Air Quality and Children's Environmental Health

Is Our Future Generation At Risk?

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ABSTRACT

The text discusses on the air quality with various types of pollutants which included primary and secondary gaseous pollutants, respirable particulate, lead and microbes. Health impacts on children such as respiratory symptoms, asthma, allergies, and impairment of the lung functions, cognitive, as well as hearing ability have been discussed as evidence from previous research findings in other countries generally and in Malaysia specifically. Vulnerability of children to the gaseous ambient air pollutants were highlighted. The indoor air quality has also lately posed threats to children especially microbes in indoor environment. These microbes specifically fungus, caused allergic reactions as well as trigger asthmatic related symptoms to sensitive groups. With the increase of environmental threats such as climate change, air quality will deteriorate further which may amplify respiratory diseases and health problems among children especially the urban poor. Health risk assessment as a tool, can be used to assess the risk, however, it has limitations and is not protective of children. The US EPA has developed a "A Framework for Assessing Health Risk of Environmental Exposures to Children" document with the purpose of providing an overarching framework for a more complete assessment of children's exposure to environmental agents and the resulting potential health risks. The future direction spells out the responsibilities of parents, teachers, ministries and governmental roles in the protection of children's health.

INTRODUCTION

The health effects of air pollution exposure have become an area of increasing focus in the past 30 years. Evidences have demonstrated that there are serious health consequences to community air pollutants, however; these consequences are not spread equally among the population (Sacks et al. 2011, WHO, 2011, WHO, 2005). Children's exposure to air pollution is a special concern because their immune system and lungs are not fully developed when exposure begins, raising the possibility of different responses than seen in adults (Schwartz 2004).

Children are affected by exposures to environmental hazards, because they are uniquely vulnerable. Many children live in communities that are disproportionately impacted by environmental exposures. Many environmental hazards and pollution know no boundaries. The health of children worldwide is intrinsically linked to the health of our environment. Healthy children grow into healthy adults. The health of our children is one of the most important investments that we can make and should be among our top priorities.

AMBIENT AIR QUALITY

Nitrogen and oxygen are the predominant gases in the atmosphere, and together they constitute 99 percent of the mixture by volume. Nearly all of the remainders of the atmosphere are made up of argon and carbon dioxide. The total volume percent of these four components in clean, dry air is 99.99 percent (Table 1). The minor components of air are numerous, and a number of them are the result of various natural processes. Hydrogen sulfide (H_2S), sulfur dioxide (SO_2), and carbon monoxide (CO) are all put into the atmosphere by volcanic action. The decay of plants and animals under conditions

that exclude oxygen (anaerobic decay) produces methane (CH_4), ammonia (NH_3), and hydrogen sulfide. Nitrogen oxides (N_2O, NO, NO_2) are produced by electrical discharges during thunderstorms, and tons of carbon monoxide are generated by forest fires (Table 2).

Table 1 Atmospheric gases at constant concentration

Gas	Concentration (ppm)
Nitrogen	780,840.00
Oxygen	209,460.00
Argon	9,340.00
Neon	18.18
Helium	5.24
Krypton	1.14
Hydrogen	0.50
Xenon	0.09

Source : Godish, 1997

Air Pollution

The addition of any substance will alter to some degree the physical and chemical properties of clean air. Such a substance could thus be considered to be an air pollutant. However, pollutants are usually classified as only those substances which are added in an amount sufficient to cause measurable effects on humans, other animals, vegetation, or materials.

Table 2 Atmospheric gases at variable concentration

Gas	Concentration (ppm)
Water vapor	0.1-30,000.00
Carbon dioxide	360.00
Methane	1.72
Nitrous oxide	0.33
Carbon monoxide	0.11
Ozone	0.02
Ammonia	0.004
Nitrogen dioxide	0.001
Sulfur dioxide	0.001
Nitric oxide	0.0005
Hydrogen sulfide	0.00005

Source : Godish, 1997

Air pollution occurs as a consequence of natural processes such as volcanic eruptions, forest fires and wind blown dust as well as anthropogenic air pollution from sources like motor vehicles and industries which continue to be a serious problem. Its seriousness is due to the fact that elevated levels of air pollutants from these anthropogenic sources in the environments tend to cause serious harm to human health and welfare especially in densely populated urban areas.

Air pollutants are heterogeneous mixture of gases and particulate matter which can be grouped into two; the primary pollutants and secondary pollutants. Trace of sulfur and incomplete combustion resulted in emission of carbon monoxide, sulfur dioxide, oxides of nitrogen, unburned hydrocarbons and particulate are grouped as primary pollutants. Whereas secondary pollutants are formed in the atmosphere through chemical reactions of primary pollutants

producing ozone, acid rain, peroxy-acetyl nitrate (PAN) and photochemical smog.

Sources and Types of Air Pollutants in Malaysia

Five types of substances, known as primary pollutants, account for more than 90 percent of the nationwide air pollution problem are:

1. Carbon monoxide (CO)
2. Nitrogen oxides (NO_x)
3. Hydrocarbons (HC)
4. Sulfur oxides (SO_x)
5. Respirable particulates (PM_{10})

In Malaysia, air pollution has been recognized as one of the major concern that has high potential for deleterious effects on health (Junaidah et al. 2010, Jamal et al. 2004, Zailina, Juliana and Jamal 2004, Juliana et al. 2002, Zailina et al. 1997, Zailina et al. 1996b, Zailina, Juliana and Jamal 1996c). Increased urbanization, human activities and changing urban setting in the country as a result of development have resulted in elevated air pollution and the occurrence of urban heat islands.

The sources of air pollutants are both localized and trans-boundary. Localized sources come from both mobile as well as stationary sources. Mobile sources are from private and public motor vehicles which are especially significant in the Klang Valley. Stationary sources include power plants, industrial waste incinerators, cement plants, iron and steel mills from the industrial and suburban areas, the clearing and burning of old oil palm trees in plantations, emissions of dusts from urban constructions and quarries, and open burning at some solid waste dumpsites (Table 3). The combustion of these fuels produces a range of gaseous and particulate emissions which can be typically grouped as:

i. Greenhouse gases : carbon dioxide (CO_2), methane (CH_4) and nitrous oxide (N_2O).

ii. The common (sometimes called 'criteria') ambient air pollutants : carbon monoxide (CO), nitrogen oxides (NO and NO_2 expressed as NO_2), ozone (O_3), sulfur oxides (SO_2 and other oxidized forms of sulfur expressed as SO_2), particulate matter (TSP, PM_{10} and $PM_{2.5}$), and lead (Pb).

iii. Toxic (or hazardous) air contaminants (also known as air toxics): aldehydes, polycyclic aromatic hydrocarbons (PAHs), benzene and 1,3 butadiene.

Table 3 Malaysia air emission load and pollutant sources 2007

Pollutants	Overall emission load (tonnes)	Motor vehicles	Industries	Power plant	Others
CO	1,774,396	98%	1%	1%	-
SO_2	158,120	16%	23%	41%	20%
PM	38,763	28%	40%	14%	18%
NO_x	563,078	70%	12%	16%	2%

Source: DOE, 2009

Trans-boundary pollutants from neighboring countries such as Indonesia are from forest fires during the dry seasons, especially between the months of August to November each year. This transported air pollution from forest fires has resulted in what is now commonly referred to as the haze, not only in Malaysia but also in other countries in the region including Singapore, Brunei and Thailand. The worst of these recurring haze episodes was the

one that prevailed over much of Southeast Asia in 1997. Widespread forest fires in Kalimantan and Sumatra were triggered by extremely dry weather brought about the El Nino phenomenon. The Klang Valley and Kuching in Sarawak were among the worst hit areas in Malaysia. (Jamal and Zailina 1994, Brauer and Jamal 1998).

Malaysian Ambient Air Quality Guidelines

The Malaysian Ambient Air Quality Guidelines (MAAQG) prescribes ambient guidelines for 7 criteria air pollutants as shown in Table 4. In this study, 5 criteria pollutants were selected for assessment of their associations with patient morbidity and mortality, namely ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide respirable and particulate matter (PM_{10}). These 5 pollutants were selected because they tend to have the greatest impacts on human health, especially on respiratory and cardiovascular morbidity and mortality. These 5 pollutants are also used in the computation of the Air Pollutant Index (API).

CHILDREN'S VULNERABILITY

Usually urban children are better off than the rural children; however, it is not true for the millions living in urban poverty. Poor urban squatter areas can be the most life threatening environment. Children in the urban areas are more likely at risk of being exposed to air pollution (Junaidah et al. 2010, Junidah et al.2009). Short-term effects include irritation to the eyes, nose and throat, and upper respiratory infections such as bronchitis and pneumonia. Other symptoms can include headaches, nausea, and allergic reactions. Short-term air pollution can aggravate the medical conditions of individuals with asthma and emphysema. Long-term health effects can include chronic respiratory disease, lung cancer, heart disease, and even damage to the brain, nerves, liver, or kidneys. Continuous

Table 4 Malaysian ambient air quality guidelines

Pollutant	Averaging time	Malaysian guideline	
		ppm	ug/m^3
Total suspended particulate (TSP)	24 hour 1 year		260 90
Particulate matter (PM10)	24 hour 1 year		150 @ 50
Nitrogen dioxide	1 hour 24 hour	0.17 0.06	320 113 @
Sulfur dioxide	10 minutes 1 hour 24 hour	0.19 0.13 0.04	500 350 105 @
Carbon monoxide	1 hour 8 hour	30 9	35 * 10 * @
Ozone	1 hour 8 hour	0.10 0.06	200 120 @
Lead	3 months		1.5

* mg/m^3, @ API = 100
API : 0 - 50 (Good) 51 - 100 (Moderate) 101 - 200 (Unhealthy)
201 - 300 (Very unhealthy) 301 - 500 (Hazardous)

Source:DOE (2007)

exposure to air pollution affects the lungs of growing children and may aggravate or complicate the lung functions.

There is also concern over the children's vulnerability due to their increased exposure as children in tropical climate, spend more time outdoor and are active in their daily activities especially in the afternoon when pollutants are high. Typical adults spend 85 percent

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.

Size and Physiological Development

Perhaps the most important difference between adults and children is that children are growing and developing. Along with their increased body size, children's lungs are growing and changing. Therefore, they are physiologically not matured to deal with environmental changes. Their metabolic rates are fast and most organs and bodily systems are not matured. Irritation or inflammation caused by air pollution is more likely to obstruct their narrower airways. Their lungs are more susceptible to injury caused by pollutants whereby their oxidant induced injury will most likely induce inflammation that could result in the structure alteration. Exposure to pollutant during infancy or childhood could affect the development of the respiratory system and may result in decreased lung capacity or size due to hindered growth (Zailina et al. 2004, Abdul Mujid et al. 2003, Juliana et al. 2002, Rosenlund et al. 2009). They have limited experience and their cognitive capabilities are developing. Children do not necessarily respond to air pollution the same way as adults. Adults who are exposed to low levels of the pollutant ozone will experience symptoms such as coughing, soreness in their chests, sore throats, and sometimes headaches. Children, on the other hand, may not feel the same symptoms. Therefore, they usually do not complaint, thus the exposure related symptoms are not noticed.

Metabolic Rate

In many health effects research studies, children are considered as less resistance to adapt the health risk factors from the environment.

