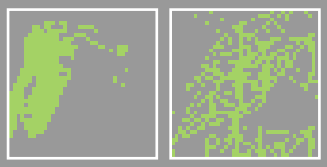

Ethanol and Health

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This study shows that even a partial substitution of petroleum derivatives by ethanol in the vehicle fleet in the São Paulo metropolitan region would save hundreds of lives and prevent thousands of hospitalizations, saving public coffers hundreds of millions of dollars.

The impacts of air pollution on health are well known in the scientific community. Fuel replacement is equivalent to a reduction in ozone formation (ethanol instead of gasoline) and particulate emissions (ethanol instead of diesel). Furthermore, the use of ethanol reduces greenhouse gas emission and helps combat global warming.

Over 12,000 hospitalizations and 875 deaths would be avoided per year in a scenario of total oil and diesel replacement by ethanol in Greater São Paulo. During this period, public coffers would save nearly US$190 million.

A more realistic goal – using ethanol in the bus fleet – would result in the reduction of 1,350 hospitalizations per year due to ailments caused by diesel-generated pollution. In this way more than US$3.8 million would be saved each year. Also, 220 deaths would be avoided per year, equivalent to half the number of deaths from tuberculosis in the region in 2007.

Pollutants considered in the analysis were fine particulate matter and ozone. These pollutants were chosen because they exceed the recommended limits of the World Health Organization and are considered the main challenges in the area of air pollution in Brazil.

Greater São Paulo was chosen for the study because of the availability of good quality observational data on health and the environmental. Since air pollution is recognized as a problem that affects other densely-populated regions, the results of this study can be extrapolated to other areas with similar characteristics.

Another aspect that should be emphasized is that the results are underestimated, because health affects are only measured in terms of hospital admissions and mortality. This is due to the availability of official information. However, it is known that these cases represent a minority of adverse health events and do not take into account diseases that do not require hospital care or cause death. In other words, the impact is even more serious than the numbers presented in the simulation.

Presentation of the problem

Mankind is central to prospection and marketing in the oil, gas and renewable fuels industry. The availability of energy that can be stored and "packaged" into fuel tanks or gas drums has opened the way for mechanized mobility. Hydrocarbons contained in petroleum have made possible the development of new products and compounds that have changed the format and efficiency of several items of equipments and the production of new medicines. More importantly, the availability of energy and new compounds has allowed society to incorporate new attitudes and patterns of consumption, creating habits that require ever-increasing energy production. These new habits have become part of daily life and any change to the energy matrix or the way that we generate and consume energy do not seem to be feasible and efficient in the short term. Changes are happening slowly, and are subject not only to environmental but also to economic decisions.

In this scenario, we are now confronted with extremely relevant issues for the energy industry. In our view, the following are the most important points:

a What are the energy alternatives that will reconcile society's increased energy demand with questions of efficiency, price and sustainability? The concentration of oil and gas production in some critical regions of the planet has led to various moments of tension in recent decades, impacting the price of these products in the international market. An expanded group of feasible energy alternatives is the best antidote to these difficulties.

b What are the energy sources that imply least environmental impact, be it at a global scale (minimizing the effects of climate change) or at a regional scale (minimizing adverse effects of fuel production and emissions)? Global warming from the emission of CO_2 and other greenhouse gases is an issue that has moved beyond the technical sphere of academia and industry to impact the daily life of ordinary people. The same can be said for the adverse effects of vehicular emissions, which have been the object of increasingly restrictive political control in order to safeguard human health. As a result of these problems, created through the consumption of fossil fuels by industry and transportation, there is a growing sentiment in society that we should reduce pollution emissions from stationary and mobile sources, which will affect the future market for these fuels. It is important to note that, at the current technological level and for most uses of oil and gas (and their derivatives), no significant reduction in emissions can be achieved just by technological improvement of the industrial process or of motor engineering. It must necessarily consider fuel composition as a determinant factor. A clear example of this is diesel vehicles, where the catalytic converter technology is dependent on the fuel formulation.

c How can we transform the energy generation process into one of the tools that helps to achieve socioeconomic equality between rich and poor nations, and also reduces social and health inequalities within a country? Energy production is a source of wealth. In general, the choice of new energy sources is made in terms of cost-effectiveness, determined from the point of view of the productive process. Activities related to fuel production, distribution and trade produce impacts on human life. **Table 1** presents a summary comparison between the potential risks to human health due to the use of petroleum-based fuels and some biofuels (ethanol and biodiesel).

