

Metropolis in Decay - Air Pollution

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Air pollution in Metro Manila has turned from bad to worse. The metropolis has now acquired a canopy of pollution, a dark haze resembling the clouds of an approaching storm. The air, especially in traffic congested areas, smells, and contains particles and gases that make breathing difficult. In fact, one feels drowsy or nauseous when breathing this air for some time.

Can we remain indifferent to this situation? Are we not concerned with the ill effects of the pollutants in the air we breathe? Should we not take positive measures to arrest and reverse the trend in the destruction of the air for survival - ours, our children's and the coming generation's?

Most of the pollutants in the air are gases and particulates from combustion processes, by-products and wastes from the physico-chemical processes of industrial and manufacturing plants, leakages from storage tanks and pipelines of industrial complexes, and products of natural decomposition processes and other human activities.

SOURCES OF AIR POLLUTION

Motor vehicles (jeeps, buses, trucks, cars, and heavy construction equipment) contribute about 70% of the pollutants in air. These consist of the oxides of nitrogen, carbon and sulfur, hydrocarbons, halogenated gaseous lead compounds, lead particulates, and soot.

Vehicle registration data for Met-

ro Manila for the last three years show a trend of about 16,000 additional units per year (Table 1). With the relaxation in the government's policy on the importation of used cars, which may include those that do not pass the stringent environmental pollution standards of the exporting countries, an increase in the number of vehicles in Metro Manila is imminent. The

Table 1. Vehicle registration in Metro Manila, 1985-1987.

TYPE	1985	1986	1987
Gasoline	340,359	304,898	359,469
Diesel	117,159	169,321	130,960
Total Units	457,518	474,219	490,429

maintenance of old vehicles is a problem, considering the cost and availability of spare parts. Non-uniform or erratic changes in driving speed also increases the amount of emissions from vehicles. This is compounded by poor traffic management and the continued use of leaded gasoline. With all these factors in consideration, air pollution in the metropolis could reach a disastrous level.

Power or electricity generating plants rank second as pollution sources. These include plants of government and private agencies, like the National Power Corporation, of commercial establishments, and of industri-

al and manufacturing plants, which burn bunker oil, diesel, coal, and other fossil fuels. These plants generate the same type of pollutants as motor vehicles although composition may vary depending on the type and source of the fuel and the operating conditions.

Cement plants, distilleries, chemical plants, industrial complexes, incinerators, etc., also add pollutants to the air. In 1983, of the 4,059 firms inspected by NEPC, about 41% were found to be air pollutive firms. There are 1,733 of these firms in Metro Manila. It is said that 800 manufacturing and industrial plants discharge about 100 tons of air pollutants daily (1).

EFFECTS OF POLLUTANTS FROM VEHICLE EMISSIONS

Carbon dioxide (CO₂). Normally, carbon dioxide poses no danger to health. It permits the passage of ultraviolet (UV) and visible light from the sun to the earth's surface but absorbs infrared light, preventing it from passing through. During the past century, the average concentration of CO₂ has risen, resulting in what is called the Greenhouse Effect. The temperature of the earth has risen. A possible consequence of this global warming is the melting of the polar ice caps, which can raise the level of the oceans several feet high.

Carbon monoxide (CO). Carbon monoxide has been shown to be detrimental to higher plant life because it inhibits the action of nitrogen-fixing bacteria. In humans and other animals, CO interferes with the transport of oxygen by forming a coordination complex with hemoglobin in the red blood cells. This complex is 200 times more stable than the oxygen-hemoglobin complex, therefore depriving the tissues and various organs, especially the heart and the brain, of the much-needed oxygen. A complicating fact is that cigarette smoking can raise the level

of carbon monoxide in the lungs to between 400 and 500 ppm (2,3).

Nitrogen oxides (NO_x). Prolonged exposure to nitrogen oxides have been shown to be detrimental to both plants and animals. The direct effects of atmospheric NO₂ (4) include acute respiratory disease in adults; acute bronchitis in infants and children; structural changes in lung collagen of rabbits; morphological changes in lung mast cells, characterized by degranulation in rats, pneumonitis and alveolar distention in mice; tissue changes in lungs, heart, liver, and kidneys of monkeys; and leaf abscission, chlorosis, and decreased yield in plants.

In addition to direct effects, nitrogen oxides have been found to be initiators of photochemical smog.

Atmospheric NO₂ undergoes reaction with solar radiation to produce reactive oxygen, which initiates a number of important reactions with hydrocarbons and other compounds and radicals present in the air. The hydrocarbons form aldehydes, ketones, and free radicals, some of which speed up further production of NO₂ and NO. Ozone, O₃, also forms radicals with hydrocarbons, which may also react with O₂ to form peroxide radicals. The aldehydes, ketones, and acids formed, the structures of which depend on the parent hydrocarbons, eventually condense to form aerosols which limit visibility (smog) and disturb atmospheric conditions, resulting in temperature inversion.

