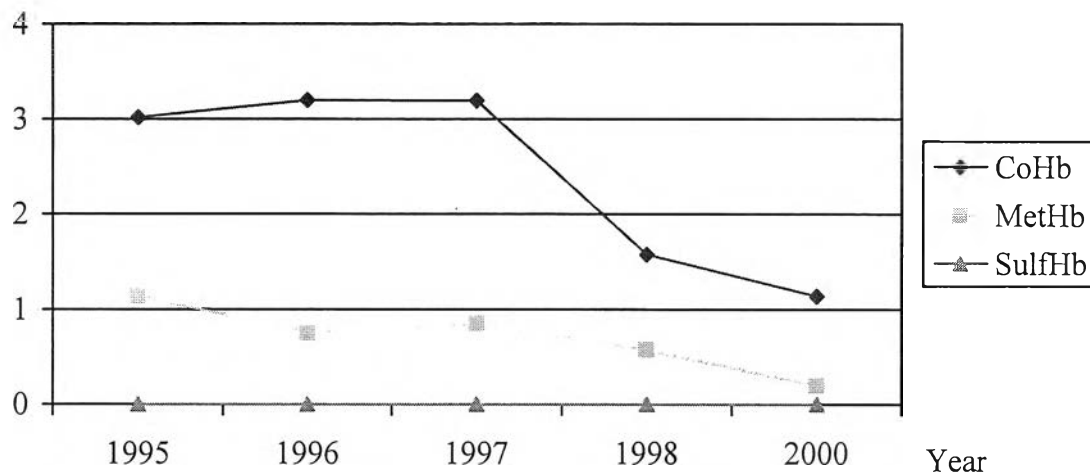


Figure 2.9 Levels of carboxyhemoglobin, methemoglobin, and Sulfhemoglobin, 1995-2000

Source: Saenghirunvattana *et al.*, 2000

In 1994, unleaded premium gasoline was introduced, and lead free gasoline was made mandatory in 1997, leading to a dramatic decline in lead levels in the air. In 1989, reduction of lead in gasoline was made from 0.45 g/l to 0.4 g/l, and aggressively dropped to 0.15 g/l in 1992. In 1994, unleaded premium gasoline was introduced, and lead free gasoline was made mandatory in 1997, leading to a dramatic decline in lead levels in the air. As the regulation in 1993, catalytic converters were required to be installed in all new passenger cars; carbon monoxide had been consequently declined. Ozone was first detected exceeding the ambient standard in some monitoring stations in Bangkok in 1998. Nitrogen dioxide and sulfur dioxide levels are always under the Standard (PCD, 2001) (Table 2.9).

Table 2.9 Annual average of other air pollutants in the general areas of Bangkok during 1999-2003

Pollutants	Year	Concentrations ^a		
		Average	Standard	Frequency of Exceeding Standard (%)
Pb (1-month), $\mu\text{g}/\text{m}^3$	1999	0.10	1.5	0/116 (0)
	2000	0.09		0/93 (0)
	2001	0.12		0/107 (0)
	2002	-		-
	2003	-		-
CO (1-hr), ppm	1999	1.25	30	0/54,218 (0)
	2000	0.96		0/70,186 (0)
	2001	0.94		0/67,368 (0)
	2002	0.85		0/81,379 (0)
	2003	0.70		0/74,991 (0)
CO (8-hr), ppm	1999	1.27	9	118/59,785 (0.20)
	2000	0.97		0/71,609 (0)
	2001	0.94		0/68,654 (0)
	2002	0.90		0/83,928 (0)
	2003	0.70		0/77,643 (0)
O ₃ (1-hr), ppb	1999	13.9	100	59/42,026 (0.14)
	2000	15.6		161/54,415 (0.29)
	2001	14.3		75/54,764 (0.14)
	2002	13.7		93/62,669 (0.15)
	2003	15.7		155/61,789 (0.25)

a: - = No detectable

Source: PCD, 2000-2004

Table 2.9 Annual average of other air pollutants in the general areas of Bangkok during 1999-2003 (cont.)

Pollutants	Year	Concentrations		Frequency of Exceeding Standard (%)
		Average	Standard	
SO ₂ (1-hr), ppb	1999	4.8	300	0/57,306 (0)
	2000	6.7		0/72,750 (0)
	2001	5.6		0/69,272 (0)
	2002	5.2		0/76,252 (0)
	2003	4.7		0/77,176 (0)
SO ₂ (24-hr), ppb	1999	4.9	120	0/2,430 (0)
	2000	6.7		0/3,062 (0)
	2001	-		-
	2002	5.2		0/3,236 (0)
	2003	4.7		0/3,206 (0)
NO ₂ (1-hr), ppb	1999	22.8	170	0/44,907 (0)
	2000	22.8		0/67,094 (0)
	2001	22.5		0/73,290 (0)
	2002	23.9		0/79,930 (0)
	2003	23.0		0/78,041 (0)

a: - = No detectable

Source: PCD, 2000-2004

The level of PM₁₀ has become a significant problem along roadside areas during the traffic congestion periods. The short-term (24-hour) and the long-term (1-year) average levels of particulate matter are monitored using high volume sampler technique by PCD, Ministry of Science and Technology. There are 7-fixed, 21-temporary roadside, and 10 ambient (residential area) air quality-monitoring stations installed in order to monitor the air pollution. In 2004, the 24-hour average

concentrations of TSP and PM₁₀ at roadside monitoring stations were about 0.18 mg/m³ and 78.5 µg/m³ that exceeded the standard in Thailand by approximately 8.3 and 8.4%, respectively (PCD, 2005).