The analysis in **Table 1** shows that risk to human health is inevitable at every stage of fuel production. What matters is choosing the alternative with the smallest impact. It is also important to implement and develop new practices that ensure processes have maximum sustainability, taking into account environmental, economic and social aspects (for example, the elimination of sugarcane burning prior to the harvest).

The above-mentioned set of situations indicates that mankind and the fuel production industry have forged such close bonds that they have sealed their fate in an implicit pact. The future of mankind depends on increasing the production of clean and sustainable energy sources, while the future of the energy market will depend on its ability to meet the needs of mankind through sources that ensure climate stability, with the least possible damage to human health, and that lead to a future with less socio-economic inequality. In other words, by including sustainability and a lower risk to health and the environment, the price of new fuels will depend, in the near future, on the incorporation of values that go beyond the costs of prospection, production, refining and distribution. The incorporation of questions related to the impact on humans of the new energy alternatives that Brazil has adopted could add new value to these fuels and provide information that may assist the strategic planning of energy markets in the coming decades.

Comparison of potential risks to human health Table 1

	Petroleum	Biofuels
Production	Water and soil contamination from production waste or leaks	Water and soil contamination from pesticides and production waste
	CO_2 emissions (flaring)	
	Fugitive emissions	Straw burning (ethanol)
	Work conditions: onboard work on platforms, contamination from chemicals present at refineries	Work conditions: exhaustion, inhalation of gases and particles after straw burning
Transport and Storage	Leaks (during transport, tanks and pipelines)	Increased solubility of leak plume
	Fugitive emissions	Fugitive emissions
Emissions	Atmospheric pollutants (particles, hydrocarbons, volatile organic compounds precursor of O_3)	Atmospheric pollutants (carbonyl compounds in the case of ethanol, NO_2 and long-chain hydrocarbons such as acrolein in biodiesel)
	Increased greenhouse gas emissions	Variable greenhouse gas emissions balance, depending on the grid

General considerations about the relation between atmospheric pollution and human health

The assessment of the impact of energy sources on health requires the availability of information at a series of interconnected levels, as shown in **Figure 1**.

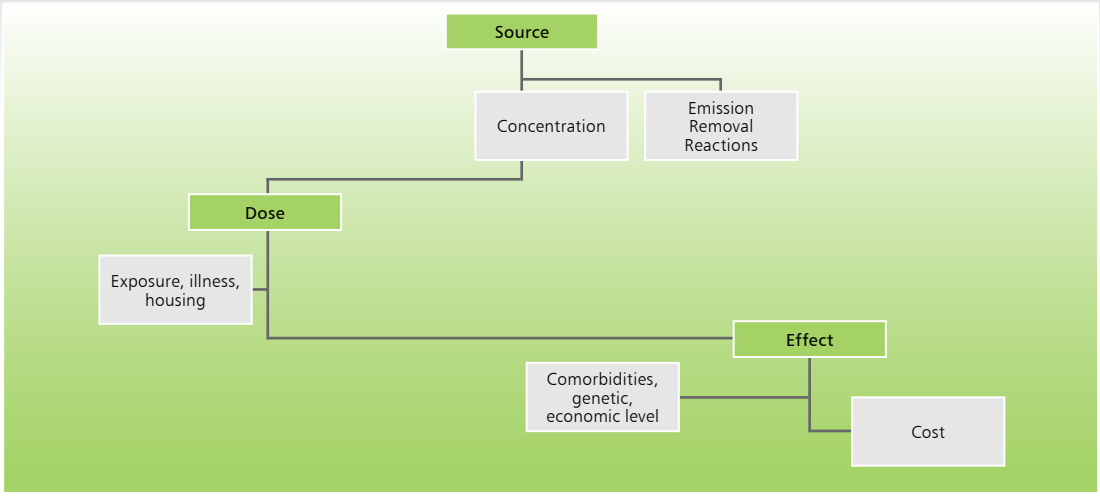
Each element in **Figure 1** represents critical information for determining environmental risk.