There are differences between children and adults in the ways that they respond to air pollution. For example, children take in more air per unit body weight at a given level of exertion than adults. When a child is exercising at maximum levels, such as during a soccer game they take in 20 percent to 50 percent more air and more air pollution than adult in comparable activity. Children with asthma are at much greater risk of increased asthma symptoms when they live in communities with higher levels of ozone and particles and participate in three or more competitive sports (Schwartz, 2005).

AIR POLLUTANTS AND HEALTH

Ozone

Ozone is one of the most important air pollutants affecting human health in tropical regions with plenty of sunlight. Ozone is formed by the reaction with sunlight (photochemical reaction) of pollutants such as nitrogen oxides (NO_x) from vehicle and industry emissions and volatile organic compounds (VOCs) emitted by vehicles, solvents and various industries. Because of these, the highest concentrations of ozone in the atmosphere occur when sunlight is at its most intense. Thus, ozone generally reaches peak levels during the middle of the day.

Ozone (O_3) is a molecule built of three atoms of oxygen linked together in a very energetic combination. When ozone comes into contact with a surface, it rapidly releases this extra force in the form of chemical energy. When this happens in biological systems, such as the respiratory tract, this energy can cause damage to sensitive tissues in the upper and lower airways. Tager et al. (2005) found that lifetime exposure to ozone in adolescents is associated with reduced levels of lung function measures which reflect the functions of the small airways.

Similar to oxygen, ozone is soluble in the fluids that line the respiratory tract. Therefore, some ozone can penetrate into the gas-exchange, or alveolar, region of the deep lung. It can cause irritation, lung inflammation, lung functions impairment, small airways dysfunction, shortness of breath, chest pain wheezing, coughing and exacerbation of asthma and death (Schwartz 2005). Long-term exposure or after a lifetime exposure or with more than 4 years of residence in high ozone area may lead to chronically reduced lung function (Kunzli et al. 1997).

Sulfur Dioxide

SO_2 is produced from the burning of fossil fuels (coal and oil) and the smelting of mineral ores that contain sulfur. The main anthropogenic source of SO_2 is the burning of sulfur-containing fossil fuels for domestic heating, power generation and motor vehicles. SO_2 can affect the respiratory system and the functions of the lungs, and causes irritation of the eyes. Inflammation of the respiratory tract causes coughing, mucus secretion, aggravation of asthma and chronic bronchitis and makes people more prone to infections of the respiratory tract. Hospital admissions for cardiac disease and mortality increase on days with higher SO_2 levels. When SO_2 combines with water, it forms sulfuric acid; this is the main component of acid rain which is a cause of deforestation (WHO 2011).

Respirable Particulate Matter (PM_{10})

PM_{10} consists of small microscopic particles of less that one seventh of human hair that are of respirable sizes. Several studies have documented increased respiratory symptoms or increased hospitalizations for acute respiratory illness among asthmatic

children in association with PM exposures (Jamal et al. 2004). Findings in Table 5 and 6 show that PM_{10} and NO_2 levels in the ambient air significantly influenced the admission of respiratory and cardiovascular cases in the Kuala Lumpur Hospital. The respiratory effects were highly significant and were immediate, within the same day of the pollutant level. After 1-3 days, the effects were significant but less. The moving average of the PM_{10} and NO_2 for 3 and 5 days were very significantly related to the admission due to respiratory problems.

The major components of PM are sulfate, nitrates, ammonia, sodium chloride, carbon, mineral dust and water. It consists of a complex mixture of solid and liquid particles of organic and inorganic substances suspended in the air. The particles are identified according to their aerodynamic diameter, as either PM_{10} (particles with an aerodynamic diameter smaller than 10 μm) or $PM_{2.5}$ (aerodynamic diameter smaller than 2.5 μm). The latter are more dangerous since, when inhaled, they may reach the peripheral regions of the bronchioles, and interfere with gas exchange inside the lungs (Franck et al. 2011). Increased risk of PM_{10} related health effects include life stage (i.e. children and elderly) pre-existing cardiovascular or respiratory diseases, genetic polymorphisms and low socio-economic status (Sacks et al. 2011).

Nitrogen Dioxide

As an air pollutant, NO_2 has several correlated activities.

- At short-term concentrations exceeding 200 $\mu g/m^3$, causes significant inflammation of the airways.
- NO_2 is the main source of nitrate aerosols, which form an important fraction of $PM_{2.5}$ in the presence of ultraviolet light, of ozone.

The major sources of anthropogenic emissions of NO_2 are combustion processes (heating, power generation, and engines in vehicles and ships). Epidemiological studies have shown that symptoms of bronchitis in asthmatic children increase in association with long-term exposure to NO_2. Reduced lung function growth is also linked to NO_2 at concentrations currently measured (or observed) in cities of Europe and North America (Brook et al. 2004, WHO, 2005).There was a strong association between estimated NO_2 exposure per 10 ug/m^3 and lung function, especially expiratory flow when adjusted for age gender, height and weight, in linear regression model conducted by Rosenlund et al. (2009). Combustion products containing SO_2 were risk factors for the development of wheezing bronchitis in female children (Pershagen et al. 1995).

Table 5 Relative risk (RR) of children admissions for respiratory and cardiovascular cases at Hospital Kuala Lumpur by quartile increments of ambient PM_{10} concentration, 2000 – 2003 (N = 1277)

Admission cases	Lag day/ moving average	PM_{10} concentration increment						p
		56.94 mg/m³ (First quartile)		67.90 mg/m³ (Median)		79.00 mg/m³ (Third quartile)		
		RR	95% CI	RR	95% CI	RR	95% CI	
Respiratory	0	0.64	0.51 – 0.81	0.59	0.45 – 0.78	0.55	0.40 – 0.75	<0.001***
	1	0.71	0.57 – 0.89	0.67	0.51 – 0.87	0.63	0.46 – 086	0.003**
	2	0.75	0.60 – 0.94	0.71	0.54 – 0.93	0.68	0.50 – 0.92	0.014*
	3	0.81	0.64 – 1.01	0.78	0.59 – 1.01	0.75	0.55 – 1.02	0.065
	MA3	0.62	0.47 – 0.81	0.56	0.41 – 0.77	0.51	0.36 – 0.74	<0.001***
	MA5	0.62	0.47 – 0.83	0.57	0.40 – 0.80	0.52	0.35 – 0.77	0.001***
Cardiovascular	0	1.15	0.10 –13.53	1.18	0.06 – 21.82	1.21	0.04 – 35.44	0.913
	1	1.38	0.11 –17.89	1.47	0.07 – 30.37	1.56	0.05 – 51.95	0.804
	2	1.85	0.15 – 0.15	2.07	0.10 – 0.10	2.32	0.07 – 0.07	0.633
	3	0.60	0.03 –10.47	0.54	0.02 – 16.11	0.50	0.01 – 24.94	0.725
	MA3	1.36	0.07 –24.90	1.44	0.05 – 44.91	1.52	0.03 – 81.71	0.835
	MA5	0.48	0.01 –15.96	0.42	0.01 – 26.54	0.37	0.00 – 44.44	0.681

Note : MA3 = 3-day moving average* Significant at p<0.05
MA5 = 5-day moving average** Significant at p<0.01
# CI ³ 1 *** Significant at p<0.001

Source: Jamal et al. (2004)

Table 6 Relative risk (RR) of children admissions for respiratory and cardiovascular cases at Hospital Kuala Lumpur by quartile increments of ambient NO_2concentration, 2000 – 2003 (N = 1277)

Admission cases	Lag day/ moving average	NO_2concentration increment						p
		0.017 ppm (First quartile)		0.026 ppm (Median)		0.036 ppm (Third quartile)		
		RR	95% CI	RR	95% CI	RR	95% CI	
Respiratory	0	0.88	0.81 – 0.96	0.82	0.72 – 0.93	0.76	0.64 – 0.91	0.003**
	1	0.89	0.89 – 2.53	0.84	0.84 – 4.13	0.78	0.78 – 7.13	0.827
	2	0.92	0.84 – 0.84	0.88	0.77 – 0.77	0.84	0.70 – 0.70	0.046*
	3	0.91	0.84 – 0.99	0.87	0.76 – 0.99	0.82	0.69 – 0.98	0.029*
	MA3	0.87	0.79 – 0.96	0.81	0.70 – 0.94	0.75	0.61 – 0.92	0.006**
	MA5	0.86	0.77 – 0.95	0.79	0.67 – 0.93	0.72	0.58 – 0.90	0.004**
Cardiovascular	0	0.83	0.34 – 2.03	0.75	0.19 – 2.95	0.67	0.10 – 4.48	0.683
	1	0.89	0.31 – 2.53	0.84	0.17 – 4.13	0.78	0.09 – 7.13	0.827
	2	1.57	0.51 – 4.84	1.99	0.35 –11.17	2.52	0.25 - 25.74	0.436
	3	0.78	0.31 – 1.96	0.68	0.17 – 2.79	0.59	0.08 – 4.14	0.597
	MA3	0.97	0.30 – 3.08	0.95	0.16 – 5.59	0.93	0.09 - 10.15	0.956
	MA5	0.69	0.21 – 2.30	0.57	0.09 – 3.58	0.47	0.05 – 5.57	0.549

Note : MA3 = 3-day moving average
MA5 = 5-day moving average
# CI ³ 1

* Significant at p<0.05
** Significant at p<0.01
*** Significant at p<0.001

Source: Jamal et al. (2004)

RESPIRATORY HEALTH

Asthma

Air pollution related to asthma is reported worldwide. On top of that, traffic generated pollutants are related to asthma among school children in Taiwan (Guo et al. 1999, Hwang et al. 2005, Wang et al. 1999). A moderate increase in long-term exposure to background ambient air pollution SO_2, PM_{10} and O_3 are significantly associated with an increase risk of lifetime asthma (Pénard-Morand et al. 2005).

For the criteria pollutant, some of the acute effects are the increased in respiratory symptoms and lower respiratory illness. Many epidemiological studies found that air pollutants contribute to the increase hospital admission and increase in asthma development among children (Jamal et al. 2004). The different composition of air pollutants, the dose and time of exposure and the fact that human are usually exposed to pollutant mixture than to single substance can lead to diverse impact on human health (Luke et al. 2006, Pope et al. 1995).

A community-based epidemiological study of asthma incidence rates in the USA from 1964 to 1983 showed that the rise in asthma incidence was entirely accounted by the increased incidence rates in the age range of 1 to 14 years (Yunginger et al. 1992). Hospital admission rates for asthma have also increased over time in United Kingdom and Wales, the United States, Canada, and Australia among 1 to 14 year old age group. The increase was three to ten times over a period of 15 years (1966–1981). School children were exposed to both outdoor and indoor air pollutions. In school for example, they are exposed to various kind of pollutants such as gaseous, particulate matter and microbiological agent. They spend most of their time in school which nearly six hours per day.

Unhealthy state of air pollutions may affect their health status especially among asthmatic children (Gauderman et al. 2005). Among the environmental influences that have been implicated in modifying the development of respiratory asthma are the conditions of exposure to allergens, indoor air quality and outdoor air pollution (Cai et al. 2011, Nor Husna et al. 2011, Kimber 1998). A review by Tzivian (2011), concluded that outdoor air pollution affects the appearance and exacerbation of asthma in children. Schools located in the urban areas tend to expose the students to dusts containing allergens, air particulates containing sulfate, heavy metals and others (Phipatanakul et al. 2011, Patel et al. 2011). Schools located in areas with the highest air pollution levels have the lowest attendance rates which potentially indicate poor health and a high proportion of the students did not perform well academically (Mohai et al. 2011).