Other compounds which may be present in photochemical smog are the eye irritants (lachrymators) and the extremely toxic oxidants. Examples are the peroxyacetyl nitrates, the related peroxybenzoylnitrates produced by the reaction of NO₂ with peroxide radicals, the carcinogenic and mutagenic polychlorinated hydrocarbons, and the polynuclear aromatic compounds produced

from the combustion of fossil fuels.

Oxides of sulfur (SO_2 and SO_3).

These oxides react with moisture to form acids which deposit in the respiratory membranes, therefore irritating them. However, the strong, suffocating odor of these oxides serve as a warning so that direct injury from prolonged exposure is limited. Even slight exposure to sulfur dioxide, though, may cause complications to those already with respiratory disease.

Accumulation of acids of sulfur and nitrogen in the air may aggravate the problem of acid rain which has caused damage to fish in about 2,500 lakes in Sweden, about 1,750 lakes in Norway and nearly 20% of the lakes in Canada (5).

According to the World Health Organization (WHO), Manila's average SO_2 concentration during 1973-1980 was $73 \mu\text{g}/\text{m}^3$, which exceeded that of New York, Los Angeles, and Chicago, where SO_2 concentrations were 59, 35, and $34 \mu\text{g}/\text{m}^3$, respectively. It was comparable to SO_2 levels in Frankfurt and London, which were 79 and $78 \mu\text{g}/\text{m}^3$, respectively (1).

Lead compounds and lead particulates. Regular and premium gasoline contain 2.5 to 4.0 grams of tetraalkyl lead per gallon of gasoline. The use of this additive in developed countries has been regulated and it is in fact being gradually phased out. In the Philippines, phasing out this additive was supposed to start in 1985, to be completed in 1989. This was not implemented.

A bothering fact about the use of tetraalkyl lead is that dibromoethane and dichloroethane are also added to the gasoline to prevent the deposition of lead and lead oxides in the engine. During combustion, these compounds are vaporized and are emitted into the atmosphere as finely divided particles of halogenated lead and hydrocarbon compounds. More than 98% of the total lead in the atmosphere comes from

the combustion of leaded gasoline.

The lead particles are fine enough (less than $2 \mu\text{m}$ in size) to be easily retained by the lungs of humans, causing respiratory problems.

Three documented toxic effects of lead are: gastrointestinal cramps, (lead colic), central and peripheral nervous system disorders (lead encephalitis), and anemia. Kidney disease, hypertension, vascular disease, and lung cancer, have also been suggested although there has not been any definite proof of their association with lead.

Lead is a general metabolic poison and an enzyme inhibitor, like the heavy metals mercury and cadmium. Young children are particularly affected by lead and can suffer mental retardation, and semipermanent or permanent brain damage. An insidious effect of lead is its ability to replace calcium in bones and remain there to form a semi-permanent reservoir for long-term release even well after the initial absorption.

A study conducted by a medical team, who examined children aged 4 months to 14 years living in depressed areas in Metro Manila, found substantial lead levels in the children's blood. The team found average levels of about $22.83 \mu\text{g}/\text{dL}$ with 7.76% of those sampled having lead levels of $30 \mu\text{g}/\text{dL}$. Since the U.S. Center for Disease Control (CDC) had established $30 \mu\text{g}/\text{dL}$ as a measure for "elevated" blood lead levels, the result of this study is quite disturbing. A significant correlation was also found between increased blood lead levels and the proximity of the household to dense traffic (6).

Particulates. The particulates in air are largely smoke, soot, and lead particles. These are in the size range of $0.1-10 \mu\text{m}$. Because of their large surface area, they absorb the gaseous and liquid pollutants and act as catalysts in photochemical reactions (7,8).

These particles, their diameters being comparable to the wavelength of visible radiation, can cause interfe-

rence phenomena which reduce visibility as well as the amount of solar radiation reaching the ground. These particles may also serve as nuclei for the formation of water droplets which are too small to precipitate, thereby increasing cloud cover only, without any rainfall (9).

Particulates also impair the circulation of CO_2 and O_2 through plant leaf stomata. In humans and other animals, the respiratory tract is the site of most damage. Particles smaller than $0.1 \mu\text{m}$ are deposited in the lungs, causing respiratory problems.

Data from WHO and the United Nations Environment Program (UNEP) (1) revealed that the average concentration of suspended particulate matter in Manila during 1973-1980 was $85 \mu\text{g}/\text{m}^3$, exceeding that of Tokyo and New York City, with 61 and $65 \mu\text{g}/\text{m}^3$, respectively.