One key point is the characterization of the emission source, with its chemical composition and emission rates. In some situations the emission source is known, as in the case of vehicular, industrial or agricultural emissions. However, there are situations in which the emission source is unknown, as in the case of surface water pollution in a major hydrographic basin.

We must also consider various aspects related to dispersion and reactions in the environment. Primary pollutants are those emitted directly by the source – discharge points of liquid effluents, industrial smokestacks, automotive tail pipes, and re-suspension from the soil, for example. Secondary pollutants are produced in the environment from chemical reactions, and have primary pollutants as their precursors. In this group ozone and secondary aerosol particles are significant.

Pollutants with local effects are those with a very short average life and that affect mainly the area surrounding their source. Pollutants with mesoscale effect are those with an average life of hours or days, affecting larger regions because they can be carried by winds, convection or water. Pollutants with regional impacts are those that reach their highest concentrations many miles from their point of formation. Aero-

Figure 1 **Algorithm for evaluation of environmental risk**



Source: Adapted from Kovats and cols, 2005.

sol particles generated by fires in the Amazon affect the Southeast a few days after being formed. This means that pollutants with a regional impact are those that are formed or carried a great distance from their source or from the origin of their precursors. Pollutants with global effects are generally atmospheric in nature, and can be carried long distances in the troposphere or reach the stratosphere via vertical convection. CH_4 (methane) and CO_2 (carbon dioxide) are examples of this type of pollutant, and participate in global temperature change.

The physical characteristics of the source, the type of pollutant emitted and its associated emission rate, reactions that occur in the atmosphere and the phenomena of transportation and removal are all crucial to defining the environmental concentrations of pollutants.

Concentration is not the only factor that determines the dose received by any specific individual or segment of the population. Factors that significantly alter the received dose include the time spent near sources of atmospheric emission, the level of physical activity, comorbidities that alter absorption, metabolism or rate of absorption of environmental pollutants and socio-economic factors that alter housing conditions in a way that allows greater penetration of pollutants inside homes.

On the other hand, the adverse effects on health for a given dose of pollutants will depend on factors related to individual susceptibility. The adverse effect of environmental pollutants will be modulated by factors such as age, nutritional state, socio-economic level, pre-existing diseases and polymorphism of detoxifying genes. The effect of pollutants on health also depends on time of exposure. Depending on the type of pollutant, the dose and the individual characteristics of the subject some adverse effects are manifested acutely (hours or days after exposure), while others are seen only after long periods of exposure (the so-called chronic effects). Increases in mortality associated with situations of excessive accumulation of atmospheric pollutants is a typical example of the acute effects of pollutants. Indoor pollution from cigarette smoke, decreased intelligence from lead exposure, and most of the pollutants that cause cancer are examples of how the magnitude of health damage can be accurately assessed only after prolonged periods of exposure.

It is also important to establish limits for the health effects that will be assessed. Health effects on the population due to exposure to ambient pollutants are varied. Intensities and latency periods are different, and include: behavioral and cognitive effects; pulmonary and systemic inflammation; alterations to airway caliber, vascular tone and heart rate control; reproductive changes; morbidity and mortality from cardiac and respiratory diseases; and increased incidence of cancer; among others. Given the multiplicity of possible outcomes, it is necessary to define adverse health effects in an objective way. Based on this definition, we can then select which events are useful to determine the impact that an environmental change will have on the population that is exposed to it.