In Malaysia, air pollution has been recognized as one of the major concerns that has high potential for deleterious effects on asthmatic children's health (Junaidah et al. 2010, Jamal et al. 2004, Zailina et al. 2004, Juliana et al. 2002, Zailina et al. 1997, Zailina et al. 1996b). A study by Zailina et al. (1997) on asthmatic children in urban Kuala Lumpur, Malaysia showed that the asthmatic attacks were significantly related to the PM_{10} and nitrogen dioxide levels in the air (Table 7). In this study dairy cards were distributed to doctor diagnosed asthmatic school children in the city and lived in the poor urban squatters within the city of Kuala Lumpur. They were instructed to record days that they had attacks. They reported to have more attacks on the days when the pollutants such PM_{10} and NO_2 were recorded high by the Alam Sekitar Malaysia Sdn. Bhd air monitoring stations at various locations in the city.

Table 7 Relationship between percent daily asthma attacks and ambient air pollutants in Kuala Lumpur

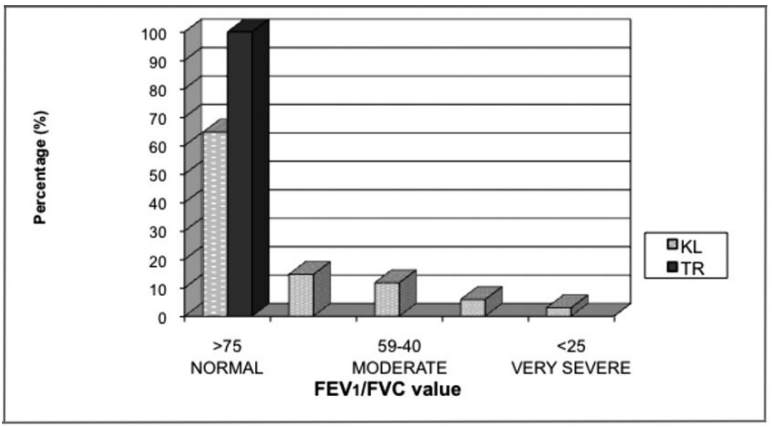
Dependent variable: Asthmatic attacks	Regression Coefficient	t	p
Constant	-0.165	-0.293	0.771
PM_{10}	0.068	3.416	0.001
NO_2	0.043		2.481
0.017			

'Multiple Regression' for Stepwise Method
probability for F to enter 0.05
probability for F to go out 0.10.
F = 55. 184, p value 0.01
r value = 0.848, R^2 value = 0.707

MODEL: ASTHMATIC ATTACKS = 0.068 PM10 + 0.043NO_2 - 0.165

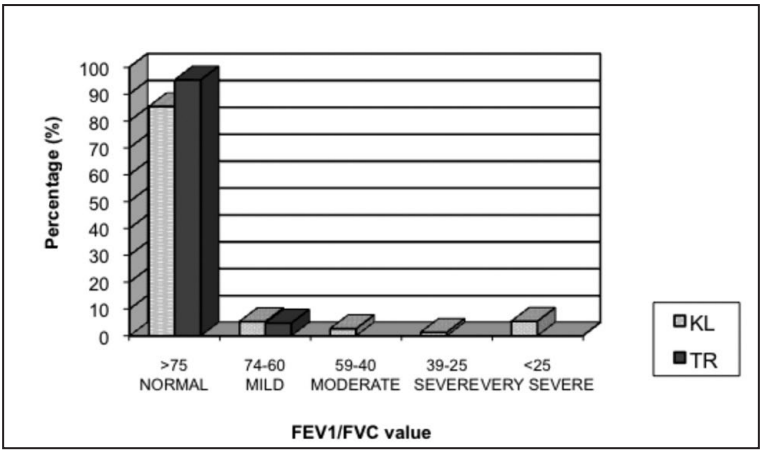
Source: Zailina et al. (1997)

The urban asthmatic children also have significantly higher symptoms than the suburban areas, and a significant correlation between the frequencies of asthma attacks with air pollutants (Zailina et al. 2004). They also showed a significantly higher lung functions impairment as well as more frequent asthmatic attacks than the children from sub-urban Bukit Kuang, Kuala Terengganu.



Source: Zailina et al. (2004)

Figure 2 Distribution of FEV_1/FEV % predicted among asthmatic boys in the study areas



Source: Zailina et al. (2004)

Figure 3 Distribution of FEV_1/FEV % predicted among asthmatic girls in the study areas

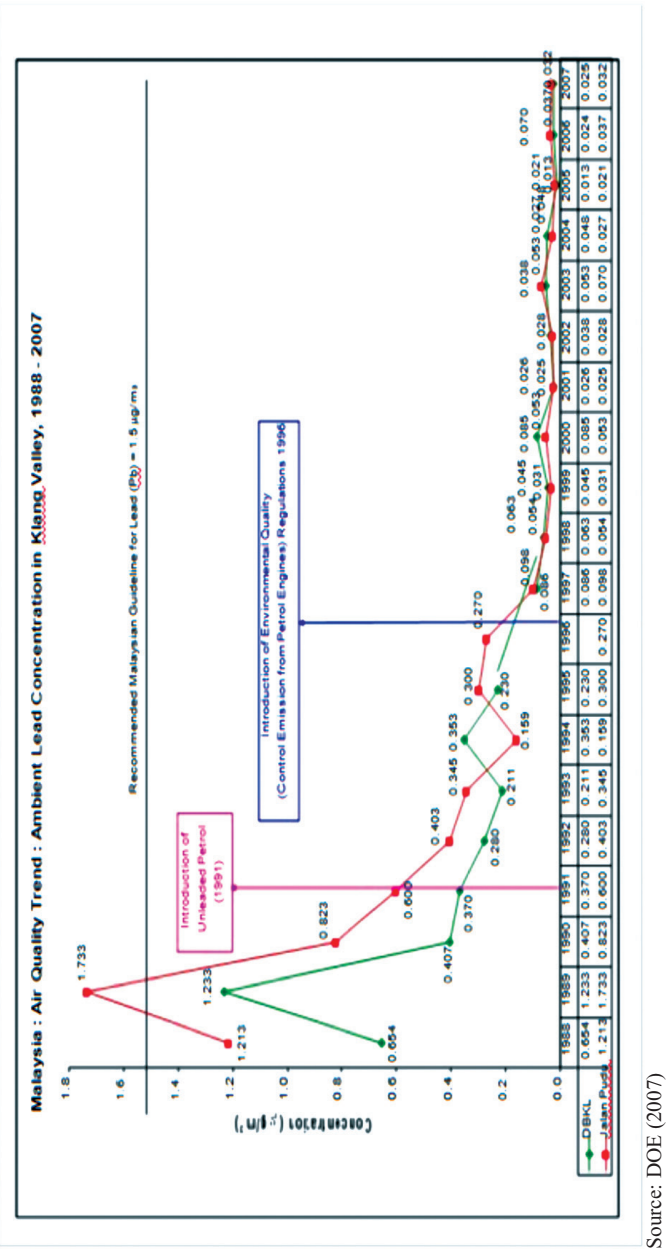
ATMOSPHERIC LEAD

Sources and Characteristics

The topography and climate of the Klang Valley has the potential to trap air pollutants. It is experiencing the worse air pollution problems due to the increase in the traffic volume, urbanizations and industrial activities. Motor vehicles are by far the major source of atmospheric lead especially in the nineties when leaded gasoline was used. In the twenties, the atmospheric lead is reduced due to the use of unleaded gasoline, however, small amount of lead is still present in the unleaded gasoline. In addition, whatever lead that has been emitted to the atmosphere settles in the terrestrial and aquatic environment. Those that settled on the terrestrial environment will be re-suspended back into the atmosphere once disturbed during the dry climate. Figure 4 shows the decline of the atmospheric lead levels from 1988 to 2007, at two locations in the city such as the City Hall and the Pudu road.

Urban vs Rural

Children living and attending schools in urban areas are more likely to be highly exposed to atmospheric lead and therefore are at high risk of experiencing chronic health effect due to lead exposure such as cognitive deficit. A study by Zailina et al. (1996a) carried out in 1992 found that children who lived in the Kuala Lumpur city center were highly exposed to atmospheric lead. This was because the ambient lead concentrations in the city centre was 16 times higher (0.483 $\mu g/m^3$) when compared to the suburban area (0.030 $\mu g/m^3$). The indoor lead concentration in the city center was also about twice higher (0.170 $\mu g/m^{3)}$ than that in the suburban areas (0.078 $\mu g/m^{3)}$. According to the report by the Department of Environment (1995), and previous studies (Zailina et al. 1996a, Jamal et al. 2000,



Source: DOE (2007)

Figure 4 Air quality trend: ambient lead concentration in Klang Valley, 1988-2007

Jamal et al. 1993), the main source of atmospheric lead was motor vehicle exhausts. The dilute acid soluble lead fraction in outdoor air was the highest compared to the strong acid and water soluble fractions, while in the indoor air, the water soluble lead fraction was the highest compared to other fractions. The dilute acid soluble fraction most likely contain halogenated species emitted from the combustion of leaded gasoline in motor vehicles (Zailina et al. 1996a, Zailina 1994).

Environmental vs Occupational Exposure

Other sources of lead would be from lead based paint which is widely used on houses, road sides and surface, playground structures and toys (Zailina and Tan 2000). The paint on the surfaces will flake in the environment can be re-suspended into the atmosphere which is inhalable to children. Table 8 shows the health effects of lead on children and adults. Children are also exposed to environmental lead from mothers who work with lead in factories since the mother's blood lead can pass through the placenta wall into the growing foetus besides the lead dust that are brought back through their work uniforms into the home environmentas as shown in Table 9 (Jamal et al., 2003). Workers who work in factories tend to live in the vicinity of the factory where they worked; therefore, their children would also be exposed to the atmospheric lead.

Table 8 Health Effects of lead on children and adults

CHILDREN	Lead concentration in blood (µg/dL)	ADULT
	150	
Death		
	100	Encephalopathy
Encephalopathy		
		Anemia
Nephropathy Anemia		
		Lifespan reduction ↓
Colic		
	50	Hemoglobin synthesis ↑
Hemoglobin synthesis ↓		Peripheral neuropathy
	40	Infertility
		Nephropathy
Vitamin D metabolism ↓	*30*	Systolic blood pressure (male) ↑
		Hearing ability ↓
		Erythrocyte protophopyrin (male) ↑
Nerve conduction potential ↓	*20*	
		Erythrocyte protophopyrin (female) ↑
Erythrocyte protophopyrin ↑		
Vitamin D metabolism ↓		
Developmental toxicity		
	10	Hypertension ↑
Intelligence quotient (IQ) ↓		
Hearing ability ↓		
Growth ↓		
Transplacental transfer		
↑ Increased function		↓ Decreased function

Source: (ATSDR 1991)

HEALTH EFFECTS OF ATMOSPHERIC LEAD

Intelligent Quotient Impairment and Neurotoxicity

Lead is a well-known neurotoxic agent that may cause severe impairment of nerve tissue, particularly in the developing central nervous system (CNS). A number of scientific evidence suggested that lead may cause damage to the CNS in the developing fetus, resulting in impairment of mental function and the induction of behavioral disorders in young children with blood-lead (PbB) levels of about 10.0 mg/dl or higher (Bellinger 1996, Dietrich, Berger and Succop 1993). Adverse effects of lead exposure on mental function in children have been demonstrated in many studies of intelligent quotient (IQ) and school performance. Lead-induced impairment of the auditory brain and cochlea is believed to contribute substantially to the mental disorders and learning disabilities associated with childhood plumbism (Bellinger 1996).