Polynuclear aromatic and polychlorinated hydrocarbons. A recent article by J. Sigsby et al., (10) indicated that even the regular and premium non-leaded gasolines emitted polynuclear aromatic hydrocarbons (PAH). According to Takashi Handa et al. (11), carcinogenic and mutagenic PAH are readily converted to their nitro derivatives which are also direct-acting mutagens, by exposure to the nitrogen oxides. Polycyclic quinones may also be formed from the appropriate PAH by oxidation under the high temperature condition in an engine. Muller and Buser (12) who studied the formation of halogenated compounds from the combustion of leaded gasoline, found that these compounds were mostly ortho-substituted phenols and therefore potential precursors of the halogenated dibenzo-p-dioxins which are acutely carcinogenic and mutagenic and hardly biodegradable.

EMISSIONS FROM INCINERATORS

In the Manila Bulletin issue dated

September 5, 1988, an article carried the concern of various environmental groups over the plan of Manila Mayor Gemiliano Lopez, Jr., to build an incinerator plant in the city and transfer the huge piles of garbage from the "Smokey Mountain" in Tondo to the Engineering Island in Manila Bay. This is a valid concern because of the hazards which incinerator emissions pose.

Incinerators are thermal destruction devices used in waste disposal which operate by exposing the waste to temperatures of about 900°C or higher in an oxidizing environment. The waste is actually used as fuel for thermal oxidation and starved-air or pyrolytic reactions, done in kilns, boilers, or furnaces.

Studies have shown that incinerator-type combustion processes also produce PAH. The presence of these compounds in the stack gases and in the ash residues of municipal incinerators has been reported (13,14,15).

Ian W. Davies et al. (16) studied the formation of PAH in municipal incinerators burning 9.14 tons refuse per hour with furnace temperature maintained between $800-900^\circ\text{C}$ and burning with 75% excess oxygen. In this study, the residue was dropped from the grate into a tank where it was quenched, freed of magnetic materials, and discharged. The gases leaving the furnace entered in an upflow into the water spray towers where they were cooled to $250-300^\circ\text{C}$ and larger particles of fly ash removed. Induced draft fans drew the gases from the spray towers through an electrostatic precipitator prior to discharging them through a 55 m-high concrete chimney. The results showed that gases leaving the furnace contained PAH both as vapor and as fly ash particulates. Solid residues made up of combinations of quenched ash residues and fly ash collected from the spray tower's electrostatic precipitator, also contained PAH, as did the washwater from the incinerator. The level of PAH in the emissions varied with combustion temperature and with refuse composition during normal oper-

ations. The water used for quenching substantially increased in PAH content during use.

Other studies of municipal incinerators showed that complex mixtures of organic compounds were present in fly ash samples at concentration levels of 1-30 $\mu\text{g/g}$ (17-20). These mixtures were resolved through solvent extraction methods and were found to contain n-alkanes, polychlorinated benzenes (PCBs), PAHs, polychlorinated phenols (PCPs), and many others. Also present in the extracts were the acutely toxic, teratogenic, and mutagenic polychlorinated dibenzofurans (PCDFs) and the polychlorinated dibenzo - p - dioxins (PCDDs) at concentration levels of 1-10 ng/g of fly ash. Since some of the isomers of PCDDs are also acutely toxic and carcinogenic (21-23), the formation and environmental fate of PCDDs have been the center of several recent studies (24-38).

The precursors and the exact mechanism of formation of PCDDs and similar compounds during the combustion process in incinerators are unknown. Laboratory studies have shown that thermal processes, including pyrolysis and the burning of precursors such as PCBs, PCPs, and other chlorinated biphenyls, produce certain PCDDs and PCDFs (39-41), reinforcing theories that these compounds are products of combustion. Studies also showed that compounds which are absorbed in the fly ash may also undergo reactions with gases during their emission into the atmosphere. An example would be the PAHs which are rapidly oxidized in the fly ash even in the absence of light (42), and which also undergo photochemically-induced gas-particulate reactions (43-44).

Results of studies cited above and other similar studies with simple organic compounds (45) suggest that even incinerators with a destruction removal efficiency (DRE) of 99.9999% would still be a health hazard because of the toxic and carcinogenic compounds that they produce.

Thus, while incineration appears a

promising means to dispose of wastes primarily because of the remarkable reduction in waste volume (up to 85%), its potential adverse effects on man's health and the environment should be a deterrent in the putting up of incinerators in Metro Manila.

The air is the Almighty's gift to all of us, rich and poor alike. But with the unabated addition of pollutants to the air, we may have to pay dearly for the air we breathe in the days to come. ■

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