Although the concept of adverse or detrimental effects to human health is widely used to define measures of risk assessment or environmental management, a precise definition of the boundaries between a finding that is statistically significant and a change that will in fact result in relevant health effects still requires further definition. A description and analysis of current definitions can be found in **Annex 1**.

The definition of adverse health effects must necessarily be accompanied by a characterization of the most susceptible groups. Increased susceptibility to pollutants depends on individual, housing and socio-economic factors. Among those of an individual nature, the most important are age, associated morbidities, and genetic characteristics. The extremes of the age pyramid have been identified as prime targets for the adverse effects of air pollutants, especially in the segments below five and above 65 years of age. Associated morbidities such as asthma, chronic bronchitis, atherosclerosis, diabetes mellitus, cardiomyopathies and cardiac arrhythmias are among the pathological conditions known to be more susceptible to air pollution effects.

Living conditions affect the dose received and, consequently, the susceptibility to pollutants. In large urban centers, there are areas where the generation and dispersion of pollutants leads to ambient pollution levels that are significantly higher than the urban average. Places that present a greater risk to residents are those adjacent to major traffic corridors, low-lying regions in urban centers, areas with high buildings, and areas subject to constant traffic jams. For example, measurement of inhalable fine particulate matter carried out under the Costa e Silva elevated highway in São Paulo showed levels three times greater than city averages.

The type of construction also affects the degree of pollution penetration inside homes. Older buildings that lack air conditioning tend to have greater air pollution penetration. We must also take into account that contributions from internal sources can significantly deteriorate air quality in homes. Socio-economic conditions also interfere with the susceptibility to air pollutants. In São Paulo, it was shown that, given the same variation of ambient pollution (expressed in terms of inhalable particulate matter, PM10), mortality is higher in neighborhoods with worse socio-economic indicators (Martins et al, 2004).

Factors that determine the greater vulnerability of the underprivileged to air pollutants can be divided into two main categories: events related to health conditions and access to care and medication, and conditions that foster greater exposure to pollutants. In the first category, we know that the less fortunate segment of the population has worse health due to deficient sanitation and nutrition, lack of access to medical services, and lower purchasing power to buy medication when ill. The second category – greater exposure – has been recognized as a relevant factor in the relationship between air pollution and health.

The relationship between social exclusion and greater exposure to pollutants occurs both at continental levels and within individual communities. Dirtier industrial processes, vehicles with obsolete technology, and fuels with higher concentrations of contaminants are found more frequently in developing countries. To a lesser degree, it is common within a given community for the professions that lead to greater exposure to pollutants (street workers, for example) to be performed by the most needy segments of the population. Similarly, it is more common for disadvantaged groups to live in houses beside roads with high traffic, and to use firewood or waste for cooking. We can thus see that the greater vulnerability of poorer social groups to air pollution is determined both by worse basic conditions of health and access to health infrastructure, and also by greater exposure to pollution.

Impacts of sugarcane ethanol production on human health

It is appropriate to emphasize that the analysis of health effects reported in this chapter will be based on the effects of atmospheric emissions, in particular for emitted pollutants and the emission of greenhouse gases. For comparative purposes, the effects observed in the case of ethanol will be compared to those present in the current alternative, i.e., petroleum derivatives.

Fuel production is strongly associated with air emissions that have the potential to interfere with human health. In São Paulo State, the current practice of burning off sugarcane straw in order to manually harvest sugarcane has been associated with increased morbidity caused by respiratory diseases in adults and children, and cardiovascular diseases in adults. Health effects appear to be strongly dependent on the fraction of particulate emissions, and have sufficient magnitude to constitute a significant public health problem for those exposed to them. As a result of these impacts, a protocol was signed in 2007 between the Environmental Secretariat of São Paulo State (SMA), and the Brazilian Sugarcane Industry Association (UNICA), establishing a progressive reduction in plantation burning with a corresponding increase in mechanized harvest. According to the SMA, mechanization accounted for 49.1% of the 2008-2009 harvest. According to the protocol, all areas with slopes of less than 12% will be mechanized by 2014. Vegetation cover in São Paulo will increase as a result of reforestation in areas where the slopes are too steep for mechanized harvest, for example along river banks.