Most of the studies of lead on children emphasized on the effects on mental function (Muñoz et al. 1993, Zailina, Jamal and Shamsul 1999). Children have long been recognized as the most sensitive sub-population to lead, with developmental outcomes being the primary concern, especially cognitive and behavioral neurological impairments (Schwartz and Otto 1991, Grant and Davis 1989, Needleman and Bellinger 1994). A study by Ruff et al. (1993) found that cognitive scores are closely related to changes in children's blood lead levels over a period of 6 months after controlling for confounding factors. Needleman and Gatsonis (1990) looked at 24 studies conducted between 1972 to 1987 on the effects of lead exposures on the neurobehavioral development of children, which verified a strong relationship between low levels of lead exposure and reduced intellectual levels in children.

Table 9 The influence of mother's blood lead, socioeconomic, occupational exposure and biological factors on the children's blood lead concentrations

Independent variables / constant	Children's blood lead concentration (µg/dL)		
	Regression coefficient (β)	t value	p value
Constant (α)	-0.704	-0.112	0.911
Mother's blood lead	0.235	3.020*	0.003
Child's age	0.152	1.242	0.216
Child's weight (standardized)	-0.298	-1.444	0.151
Child's height (standardized)	0.337	1.514	0.132
Mother's race	-0.096	-1.220	0.224
Mother's age	-0.126	-1.562	0.120
Mother's education level	-0.052	-0.560	0.576
Father's education level	0.106	1.104	0.272
Household income	0.107	1.187	0.237
Mother's hygiene score	0.025	0.312	0.756
Mother's smoking	0.071	0.902	0.368

Multiple Regression test using Enter method
$R^2 = 0.112$
* significant at $p < 0.05$

Source: Jamal et al (2003)

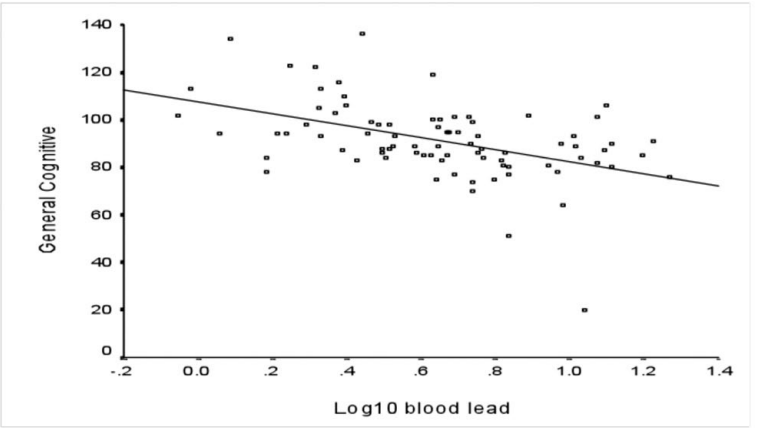
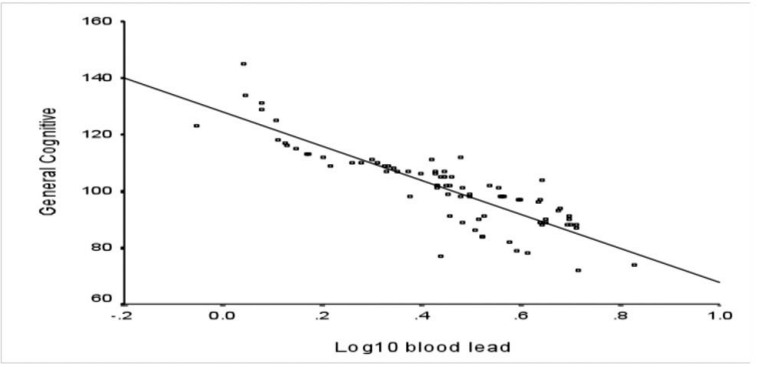


Figure 5 Scatterplot of GCI (IQ) with $\log_{10}$ blood lead of urban children



Source: Zailina et al. (1999)

Figure 6 Scatterplot of GCI (IQ) with $\log_{10}$ blood lead of suburban and rural children

Needleman et al. (1990) conducted a replicate study on 132 children he had earlier studied from 1975 to 1978. The later study showed that neurobehavioral effect was still strongly related to the dentine lead level in the study respondents. These studies indicated

that childhood lead exposures can interfere with the functions of the nervous system until adulthood.

The central and peripheral nervous systems are the main targets of lead toxicity. Effects to the central nervous system include impairment of psychological and neurobehavioral functions. In adults, neurological effect of lead is mainly on the peripheral nervous system which can result in peripheral motor neuropathy, axon degeneration and slow nerve impulse conduction. Among children, lead toxicity mainly affects the central nervous system. In more severe cases, lead toxicity can cause coma, convulsion and even death (Mahaffey 1981). Lead toxicity can disrupt neurological development such as impairment in IQ development including cognitive functions (ATSDR 1991). Behavioral and neuropsychological disorders may show immediate effect at blood lead levels as low as 10 mg/dL (Jamal et al. 1996). Other than behavioral disorders, the fatigue syndrome and loss of concentration could also occur.

Chronic lead toxicity can cause encephalopathy, convulsion, cerebral edema and coma (ATSDR 1991). Overt effects on the nervous system such as wrist drop (weakness of the wrist and finger extensors caused by the compression of a nerve), require blood lead levels of 60 mg/dL or greater (Juberg, Kleinman and Kwon 1997). Lead can also penetrate the placental wall and affects fetal development resulting in low birth weight and premature birth (ATSDR 1991).

The mean blood lead of the urban children was significantly higher than those suburban children indicating that the urban children were more at risk of lead exposure (Jamal, 2000). In general, the IQ of the suburban and rural children was better than the urban children because all the mean scores was higher than their urban counterparts (Zailina et al. 1999). There was an inverse relationship between blood lead and IQ scores for all the children

studied. The coefficient of determination values (R^2), indicated that for urban children, 21.9% of the variability in the IQ was due to the blood lead, while for suburban and rural children, 74.0% of the variability was due to blood lead . This is probably due to multiple factors beside blood lead which affects cognitive scores such as educational facilities, parents' education, socioeconomic background and other environmental factors whereby these factors were lacking in the suburban and rural areas. There is probably lack of social and physical facilities for enhancing IQ development in these areas. Therefore, even though the mean blood lead concentration was low in the suburban children, it had a significant relationship with their IQ scores.In the urban area, 1 mg/dl of blood lead reduced the intelligent quotient score by 0.602 while in the suburban and rural areas, 1 mg/dl of blood lead reduced the score by 5.4 (Zailina at al. 1999). The deficit in IQ occurred even when the blood lead is at very low concentrations and the suburban and rural children's IQ are more sensitive to lead despite other social or confounding factors (Zailina et al. 2008), Zailina et al. 2011).

Table 10 Relationship between General Cognitive Index (IQ) scores with blood lead and socio-economic factors for urban and suburban children

	Coefficient of regression (β)		t-value		p-value	
	[a]Urban	[b]Suburban	[a]Urban	[b]Suburban	[a]Urban	[b]Suburban
Constant	95.926	123.201	17.065	53.231	<0.001	<0.001
Blood lead	-24.047	-57.840	-4.659	-14.972	<0.001	<0.001
Mothers' education	1.212	-	2.757	-	0.007	-
Household income	-	0.005	-	3.372	-	0.001

[a]*n = 82*
'Stepwise' method: probability for F enter ≤ 0.05; probability for F exit ≥ 0.10
F value = 15.894 $p < 0.001$
r value = 0.538 $R^2 = 0.291$
[b]*n = 80*
Stepwise' method : probability for F enter ≤ 0.05; probability for F exit ≥ 0.10
F value = 131.525 $p < 0.001$
r value = 0.880 $R^2 = 0.774$

Source: Zailina et al. (1999)

Low Blood Lead

Monitoring of ambient lead is currently the responsibility of the Department of Environment through Alam Sekitar Sdn. Bhd. Routine blood lead monitoring is not carried out on children in schools unless there is a funded research project. The effect of lead on children's IQ has been documented, especially at higher levels of blood lead. Studies (Rummo et al. 1979, Fulton et al. 1987) reported that the blood lead levels have an inverse relationship with cognitive development, however, at blood lead levels exceeding the permissible level of 10 µg/dL. Findings from Zailina et al. (2011) are contrary to these, whereby the mean blood lead lower than the permissible level, significantly influenced the children's IQ (Table 11). This study also determined the relationship between blood lead levels and IQ (intelligent quotient) of children measured by the general cognitive development test from the McCarthy of Children's Abilities Test (MSCA).

Table11 Relationship between the IQ with blood lead for all children

Dependent variable (General Cognitive Scores)	**Coefficient regression *ß***	***t* value**	***p* value**
(Constant)	112.958	35.466	< 0.001
Log_{10} blood lead	-0.292	-6.245**	< 0.001
Fathers' education	-0.078	-1.115	0.266
Mothers' education	-0.090	-1.337	0.182
Household income	-0.007	-0.126	0.900
Number of siblings	0.015	0.179	0.858
Sequence in family	-0.073	-0.883	0.378
Length of stay	-0.024	-0.126	0.606

N=413
**Significant at $p \leq 0.01$
Regression method : Enter
F value = 13.414, $p < 0.001$
r value = 0.360, $R^2 = 0.130$
Source: Zailina et al. (2011)

The mean blood lead of children in Zailina et al. (2011) was lower compared to a local study with 4.30μg/dL) (Zailina et al. 1999). The mean was low since the objective was to determine the influence of blood lead on cognitive scores, therefore, those children with higher than 10μg/dL blood lead were excluded from the study (5.5%). The mean blood lead was low compared to some other research findings in other countries (Bonilla and Mauss 1998, Muñoz et al. 1993, Heinze et al. 1998). Studies have shown that there is no "safe" level of lead exposure and even the current "low" level of exposure in children is associated with neuro-developmental deficits (Bellinger 2008, Pamela et al. 2007). Primary prevention of exposure provides the best hope of mitigating the impact of this preventable disease. The lower blood lead concentrations could be due to lower lead emitted to the atmosphere from motor vehicles, with the reduction of lead content (from 0.40g/L to 0.15g/L) in gasoline during the early 90's (Zailina et al. 1996a) and the ban of leaded gasoline in 1998. The same blood lead trends were also reported in the National Health and Nutrition Examination Survey in the United States (Pirkle et al. 2008).