2. Respiratory and Cardiovascular Health

The pollution impact risk measured was via the respiratory pathway. Particulate matters are deposited in the lungs by one of four different ways including interception, impaction, sedimentation, and diffusion. In general, particulate matter having an aerodynamic diameter of 5 to 30 µm is deposited in the nasopharyngeal region largely by impaction. The most sensitive factor in the risk analysis was the ground level concentration of the pollutants. Air pollution contributes to preventable illness and death, including: premature mortality, chronic respiratory disease (Roemer *et al.*, 1993), respiratory emergency room visits and hospital admissions, aggravated asthma, acute respiratory symptoms, and decreased lung function (Pope *et al.*, 1991; and Pope *et al.*, 1993). Subgroups of patients who appear to be more sensitive to the effects of air pollution include young children, the elderly and people with existing chronic cardiac and respiratory disease (Clench-Aas *et al.*, 1999; Hoek *et al.*, 1994; Remes *et al.*, 1996; Tiitanen *et al.*, 1999; Timonen *et al.*, 1995; Ward *et al.*, 2004; and Ware *et al.*, 1986).

Jocelyne *et al.*, 1999 reported that the panel study of 2,300 school-age children in Oslo was initiated to study the role of traffic pollution on the exacerbation of diseases of the respiratory system and other symptoms of reduced health and well being in children 7 to 12 years of age. Parent of all children filled out a diary daily with information five times over six weeks. This study design enhanced the possibility of measuring dose-response relationships. Individual exposure to PM_{2.5} and NO₂ was estimated using DINEX method, a combination of information from the diary, coupled with continuous dispersion modeling. The relationship between the estimates of NO₂ and PM_{2.5} either as estimated at the home of the participant or as the mean exposure of the child during 6 weeks in the winter was estimated using the diary. The overall geographical distributions of the estimates NO₂ and PM_{2.5} are not

identical. Furthermore, exposure by time of day shows a trend with lowest values at night.

Neuberger *et al.*, 2002 reported health effects of particulate matter on kindergarten children living near an air monitoring station in Vienna, Austria. The results showed that a trend for negative effect after averaging concentrations over 7 days before the lung function test.

Duanping *et al.*, 2004 reported an association between air pollution and increased cardiovascular disease (CVD) mortality. The authors examined short-term associations between PM₁₀, O₃, CO₂, NO₂, and SO₂ and cardiac autonomic control using data from the fourth cohort examination (1996-1998) of the population-based Atherosclerosis Risk in Communities Study. For each participant, the authors calculated PM₁₀ and gaseous pollutant exposures as 24 hour averages and ozone exposure as an 8 hour average 1 day prior to the randomly allocated examination date. They calculated 5-minute heart rate variability indices and used logarithmically transformed data on high frequency (0.15-0.40 Hz) and low-frequency (0.04-0.15 Hz) power, standard deviation of normal R-R intervals, and mean heart rate. Linear regression was used to adjust for CVD risk factors and demographic, socioeconomic, and meteorological variables. Regression coefficients for a one-standard-deviation increase in PM₁₀ (11.5 $\mu\text{m}/\text{m}^3$) were -0.06 ms (standard error, 0.018), -1.03 ms (SE, 0.31), and 0.32 beats/minute (SE, 0.158) for log-transformed high-frequency power, standard deviation of normal R-R interval, and heart rate, respectively. Similar results were found for gaseous pollutants. These cross-sectional findings suggest that higher ambient pollutant concentrations are associated with lower cardiac autonomic control, especially among persons with existing CVD, and highlight a putative mechanism through which air pollution is associated with CVD.

Jedrychowski *et al.*, 1999 showed associations between ambient air pollutants including PM_{2.5} and PM₁₀ and poorer gain of FVC in children living in a polluted area of Krakow. Children who might already have existing respiratory diseases were more sensitive to air pollution; their risk of developing respiratory symptoms was 2 to 4

times higher than normal children. Boezen *et al.*, 1999 have demonstrated that children with bronchial hyperreactivity and high serum IgE levels are more susceptible to air pollution compared to those with low serum IgE levels.

Horak *et al.*, 2002 assessed the effects of PM₁₀ and other air pollutants on lung function in 975 schoolchildren, from eight communities in Lower Austria during 1994 to 1997. Spirometry was performed twice a year. They concluded that long-term exposure to PM₁₀ had a significant negative effect on lung-function proxy for the development of large (FEV₁) and small (FEF_{25-75%}) airways, respectively, with strong evidence for a further effect of ozone and nitrogen dioxide on the development of FVC and FEV₁.

