

## The Haze and Health: A Blog About the Fog

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The haze is an atmospheric phenomenon where pollutant particles accumulate in the air and obscure the normal clarity of the sky. In Singapore, this usually happens when the prevailing southwest monsoon winds blow the smoke from regional forest fires to Singapore. This phenomenon is not new and has been occurring regularly since 1994. In 1997, it featured on the front page of the Straits Times and news was made again in early October this year when the pollutants standard index or PSI hit 150 (>100 being labelled as unhealthy) for a 24-hour period, this being the worst since 1997 when the index reached 138.

Singapore's blue skies begin to clear up again with the start of the inter-monsoon rains in late October, bringing PSI readings to lower levels again, before these readings fade into oblivion again, at least until next year. The PSI actually measures the main gaseous (NO<sub>2</sub>, CO, SO<sub>2</sub> and O<sub>3</sub>) and particulate matter pollutants in our environment. Of these 5 types of pollutants that are measured, the reading of the predominant pollutant present is the one reported. During the haze, the predominant pollutant is PM<sub>10</sub> or airborne particulate matter less than 10 microns in diameter, which is roughly about a sixth of the diameter of human hair.

Though not without precedence, the infamous London Fog of 1952 has clearly demonstrated that air pollution can kill, with large short-term and long-term increases in mortality and cardiopulmonary disease. The US and European time series have consistently shown short-term associations between temporal day-to-day variations in PM<sub>10</sub> exposure and rate of death from all causes and cardiopulmonary illnesses.<sup>1,2</sup> There has indeed been a heightened interest in the effects of air pollution after two prospective cohort studies suggested that exposure to PM<sub>10</sub> pollution is associated with increased rates of mortality. The landmark study by Dockery et al<sup>3</sup> followed 8111 subjects over a 15-year period in 6 US cities, while the American Cancer society followed 522,138 adults from 1982 to 1989.<sup>4</sup> When follow-up time was doubled to more than 16 years for the American Cancer Society study, Pope et al<sup>5</sup> concluded that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor not only for cardiopulmonary but

also lung cancer mortality. The European confirmation of the association between long-term air pollution and cardiopulmonary mortality comes from Hoek et al's Dutch cohort of 4492 adults followed over 8 years and a larger French study of 14,284 adults followed over 25 years that was recently published.<sup>6,7</sup>

The more susceptible are the elderly and children. In a US study of children aged 10 to 18 years, Gauderman et al<sup>8</sup> reported that increased exposure to air pollution had chronic adverse effects on lung development leading to clinically significant deficits in attained FEV<sub>1</sub> as children reach adulthood. Reduced lung function in later life has been described as second only to cigarette smoking as a predictor of subsequent all cause mortality. It is as important as cholesterol in predicting mortality from ischemic heart disease.<sup>9</sup> There is also a growing body of evidence linking increased PM<sub>10</sub> exposure with increased risks of infant deaths and preterm births.<sup>10,11</sup>

The American Thoracic society has listed the adverse respiratory health effects of the haze. This includes increased incidence of cancer, acute upper and lower respiratory tract infections, exacerbations of disease in chronic cardiopulmonary illness, reduction in lung function, increase in respiratory symptoms such as wheezing, chest tightness, cough and phlegm production, and irritation of the eyes, nose and throat.<sup>12</sup> In the haze of 1997, Singapore saw a 30% increase in outpatient attendances and also increases in accident and emergency attendances for haze related conditions.<sup>13</sup> What was striking, however, was the supplementary findings from scanning electron microscopy that 94% of the particles in the haze were below 2.5 microns in diameter (PM<sub>2.5</sub>).

Does size matter? The US Environmental Protection Agency (EPA) adopted new health standards in 1997 for ozone and particulates after a scientific review by EPA and the independent Clean Air Scientific Advisory Council determined that the existing standards were not adequately protective of public health. The previous standard regulated PM<sub>10</sub> while the new standard now regulates PM<sub>2.5</sub>. Recently, there has also been interest in PM<sub>0.1</sub> (ultrafine particles). There are 2 reasons for this. Firstly, the finer the particles, the larger their total surface area and this gives them a

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greater potential to ferry toxic compounds. Secondly, finer particles are able to penetrate deeper into the lungs. While both PM<sub>10</sub> and PM<sub>2.5</sub> can penetrate the airways into the alveoli, ultrafine particles have also been shown to also pass into the systemic circulation and cause extrapulmonary toxicity.

Thus, respirable particulate matter is being increasingly recognised as an important and independent risk factor not only for respiratory diseases and lung cancer, but also for atherosclerosis, ischaemic heart disease and stroke. PM<sub>10</sub> appears to act independently of the well known associations such as smoking, hypertension, hyperlipidaemia and diabetes, and besides the development of atherosclerosis, exacerbations of cardiovascular disease can also occur such as increased risk of myocardial infarctions within a few hours after exposure and incidence of arrhythmias. The large body of epidemiological and experimental studies for this has been summarised in a recent review.<sup>14</sup> These studies are defining an emerging field that has been termed environmental cardiology.

How do inhaled fine particles induce vascular disease? This remains a subject of ongoing hypothesis, one of which is that the provoked alveolar inflammation causes a systemic inflammatory response which is associated with a procoagulant state. This hypothesis is not unlike that of the systemic inflammatory response syndrome or SIRS in critically ill patients with sepsis. Sepsis is defined as SIRS accompanying infection and patients with severe sepsis and shock also have significant coagulation abnormalities, therefore the rationale for using recombinant activated protein C in severe sepsis and septic shock. While septic shock is associated with the severe end of the spectrum of the systemic inflammatory response, the effects of exposure to particulate air pollution, though seemingly indolent in comparison, has the potential to cause significant systemic adverse effects as well.

To sum it up, the health effects of the haze range from inconsequential and irritable to significant and life threatening. When PSI readings are high and the skies are hazy, the acute effects may be easily recognised, but as PSI readings drop to lower levels, the significant long-term effects of particulate pollution are easily forgotten. We are most at risk when the PSI readings exceed the healthy range for prolonged periods and besides duration, size also does matter. On a bad day, Hong Kong's particulate pollution has been equated to smoking 8 cigarettes a day while Danish papers claim that 1000 Danes die from passive

smoking per year. In Singapore, smoking bans may be instituted in public places to curb inadvertent exposure of nonsmokers to second-hand smoke, but the haze and its health effects respects no bans or boundaries, land or sea. So, an awareness that our neighbour's smoke does not just get in our eyes is crucial and precedes action to be taken at all levels for this recurrent crisis.

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