Bone Development

Bone is a major organ for lead deposition and skeletal lead has been used as a measure of cumulative lead exposure (Pounds, Long and Rosen 1991). Lead has also been suggested to affect bone function by altering growth and stature, and by perturbing vitamin D metabolism (Juberg et al. 1997). For lead in bone, the most recent estimate of its half-life is 9 to 12 years with a shorter half-life of approximately one year for trabecular bone (Brito et al. 2000, Skerfving et al. 1995). Lead isotope studies have shown that the mobilization of bone lead is enhanced during pregnancy, lactation and trauma such as fractures (Mushak 1998). Further,

this experimental approach suggests that in cases where levels of environmental lead exposure and dietary lead intake are low, skeletal contribution is dominant, especially during pregnancy and postpartum (Smith, Osterloh and Flegal 1996, Gulson et al. 1995). Other effects such as growth retardation, have not been given due attention. Since lead is known to accumulate in the bones, high lead concentrations in the body would most likely affect children's physical growth. Studies carried out (Kafourou et al. 1997, Shukla et al. 1989) have shown that lead in the body affects children's growth. However, study by Zailina et al. (2008) did not show significant correlations between blood lead levels with physical development, mainly because these children were 5-7 years old and they were in their early period of their physical development, thus no difference can be seen explicitly (Table 12).

Nutritional Aspects

Children are also at risk of being malnourished due to the high metabolic processes for growth and development of their body. They are often regarded as a vulnerable group, because the combination of exposure to environmental pollutants and malnutrition will put them at a higher risk of getting adverse health effects.

The relationship between lead and nutritional status has created interest among researchers since a few decades ago. Nutritional status may modify the impact of an environmental toxicant. According to Mahaffey (1995), four nutritional conditions which increased the adverse consequences of environmental lead exposure were irregular food intake, high fat intake, marginal calcium intake and subtle iron deficiency. Among adults, ingestion of lead during fasting results in a greater absorption of lead than if lead were ingested with food (Rabinowitz, Kopple and Wetherill 1980). Though there are no similar data available for children, these effects

Table 12 Correlation between blood lead and anthropometric measurements

Variable	Blood lead concentrations (mg/dl)					
	All (n=268)		Urban (n=168)		Industrial (n=100)	
	r	p value	r	p value	r	p value
Height for age	-0.071	0.244	-0.095	0.219	-0.037	0.716
Weight for age	-0.011	0.856	0.019	0.806	-0.063	0.535
Weight for height	0.060	0.328	0.136	0.079	-0.069	0.493
Left arm circumference	0.007	0.907	0.041	0.595	-0.055	0.587

Source: (Zailina et al. 2008)

on adults can be extrapolated to children. Children probably have increased lead absorption following even shorter fasting periods than adults because they have more rapid gastric emptying times than adults. Due to this, the fasting or non-fasting retention of lead is likely to be even more important in children (Mahaffey 1995). Previous studies showed that high fat intake (Lucas, Sexton and Langenberg 1996, Gallicchio, Scherer. and Sexton 2002) and low micronutrient intake such as calcium (Mahaffey et al. 1976, Saliza et al. 2007) are correlated with high lead level in the body (Table 13). This proves that poor nutritional status could enhance lead toxicity. The absorption and retention of lead in the gastrointestinal tract is the main pathway of lead intake. These processes depend on the micronutrient status, including calcium, in the gastrointestinal lumen. Studies found that, insufficient dietary calcium could increase the absorptionand retentionof lead in the intestine. Since calcium and lead share the same number of valency, these elements compete for the same binding site on the intestinal mucosal protein, which is the most important absorption process (Mahaffey, Hasemen and Goyer 1973). The competition for the binding site could be the reason why insufficient calcium intake could increase lead absorption in the intestine.

Table 13 Variables that most influence the blood lead concentrations among children with deficient calcium intake

Variables	ß-value	t-value	p-value
(Constant)	11.863	5.373	p<0.001
Calcium (mg)	-0.013	-2.931	0.004
Age (years)	-0.674	-2.712	0.008

Multiple regression: Enter R=0.429; R^2 =0.184; Ad. R^2 =0.162
F=8.441; p<0.001 N=78

Source: (Saliza et al 2007)

It can be concluded that low calcium intake would enhance the absorption and retention of lead, thus increasing the risk of lead toxicity. Therefore, it is suggested that sufficient calcium intake would enhance body resistance against the risk of lead absorption especially among children. We have to ensure that children get sufficient calcium in their food. Nutritional status surveys could be introduced in schools to assess this. Involvement of parents, school authorities and government are needed to educate the children on the importance of consuming nutritious food, and on how to choose good and healthy food for consumption.

Hearing Impairment

Lead-induced impairment of the auditory brain and cochlea is believed to contribute substantially to the cognitive disorders and learning disabilities associated with childhood plumbism (Bellinger 1996). Lead exposed children and occupationally lead exposed adults develop auditory brainstem abnormalities and significant hearing loss (Schwartz and Otto 1987, Schwartz and Otto 1991).

Zailina et al. (2005), findings showed the prevalence of low frequency hearing impairment (LFHI) and high frequency hearing impairment (HFHI) in School A and B children were in a range of 1.9 – 10.9% and 4.6 – 13.5% respectively for both unilateral and bilateral impairment. The hearing impairment was higher in School B than School A children due its location near to a main road and a light rapid transport (LRT) rail. There was also a significant relationship between medical history scores with both ears thresholds at high and low frequencies in all the studied children. The relationship between blood lead levels with hearing thresholds at high and low frequencies unilaterally were statistically significant among School A children who had higher blood lead levels concentration (Table 14). Findings showed hearing impairment in

Table 14 Relationship between hearing thresholds and selected variables for School A children

Dependent variable: Mean hearing thresholds at HPTA	Right ear				Left ear			
	F		p		F		p	
	LPTA	HPTA	LPTA	HPTA	LPTA	HPTA	LPTA	HPTA
Corrected model	3.727	3.386	0.004	0.007	4.301	5.922	0.001	<0.001
Log_{10} PbB	1.704	1.543	0.195	0.217	4.279	9.463	0.041	0.003
Daily activities scores	0.105	0.617	0.747	0.434	0.675	1.755	0.413	0.188
Medical history scores	15.289	13.652	<0.001	<0.001	12.914	14.852	0.001	<0.001
House environment scores	0.149	0.611	0.701	0.436	0.009	0.135	0.923	0.714
Household income	0.022	0.117	0.884	0.733	0.931	0.252	0.337	0.617

LPTA=low pure tone average HPTA=high pure tone average

R^2 (Adjusted R^2) (LPTA) 0.158 (0.116) 0.178 (0.137)

(HPTA) 0.146 (0.103) 0.230 (0.191)

Source:(Zailina et al. 2005)

these children are mainly influenced by their medical history due to their poor socioeconomic background, however, lead as well as their daily activities significantly contributed to the problem.

The urban atmospheric lead concentration was found to be about 16 times higher than the suburban areas. The dilute acid soluble fractions most likely contain halogenated species emitted from the combustion of leaded gasoline in motor vehicles (Zailina et al. 1996a). Therefore, children who live and attend schools in the urban areas are most likely to be highly exposed not only to noise but also to atmospheric lead which most likely affect their auditory nerve which lead to hearing impairment

INDOOR AIR QUALITY

Good indoor air quality is desired for a healthy indoor environment. There are numerous situations in homes and schools which may exposure individuals to contaminants.The parameters of an indoor environment are summarized as follows: chemical contaminants, such as carbon dioxide, carbon monoxide, formaldehyde, volatile organic compounds and environmental tobacco smoke (ETS); physical conditions, such as air temperature, air velocity, humidity and radiation such as radon; biological agents, such as mites, virus, and spores. Indoor air quality is influenced by the concentrations of outdoor air pollutants, indoor sources of pollution, characteristics of the building and the habits of the residents. Indoor air pollution may arise from the use of open fires, unsafe fuels or combustion of biomass fuels, coal and kerosene. Gas stoves or badly installed wood-burning units with poor ventilation and maintenance can increase the indoor levels of carbon monoxide, nitrogen dioxide and particles. Other pollutants not associated with fuel combustion include building materials such as asbestos and cement, wood preservatives and others.Volatile organic compounds may be

released by various sources including paints, glues, resins, polishing materials, perfumes, spray propellants and cleaning agents. Formaldehyde is a component of some household products and can irritate the eyes, nose and airways. Indoor air also contains a complex mixture of bio-aerosol such as fungi, bacteria and allergens, as well as non-biological particles including products from various combustion processes (Nor Husna et al., 2011).

Home Environment

Indoor air pollution study in rural and urban houses in areas of Selangor showed higher forced vital capacity % (FVC%) predicted and forced expiratory volume per second % (FEV_1%) predicted for the boys and girls in the rural compared to the urban Kajang areas. The urban children also have significantly higher respiratory symptoms such as chronic cough, chronic phlegm, chest illness and persistent wheezing (Juliana et al. 2001). The common indoor sources of pollutants are cigarette smoke, mosquito coil use, cooking and animal fur.

Mosquito coils may represent a serious potential threat to children's health. Prolonged use has been associated with increased incidences of asthma and persistent wheezing in children. Although the active ingredient is usually small amounts of pyrethrins (considered a low-toxicity insecticide), over 99% of the mass of the coil is so-called "inert" ingredients. Liu et al. (2003) findings showed that burning mosquito coils indoors generates smoke that can control mosquitoes effectively. This practice is currently used in numerous households in Africa, Asia and South America. However, the smoke may contain pollutants of health concern. This study characterized the emissions from four common brands of mosquito coils from China and two common brands from Malaysia. The mass balance equations was used to determine emission rates

of fine particles (particulate matter < 2.5 μm in diameter; $PM_{2.5}$), polycyclic aromatic hydrocarbons (PAHs), aldehydes and ketones. It was found that pollutant concentrations resulting from burning mosquito coils could substantially exceed health-based air quality standards or guidelines. Under the same combustion conditions, the tested Malaysian mosquito coils generated more measured pollutants than did the tested Chinese mosquito coils. A large suite of volatile organic compounds, including carcinogens and suspected carcinogens, in the coil smoke was identified and the size of the particles were ultrafine and fine. The findings suggested that exposure to the smoke of mosquito coils similar to the tested ones can pose significant acute and chronic health risks. For example, burning one mosquito coil for 2 hours would release the same amount of $PM_{2.5}$ mass as burning 75–137 cigarettes. The emission of formaldehyde from burning one coil can be as high as that released from burning 51 cigarettes.

School Environment

The classrooms' indoor air quality has caught attention because children are more susceptible to poor air quality, which cause easily recognizable impacts on health (USEPA 1996). This is due to the fact that poor indoor air quality (IAQ) can reduce the student's health, productivity and ability to perform specific mental task requiring concentration as they may appear sleepy, coughing, dizzy and experienced other respiratory illnesses (Moglia et al. 2006). Besides, physically developing and growing children are more vulnerable and likely to suffer from the consequences of indoor air quality. Children spend a large portion of their time in school during weekdays which exposed them to the indoor air pollutants in school (Meklin et al. 2002). The indoor particle generation and

outdoor air filtration are key factors influencing the indoor particles' concentration.