Ostro *et al.*, 1999 suggest a positive association between ambient PM₁₀ and total mortality in children under 6 years old. It was demonstrated that an increase of 10 µg/m³ in the 24-hour average levels of PM₁₀ was associated with an increase in total child mortality of 1.8% (CI: 1.60-3.40). Wongsurakiat *et al.*, 1999 found in a cross-sectional study that traffic policemen who worked in Thonburi district of Bangkok suffered significantly more cough or phlegm and more rhinitis symptoms and the mean values of FEV₁ and FVC were significantly lower than the control group.

Karita *et al.*, 2001 have reported that PM₁₀ in Bangkok affected on pulmonary function and respiratory systems in traffic policemen. They showed that the mean level of FEV₁ and FEF_{25-75%} were significantly lower in Bangkok police than in Ayutthaya police. The prevalence of respiratory symptoms among Bangkok police was slightly higher than among Ayutthaya police. Furthermore, researchers provide some evidence of an increase in prevalence of obstructive changes in the peripheral airways among traffic police in Bangkok. Jinsart *et al.*, 2002 investigated the levels of PM_{2.5} and PM₁₀ in Bangkok, Nonthaburi and Ayutthaya and showed a significantly higher proportion of PM_{2.5} to PM₁₀ in the high-polluted area than in the low-polluted area. Bangkok traffic police were exposed to high levels of automobile-derived particulate air pollution. Tamura *et al.*, 2003 have demonstrated the prevalence of

chronic respiratory symptoms associated with exposure levels to roadside particulate matter among traffic policemen in Bangkok. They reported that age-adjusted prevalence of NSRD among nonsmokers in the heavily polluted areas was significantly higher than in the suburban control area. The increased prevalence of respiratory symptoms among traffic policemen in Bangkok was associated with urban traffic air pollution.

Furthermore, Karita *et al.*, 2004 reported health effects of PM₁₀ have been determined the prevalence of respiratory symptoms in policemen and their wives in Bangkok. They showed that the increased risk of frequent cough or phlegm in the policemen was related to smoking and working in heavy traffic locations, whereas in their wives it was related to their living locations.

Aekplakorn *et al.*, 2003a demonstrated associations of daily exposure to SO₂ and PM₁₀ with pulmonary function in asthmatic and non-asthmatic children aged 6-14 years who live near a coal-fired power plant in Maemoh, Thailand. They found that decrements in pulmonary function among asthmatic children were associated with increases in PM₁₀ rather than SO₂. Furthermore, increasing 10 µg/m³ of PM₁₀ was modestly associated with increases of lower respiratory symptom incidence and cough in asthmatic children (Aekplakorn *et al.*, 2003b).

The short-term effect of particulate air pollution on the respiratory morbidity of children has been the subject of considerable investigations over the past decade. Air pollution is a major environmental related health threat to children and a risk factor for both acute and chronic respiratory disease. Since the lungs of children are not fully developed, early damage to the respiratory tract could increase the risk of respiratory disease in later life. Children inhale several times more air than adults, breath faster, particularly during strenuous physical activity and spend more time outdoors than any other segments of the populations, so they are much more likely than adults to develop pollutants related lung damage.

Research in cities throughout the world indicates that current levels of public exposure to airborne particulate matter are associated with premature mortality and respiratory-related illness. The illnesses associated with airborne particulate matter range from severe acute and chronic illnesses such as asthma attacks and chronic bronchitis to mild acute symptoms such as coughing, wheezing, and congestion. Taken as a whole, the available epidemiological evidence shows a strong relationship between particulate matter and public health. There is evidence of health effects for both short-term fluctuations in particulate matter concentrations in a given location and long-term differences in particulate matter concentrations across locations.

Recent results suggest that adverse health effect of air pollutants exist at levels of pollutants around or even below air quality standards set by national and international institutes. Furthermore, there are indications that air pollution effects on health may be partly determined by specific mixtures of air pollutants and may be altered by other environmental, behavioral, and social patterns.

PM₁₀ in Bangkok has been associated with serious health effects, such as increased hospital admissions and mortality. Also associations were reported between air pollution and respiratory health among traffic policemen and their wives in Bangkok. However, those studies have mainly been conducted in healthy adults groups. It is not clear to what extent such associations would be revealed in children who might be more susceptible to air pollution than adults. A few researches suggested lower respiratory symptoms and decrements in lung function among asthmatic children in Maemoh, but chronic health effects for children remain uncertain, particularly for Bangkok children. Therefore, with a cross-sectional design, this study investigated the possible chronic effects of exposure to air pollution in schoolchildren living in Bangkok. The purpose of the study was to evaluate the association between level of PM₁₀ and respiratory symptoms, or lung function, using the ATS-DLD-78-C respiratory questionnaires and spirometry among Bangkok schoolchildren in four areas and compare with Ayutthaya as a rural area.