Indoor particle generation can due to the human activities or specific sources such as occupants of the building (Ramachandran et al. 2000). Previous findings also have proven that indoor occupants increased the indoor particle levels (Poupard et al. 2005). Marzuki, Nur Zafirah and Ahmad (2010) showed there were differences on particulate levels with time depending on the change of activities inside the classrooms. Human activities in coastal or industrial areas had higher ambient PM_{10} concentrations, however, the indoor PM_{10} was higher than the outdoor PM_{10} as measured due to the housekeeping activities inside the classrooms such as sweeping, cleaning habits or writing and erasing of blackboards. The indoor particle generation and outdoor air filtration are key factors influencing the indoor particles' concentration. However, Nur Zafirah and Marzuki (2011) found that indoor air quality in school was significantly influenced by outdoor air concentration as there was significant strong correlation between indoor air and outdoor air concentrations, besides the indoor air concentrations were influenced by human activities. The major sources of man-made outdoor concentration may be vehicular emissions and industrial activities while, the natural airborne dust and sea spray were the main sources since these schools are located near the coast.

Norbäck, Torgén and Edling (1990) found the average concentration of CO_2 was above 800 µl/L in all 6 primary schools studied, which indicated there was a lack of ventilation in the school. This study also found high VOC concentration at high temperature, while the PM_{10} concentrations were relatively high in classrooms with low ventilation systems and high humidity. There was also found significant relationship between PM_{10} concentrations and eye irritation.

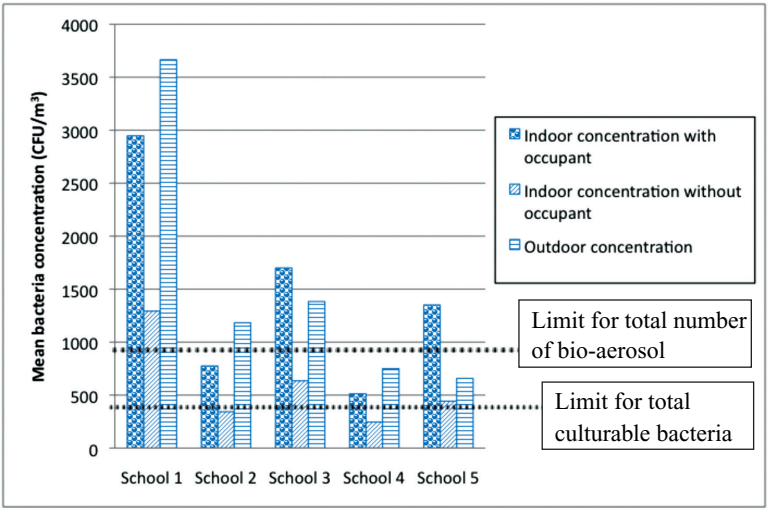
Microbes in Classroom

Exposure to these indoor air contaminants particularly among school children needs tremendous attention as the children are more susceptible to the infection as well as respiratory problem as they spend large portion of their weekday time in school. Schools have been considered as the most important indoor environment for children besides home. Research findings reported possible health effects of school environment and microbial exposures in school as potential risk factors for respiratory symptoms, asthma and allergy among children (Kim et al. 2007, Meklin et al. 2005).

Several studies have shown that the most common fungal genera in school buildings were *Penicilium*, yeast, *Cladosporium* and *Aspergillus* (Meklin et al. 2002). *Penicilium* are common in ambient environment with high carbon dioxide (CO_2) concentration that frequently shows inadequate ventilation of a building which affects the students' concentration and teachers' productivity. High concentration of *Cladosporium* and *Aspergillus* has been proven to be associated with few allergic symptoms in respiratory systems (Fischer and Dott 2003, Aydogdu and Asan 2007). Moreover, airborne concentrations of *Cladosporidium, Epicoccum, Coprinus* and *Fusarium* spores are associated with peak expiratory flow rates (PEFRs) deficiency in children showing decrease in lung function (Neas et al. 1996).

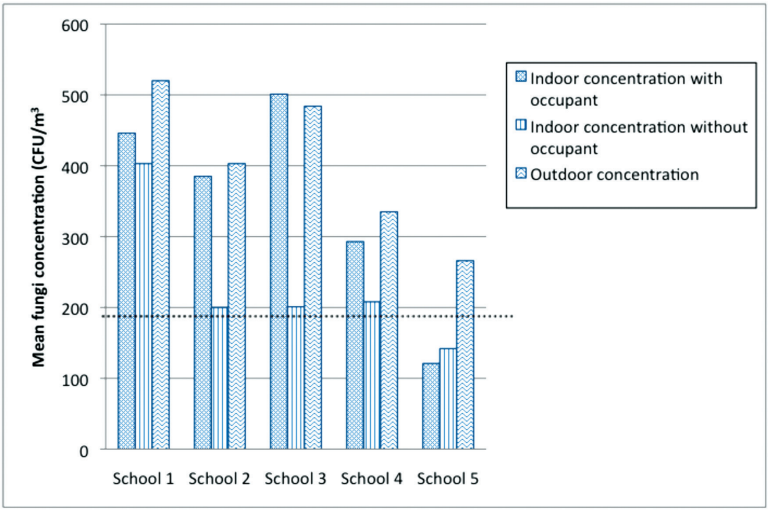
Nor Husna et al. (2011) found high indoor bio-aerosol concentrations in schools which reflected their state of cleanliness and over crowdedness. Outdoor air bio-aerosol concentrations were contributed by the nearby activities. The indoor bacteria and fungi concentrations in schools with occupants were significantly higher than those without occupants, due to the contamination sources from the occupants. The ratio of indoor to outdoor bio-aerosol concentrations was below 1.0 except for some schools indicating the

state of indoor conditions that would provide favorable conditions for both bacterial and fungal growth. More than half of the indoor bacteria concentrations (56%) exceeded the ACGIH and WHO recommended level of 500 CFU/m^3 while 33.8% of the indoor fungal samples exceeded the recommended level of 200 CFU/m^3. This indicated poor microbial indoor air quality in the schools and inferring potential harmful effects to the children's health (Figures 6 and 7).



Source: Nor Husna et al. (2011)

Figure 6 Mean indoor and outdoor bacteria concentrations with and without indoor occupants



Source: Nor Husna et al. (2011)

Figure 7 Mean indoor and outdoor fungi concentrations with and without indoor occupants

Findings from Nor Husna et al., (2011), showed higher prevalence of chronic cough (29.9%), wheeze (20.9%) and doctor diagnosed asthma (8.5%) among school children, as compared to previous studies conducted in Kuala Lumpur and Kota Bharu (Omar 1990, Quah et al. 2005). Findings indicated that respiratory symptoms are increasing as urbanization influenced children's health.

Some of the respiratory symptoms reported among respondents were found to be statistically associated with the bio-aerosol exposure in the school environment. Fungi bio-aerosol exposure seemed to be the most prominent factor associated with respiratory symptoms as compared to the bacteria (Table 15). The studied school buildings had signs of dampness and growth of mold and fungi in the ceiling of the classrooms and washrooms. Mold is often the culprit in indoor air quality issues. Mold produces allergy

symptoms and in some cases weakens the body's natural defenses. Exposure to mold can result in developing other allergies and hypersensitivity to allergens. According to Zhao et al. (2006) dampness was one of the indoor environmental factors associated with children's respiratory health. These observations were explained by the responses that the respondents felt better after school. Bacteria and fungi bio-aerosol concentrations show different trend in the association with respiratory symptoms reported. The prevalence of respiratory symptoms including wheezing and whistling in chest and chronic cough with phlegm were found to be significantly associated with fungi concentrations in school, while nasal symptoms and itchy, watery eyes and nose were found to be significantly associated with both bacteria and fungi concentrations.

Table 15 Association between current respiratory symptoms and bioaerosol concentrations in the school for the last 3 months

Symptoms at school in the last 3 months	**Fungi**		**Bacteria**	
	X^2	**p**	**X^2**	**p**
Fatigue/ weakness	33.79	<0.001***	13.867	0.001*
Difficulties in breathing	29.85	<0.001***	9.552	0.008*
Chronic cough	23.53	<0.001***	0.917	0.632
Sore throat	31.99	<0.001***	3.251	0.197
Dry throat	37.08	<0.001***	8.705	0.013*
Headache	31.19	<0.001***	5.320	0.070
Nausea	17.52	<0.001***	5.862	0.054*
Sneeze and flu	9.92	0.007*	1.326	0.515
Itchy watery eyes	22.71	<0.001***	7.665	0.022*
Itchy face	49.74	<0.001***	6.122	0.047*

***significant at p<0.001
*significant at p<0.05

Source: Nor Husna et al. (2011).

Findings from Cai et al. (2011), showed that fungal DNA and cat allergen were common in the studied Malaysian schools and there were high prevalence of both doctor-diagnosed asthma and respiratory symptoms among the students. There were significant association between specific DNA from the fungal species *A. versicolor* and the bacteria *Streptomyces* spp. and respiratory health illustrating the importance of analysing specific microbes when studying respiratory effects.

Respiratory symptoms, daytime breathlessness (exercise or rest), parental asthma or allergy, doctor-diagnosed asthma, and pollen or pet allergy were commonly reported by the secondary schools students in Johor Bahru, Malaysia. Night-time breathlessness, asthma in the last 12 months, medication for asthma, and smoking were however, rarely reported. Moreover, girls reported higher prevalence of some respiratory symptoms such as breathlessness, respiratory infections, and allergy than boys. *A. versicolor* DNA was a risk factor for both wheeze and daytime breathlessness, and *Streptomyces* DNA was a risk factor for doctor-diagnosed asthma (Cai et al. 2011).

There was a significant difference in the prevalence of respiratory symptoms and infections among girls and boys (Table 16). More girls reported daytime breathlessness (exercise or rest), morning breathlessness, or airway infections in the last 3 months compared to boys. Moreover, there was a higher rate of prevalence of pollen and/or pets allergy in girls. Similar gender differences have been reported among students in Taiyuan (Zhao et al. 2008) and Shanghai (Mi et al, 2006) in China. These studies showed relationship between dampness, mould and lung functions. Apart from airways symptoms, findings demonstrated the presence of general symptoms that include fatigue, headache and symptoms from the central nervous system.

Table 16 Prevalence of respiratory symptoms and infections among male and female students (N=462) in 8 lower secondary schools in Johor Bahru, Malaysia

Respiratory symptoms and infections	**Male N (%)**	**Female N (%)**	**Overall N (%)**	***p*-value**
Wheeze	21 (10)	26 (11.0)	47 (10)	0.58
Breathlessness during wheeze	11 (5)	20 (9)	31 (7)	0.33
Breathlessness at rest	13 (6)	32 (14)	45 (10)	0.005
Breathlessness after exercise	65 (29)	101 (43)	166 (36)	0.003
Daytime breathlessness (exercise or rest)	72 (32)	114 (48)	186 (41)	0.001
Night-time breathlessness	12 (5)	20 (9)	32 (7)	0.19
Morning breathlessness	7 (3)	18 (8)	25 (6)	0.04
Respiratory infections in last 3 months	27 (12)	60 (25)	87 (19)	<0.001
Respiratory infections in last 12 month with antibiotic treatment	21 (10)	33 (16)	54 (13)	0.09

Source: Cai et al.(2011)

Hypersensitivity to moulds is immediate (type 1) and includes: acute asthma, allergic rhinitis and rarely urticaria (hives). Colonization associated with chronic asthma is rarely serious,

life-threatening diseases are not caused by common household moulds. The major exception is aspergillosis. Moulds usually infect devitalized tissue or an immune compromised host. Mycotoxins are associated with human disease and cause acute and chronic effects.

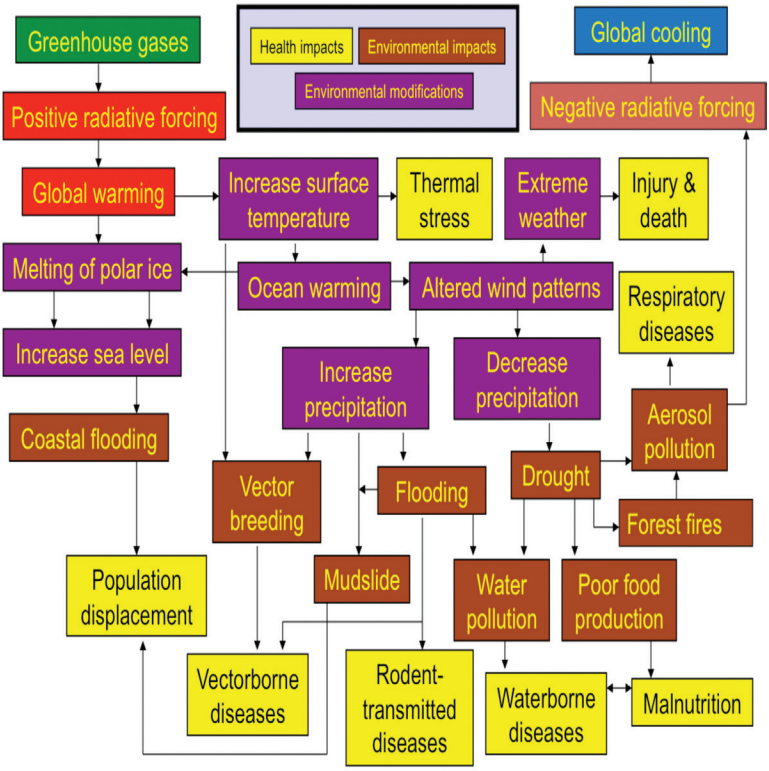
CLIMATE CHANGE

The World Health Organization recently estimated that 34% of all childhood illness in the world (compared to 24% of all age illness) and 36% of deaths in children under age 14 are due to modifiable environmental factors. Climate change increases these hazards by worsening air quality, stimulating more extreme weather events, creating conditions that favour the increase in food-, water- and vector-borne infections, and enhancing heat stress conditions as shown in Figure 8 (Jamal and Zailina 2012). Air quality is threatened via at least three mechanisms: heat-driven increases in ground-level ozone, energy production-driven increases in particulates and other fossil fuel-related air pollutants, and changes in aeroallergens. Climate change will affect the weather which then potentially changes the air quality in terms of the air pollutants. Numerous pediatric respiratory problem related to air pollutants will potentially change as climate changes (Silverman and Ito 2010; Trasande and Thurston 2005)

Health Impact

Ozone is expected to increase in some regions, and other pollutants, such as nitrogen oxides, particulate matter, and sulfur oxides, will also potentially change because the warming temperatures can affect chemical reaction rates and pollutant transport mechanisms (Ebi and McGregor 2008; Kinney 2008). Wildfires, also a climate-sensitive exposure, can generate significant particulate matter and have been

documented to increase risk of respiratory effects and eye irritation among children in affected areas (Kunzli et al. 2006). There will be an increased in air pollution related illness, respiratory diseases and heat related which are potentially fatal.



Source: Jamal and Zailina, 2012

Figure 8 Health effects of global climate change

Ground level ozone is formed by the action of sunlight on nitrogen oxides and volatile organic compounds emitted from motor vehicles and other sources. Ozone production is favored at high temperatures even without additional precursor pollutants. Ozone exposure increases the rate and severity of asthma attacks and may play a causal role in asthma onset when exposures are high and prolonged. With the population growth and warmer climate, the increased in energy demands will be met by burning more fossil fuels. The increased burning of fossil fuel will produce more major air pollutants including ozone. Studies clearly show that childhood exposure to specific air pollutants is related to decreased lung growth and permanent decrements in pulmonary function as well as increases in respiratory infection, asthma (Junaidah et al. 2010, Jamal et al. 2004, Zailina et al. 2004, Juliana et al. 2002, Zailina et al. 1997, Zailina et al. 1996b), infant mortality (Hajat, et al., 2007) and all age mortality, miscarriages, preterm delivery (Leem et al., 2008) and low birth weight (Bell et al. 2007).

Allergic Diseases from Allergens

Finally, we expect that climate change will result in changes in the quantity, quality and distribution of pollens and other aero allergens. For example, poison ivy grown at high carbon dioxide concentrations is more allergenic than poison ivy grown at current outdoor levels. Ragweed pollen production increases with increased CO_2 concentrations and temperature. Thus, asthma and allergies are likely to be worsened regionally in a warmer world. To the degree that allergy plays a role in the causation of asthma, we can expect that more asthma will result from increases in aeroallergen exposure. Weed pollen, shown to increase in climate change simulation studies, and grass pollen have been associated with children's asthma exacerbations, emergency department visits,

and hospitalizations (Schmier and Ebi 2009; Ziska et al. 2008). The last several decades have shown an increasing global trend in the incidence of asthma and potentially other allergic diseases, and some hypothesize that increased aeroallergen exposure from climate change might be partially responsible (Beggs and Walczyk 2008).

ENVIRONMENTAL HEALTH RISK ASSESSMENT APPROACH

Environmental Health Risk Assessment

The US EPA uses risk assessment to characterize the nature and magnitude of health risks to humans and ecological receptors (e.g., birds, fish, wildlife) from chemical contaminants and other stressors, that may be present in the environment (US EPA, 2010). Risk assessment has been used as an approach for organization of information about hazard on health and safety which are being used in many countries with various approaches. Health risk assessment if properly conducted provides information on the magnitude of threat or uncertainty.

Limitations

It provides the framework on the estimation of environmental threats to children. However, it has its limitation as far as children's environmental health issues are concerned since the current approach in the determination of permissible exposure level or intake for an individual may not be safe for children who may be faced with higher risk than an adult. The modeling of exposure to a hazard in quantitative health risk assessment is based on a number of assumptions such as the average human body weight, rate of respiration, rate of food ingestion, the frequency and duration of exposure which may be different for children. The risks to infants

and children are not consistently and explicitly considered as a part of risk assessments generated during its decision-making process, including the setting of standards to protect public health and the environment. There has been concern that the current approach of determining acceptable exposure levels or intake for an individual may not a safe intake limit for children (Samet, 2004).

Children have more time to develop chronic diseases triggered by early exposure as they have more future years of life than most adults. Toxicants in the environment require many years to develop diseases. Carcinogens and toxic agents exposure sustained in early life, including prenatal exposures, in many cases, appear more likely to lead to diseases later in life. Risk assessment continues to develop in the ability to consider the special exposures and the unique susceptibilities of children.

The scientific basis on how, when, and by how much children differ from adults in their susceptibilities to environmental hazards should be studied and further understood, so that risk assessment will continue to improve on the identification, understanding, control and prevention of health problems or diseases in children. The development of child specific risk assessment methods, improved mechanism of underlying children's sensitivity to environmental toxicants, and child specific toxicity and exposure information are all important considerations. At presence, there is a continued reliance on the 10-fold safety factor when the true differences between children and adults may be smaller or larger (NACEC, 2003)

FUTURE DIRECTIONS

Roles of Parents, Teachers and Related Ministries

On individual basis, parents should be aware of the vulnerability of their children to environmental hazard especially sensitive children with asthma. Through health service establishment parents need to be educated on the environmental hazards such as air pollution. Parents can work closely in PIBG with schools in promoting healthy environment through various existing programmes in the schools.

The Ministry of Education with the existing "Program Bersepadu Sekolah Sihat "(PBSS) which was officially launched in September 1997 may use the programmes as a mechanism with the aim of having a safe, healthy and quality environment for school children in achieving the Malaysia 2020 Vision. Basically, under PBSS, which had 6 cores of emphasis; include the school health policy, physical and social environment, community involvement, self-health skills and school health program. Therefore, the environmental health and hygiene aspects of the school environment can be included into this programme.

The Ministry of Health introduced the School Health Programme in 1967 in all government schools nationwide. In this programme, teachers are the main role in making it a success. Teachers are responsible for multiple functions such as screen for vision defects, measurements (height and weight), manage school health records and liaise with parents. The concept of health promoting school was to build a school with healthy setting for living, learning and working. In 2003, WHO had set 5 main characteristics of health promoting school.

1. Fosters health and learning
2. Engages health, education offices, teachers, parents, community and students to promote health

3. Strives to promote a healthy environment
4. Implements policies, practices and other measures that respect individuals' self esteem, opportunities for success, acknowledge good efforts and personal achievements
5. Strives to improve health of school personnel, families and community.

For quality control, school health programme has been created through a capacity-building and work is guided by written standard operating procedures. Parents can be involved in such activities in order to educate themselves as well as for the human resource and capacity cooperation.

The Ministry of Education could provide a buffer zone between schools and its human activities when building up new school (especially for those in industrial and heavy traffic areas). Preliminary data on air quality status of the planner new schools should be conducted in order to determine the baseline concentration of PM_{10} while for existing schools, mechanical ventilation system should be installed or improved to ensure conducive and comfortable teaching and learning environment.

The Department of Environment in their policy statement under the 9th Malaysia Plan noted that a new Clean Air Action Plan will be developed and implemented to improve on air quality. The strategies include reducing emission from motor vehicles and industries. The Department of Transportation policy on public transport would be to upgrade the quality of transport infrastructure nationwide, improve public transport services and encourage public to use public transportation. As a result, these will reduce the number of vehicles on the road, ease traffic congestion and finally reduce air pollution.

Role of Scientist and Governmental Policies

As far as exposure to air pollutants, studies should be conducted on the exposure assessment to assess on how much the children are exposed, in which the risk can be estimated and characterized. To assist scientists in assessing risks specifically to children, EPA has developed A Framework for Assessing Health Risk of Environmental Exposures to Children (EPA 2006a). The purpose of this document is to provide an overarching framework for a more complete assessment of children's exposure to environmental agents and the resulting potential health risks within the U.S. Environmental Protection Agency's (EPA's) risk assessment paradigm. This framework examines the impact of potential exposures during developmental life stages and subsequent life stage, while emphasizing the iterative nature of the analysis phase with a multidisciplinary team. In addition to outlining the risk assessment process from a life stage perspective, the document points out published sources for more detailed information. In addition, another document that came along with specific guidance to risk assessors includes guidance on selecting age groups for monitoring and assessing childhood exposures to environmental contaminants and supplemental guidance for assessing susceptibility from early-life exposure to carcinogens.

Environmental exposure is a comprehensive evaluation of the potential for vulnerability of various life stages. In contrast, assessments that use only available chemical-specific data, are often limited with data from adults, do not necessarily account for other life stages. The approach outlined here encourages evaluation of the potential for toxicity during all developmental life stages, based on what is known about critical windows of development for different organ systems and differences in anatomy, physiology, and behaviour that can impact external exposure and internal dose

metrics. In developing an assessment, the lack of data for certain life stages is not meant to imply susceptibility and/or greater uncertainty in the assessment of risk from childhood exposure.

Risk assessment has been a cornerstone of risk based decision making frameworks for many government and multilateral organizations. In the Malaysian context, more research should be conducted to obtain the baseline data so that the exposure information on children can be used in the framework for assessing the children's health risk. It can be useful and reliable when the scientific information base is strong.

CONCLUSION

Air pollutants have health implications on children especially in Malaysia, as they are vulnerable due to the difference in the physiological development. Children are at higher risk with the threat of climate change occurring now and in the future. Health risk in children can be assessed using the health risk assessment tool, however, it has limitations. Future direction would emphasize the role of parents, teachers, the related ministries as well as the government in their policies to protect children's health. Scientist are also urged to do more research to obtain basic data to be used in the US EPA's Framework for Assessing Health Risk of Environmental Exposures to Children.This framework examines the impact of potential exposures to children during their developmental life stages and subsequent life stage.

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BIOGRAPHY

Professor Dr. Zailina Hashim was born in Sungkai, Perak. She went to Jalan Pasir Puteh National Primary School, Ipoh (1962-1966), Sekolah Tun Fatimah (1967), Methodist Girls Secondary School, Ipoh (1968-1971 and Tunku Kurshiah College, Seremban (1972-1973). Dr. Zailina was awarded a scholarship by Majlis Amanah Rakyat (MARA) to study in the USA. She obtained her B.A in Chemistry and Environmental Studies from Macalester College, St Paul, Minnesota in 1978. In early 1981, she obtained her MSc. in Public Health from the University of Minnesota, Minneapolis, USA, and in 1994, she obtained her PhD in Environmental Health Science from the University of Michigan, Ann Arbor, USA under the sponsorship of the Department of Public Service. She had the opportunity to work under Professor Khalil H. Mancy for her dissertation entitled " Assessment of Atmospheric Lead Exposure on Children in Klang Valley, Malaysia".

In 1983, Prof. Zailina was appointed by UPM as a lecturer in the Faculty of Human Ecology and in 1998 as Associate Professor in the Faculty of Medicine and Health Sciences and later in 2007 was promoted to be the Professor of Environmental Health. She was formerly one of the members in the AdHoc Working Committee for the formation of Faculty of Biomedicine and Health Sciences in 1996. As an academician, Prof. Zailina carried out teaching, research and consultation in the field of Environmental Health. She teaches the undergraduate programme BSc (Environmental and Occupational Health) in Environmental and Occupational Health Risk Assessment, Management and Safety and Health Research Methods. She supervises undergraduate students in final year project, graduate students in MSc and PhD in the Environmental and Occupational Health discipline. So far, she has supervised over 15 MSc and PhD students and over 50 undergraduate students.

In 1998, Prof. Zailina started off BSc (Environmental and Occupational Health) in the faculty which the first of its kind in the country. The Accreditation of the BSc(EOH) by the Chartered Institute of Environmental Health, Chadwick, London, UK was obtained in February 2010 in which she served as the Chairman of the Committee.

As a researcher in the field, she has completed as many as 20 research and has published more than 100 articles in local and international journals, proceedings and books. She has been invited to speak in conferences and has presented more than 88 papers at local as well as international conferences. She received travel grants from the Commonwealth Science Council in 1985, International Society of Environmental Epidemiology in 2000, 2004 and 2007, Society of Risk Analysis in 2005 and Asia Pacific Rim Universities World Institute (APRU-WI) in 2011.

Prof. Zailina's research have been on the air pollution as well as atmospheric lead exposure and health assessment on school children since the nineties when the use of leaded gasoline was being phased out in the country. She found that the blood lead levels among children decreased, however, even though the levels were lower than the permissible standard of 10ug/dL, the intelligent quotient (IQ) of these children were affected. Therefore, lead exposure has no safe threshold level. Prof. Zailina is also working with researchers from the University of Uppsala, Sweden, and University of Pittsburgh, Philadelphia, on the indoor air quality such the gaseous pollutants, and microbes in schools. The research findings have been published in high impact journals. She had also been awarded silver medals for her researches in the previous annual PRPI exhibition in UPM.

Prof. Zailina serves as consultant for government as well as private industries. She is a Panel Reviewer for public health impact in the Environmental Impact Assessment (EIA) reports. She is the

co-author of the Guidance Document for Health Impact Assessment in EIA prepared for the Department of Environment and carry out training courses on the topic. She is also the Panel Reviewer with the Malaysian Quality Agency for the accreditation of Diploma and Degree programme in Environmental Health in private university colleges. So far, she has reviewed more than 10 programmes. Dr. Zailina is also an External Examiner to various Environmental Health programmes in UiTM, Consist College and UniKL. Besides these she is also a referee for various publishers such as UiTM Publisher and Dewan Bahasa dan Pustaka. She also serves as an editor to Journal of Indoor Build Environment and MASAUM Journal of Medicine and Medical Sciences and a referee to various local and international journals.

Prof. Zailina specialises in Chemical Management and Health Risk Assessment, as she prepared training module in chemical management for the DANIDA and EiMAS, Department of Environment, Malaysia and has participated in Globally Harmonized System training. She also trains Department of Environment officers on environmental health impact assessment (EHIA).

Prof Zailina is also involved in non-governmental organizations such as MAKNA and KanWork. Her involvement as a cancer survivor to educate and share experiences with cancer patients and other interested parties. At the same time, she also conducts research on the occupational psychological distress experienced by working cancer survivors and has written a few chapters and books on them.

Administratively, Prof. Zailina is the Head of Environmental and Occupational Health Department, FMHS, a member of UPM Technical Committee for Health and Safety, a member of UPM Scholarship and Study Leave Committee, a member of UPM Grant Evaluation for Medical and Health Cluster and a member of FMHS

Graduate Committee. She is the Chairman of FMHS Information and Technology Committee.

As an academician, she was awarded the Excellent Service Award in 1997, 1998, 2005 and 2010.

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Thanks to all my students in BSc Environmental and Occupational Health, my former graduate students Dr. Norazura Ismail, Dr. Majid Aghasi, Chua Swee Kim, Junidah Raib, Nurrul Izzah Abd Samad, Rumaizah Ruslan, Junaidah Zakaria, Yong Heng Weay and Nur Husna Hussin for their contribution to the published paper. I wish to say "your success is my success and my joy".

Thank you to my PhD supervisor at the University of Michigan, Prof. Khalil H. Mancy, for his guidance in research when I was his student, to my other international research collaborators, Prof. Dr. Bernard Goldstein from the University of Pittsburgh, Assoc. Prof. Dr. Dan Norback and Prof. Dr. Gunella Weislander from the University of Uppsala, Sweden.

Heartfelt appreciation for the love, encouragement and contribution from my husband Prof. Dr. Jamal Hisham Hashim who is not only my life partner but also my professional and research collaborator. The love and endurance of my children Hasliza and Muhammad Ariff.

Coming from a family of 10 siblings, I appreciate the love and support from my mother, Zaini Md Yunan, my late father, Hashim Gera, my brothers, sisters and in-laws; Khadijah, Norhayati,

Salehuddin, Baharuddin, Ahmad Tajuddin, Zainal Ariffin, Mazlan and Fauziah.

My utmost gratitude to my dear sister Fatimah who donated her stem cells so that I can be here today to deliver my inaugural lecture. Not forgetting Prof. Dr. Yip Cheng Har from UMMC and Prof. Dr. Cheung from UKMMC for their excellent and dedicated service when I was under their care.

My research and publication would not be possible without the financial support from MOSTI for the IRPA grant, the Ministry of Health, WHO and UPM for the RUGS and Short Term Grants.

Most of all, thank you to Allah Al-Mighty for the knowledge granted and blessings on all my professional work.

LIST OF INAUGURAL LECTURES

1. Prof. Dr. Sulaiman M. Yassin
   The Challenge to Communication Research in Extension
   22 July 1989

2. Prof. Ir. Abang Abdullah Abang Ali
   Indigenous Materials and Technology for Low Cost Housing
   30 August 1990

3. Prof. Dr. Abdul Rahman Abdul Razak
   Plant Parasitic Nematodes, Lesser Known Pests of Agricultural Crops
   30 January 1993

4. Prof. Dr. Mohamed Suleiman
   Numerical Solution of Ordinary Differential Equations: A Historical Perspective
   11 December 1993

5. Prof. Dr. Mohd. Ariff Hussein
   Changing Roles of Agricultural Economics
   5 March 1994

6. Prof. Dr. Mohd. Ismail Ahmad
   Marketing Management: Prospects and Challenges for Agriculture
   6 April 1994

7. Prof. Dr. Mohamed Mahyuddin Mohd. Dahan
   The Changing Demand for Livestock Products
   20 April 1994

8. Prof. Dr. Ruth Kiew
   Plant Taxonomy, Biodiversity and Conservation
   11 May 1994

9. Prof. Ir. Dr. Mohd. Zohadie Bardaie
   Engineering Technological Developments Propelling Agriculture into the 21st Century
   28 May 1994

10. Prof. Dr. Shamsuddin Jusop
    Rock, Mineral and Soil
    18 June 1994

11. Prof. Dr. Abdul Salam Abdullah
Natural Toxicants Affecting Animal Health and Production
29 June 1994

12. Prof. Dr. Mohd. Yusof Hussein
Pest Control: A Challenge in Applied Ecology
9 July 1994

13. Prof. Dr. Kapt. Mohd. Ibrahim Haji Mohamed
Managing Challenges in Fisheries Development through Science and Technology
23 July 1994

14. Prof. Dr. Hj. Amat Juhari Moain
Sejarah Keagungan Bahasa Melayu
6 Ogos 1994

15. Prof. Dr. Law Ah Theem
Oil Pollution in the Malaysian Seas
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16. Prof. Dr. Md. Nordin Hj. Lajis
Fine Chemicals from Biological Resources: The Wealth from Nature
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17. Prof. Dr. Sheikh Omar Abdul Rahman
Health, Disease and Death in Creatures Great and Small
25 February 1995

18. Prof. Dr. Mohamed Shariff Mohamed Din
Fish Health: An Odyssey through the Asia - Pacific Region
25 March 1995

19. Prof. Dr. Tengku Azmi Tengku Ibrahim
Chromosome Distribution and Production Performance of Water Buffaloes
6 May 1995

20. Prof. Dr. Abdul Hamid Mahmood
Bahasa Melayu sebagai Bahasa Ilmu- Cabaran dan Harapan
10 Jun 1995

21. Prof. Dr. Rahim Md. Sail
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22. Prof. Dr. Nik Muhammad Nik Abd. Majid
The Diminishing Tropical Rain Forest: Causes, Symptoms and Cure
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23. Prof. Dr. Ang Kok Jee
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24. Prof. Dr. Sharifuddin Haji Abdul Hamid
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Fish Processing and Preservation: Recent Advances and Future Directions
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27. Prof. Dr. Mohamed Ismail Abdul Karim
Microbial Fermentation and Utilization of Agricultural Bioresources and Wastes in Malaysia
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28. Prof. Dr. Wan Sulaiman Wan Harun
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Sustained Growth and Sustainable Development: Is there a Trade-Off 1 or Malaysia
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Sharecropping in Perfectly Competitive Markets: A Contradiction in Terms
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