Activities related to petroleum production and refining are harmful to human health. Several epidemiological studies have reported an increase in cases of respiratory diseases, cardiovascular diseases and tumors (leukemia and cancers of the central nervous system) in areas near refineries and petrochemical plants. Recent studies in the Paraíba Valley in São Paulo showed increased rates of change in biomarkers in the vicinity of the oil refinery, along with increased rates of cardiovascular disease and cancer. Organic compounds in the gaseous and particulate phases of emissions from refineries and petrochemical complexes are mutagenic and lend biological plausibility to the toxicological and epidemiological findings outlined above.

The set of information concerning the production process shows that the nature of chemical compounds and the severity of health findings indicate that sugarcane production is significant in terms of atmospheric emissions. This points to the need to reduce or end sugarcane straw burning prior to harvest.

Climate change and public health

Sugarcane ethanol has significant advantages over petroleum-based fuels in terms of greenhouse gas emissions. This question – the health consequences of predicted climate change due to global warming – deserves some special consideration.

Medical literature has been devoting increasing attention to the potential impacts of climate change on human health. Within this scenario, biofuels such as ethanol may also help reduce health impacts due

to global warming, because they are more neutral in terms of greenhouse gas emissions compared to petroleum-derived fuels: the balance between the absorption of CO_2 during plant growth and emissions during production and fuel burning is almost neutral, while the elimination of straw burning, among other advances, further reduces emissions. This document focuses on three aspects of the relationship between health and climate change: food security, water shortages and heat stress.

Food security is one of the most evident problems of global warming. Climate models project that, maintaining the current rate of warming, areas of Brazil such as the semi-arid region of the Northeast may present a process of desertification. Paradoxically, increased levels of atmospheric CO_2 may cause some crops, especially in the South, Southeast and Midwest of the country, to increase in productivity if water is available. If these forecasts come true, there will be an increase in social and economic inequality, with migration from new desert areas and an increase in the poverty belt around major cities. This process will tend to be more intense in regions with a greater proportion of family or small farms, which will be less able to make the necessary adjustments.

The quality and quantity of water available for human consumption are crucial determinants in the relationship between health and illness. Infectious diseases transmitted via water are among the leading causes of morbidity and mortality worldwide. The desertification process in the semi-arid region will exacerbate water shortages in the area. Moreover, climate changes are causing rainfall in the Northeast to occur in greater intensity at the beginning of the rainy season, petering out later in this season. The example of 2009 is a clear indicator of this phenomenon. Systems of storage tanks and ponds were compromised around the flooded areas. This could have led to water shortages if rains had been insufficient, as well as to contamination of reservoirs by human and animal waste. In coastal regions, the rise in sea level points to increased salinity of aquifers, with the consequent reduction in the quantity and quality of water. If this situation persists, it is forecast that global warming will increase morbidity and mortality from waterborne diseases, while forcing the migration of the population from affected regions.

Finally, it is appropriate to discuss thermal stress. Our bodies are maintained in a narrow temperature range around 37° C, independent of the temperature range that the external environment imposes on us. The fine control of body temperature is the result of the body's thermal regulatory centers, and the adaptation of our clothing and dwellings. Each population has a range of thermal comfort, and this varies for different regions. When the external environment presents temperatures outside of this comfort zone, health indicators such as hospital consultations and excess mortality start to rise. A schematic representation, which translates empirically what happens in São Paulo, is shown in **Figure 3**.

Figure 3 shows that the relationship between excess mortality from temperature extremes is not linear, but increases disproportionately with extremes of minimum daily temperature. The range of thermal comfort can be defined as lying between a minimum daily temperature of 10 and 20 degrees centigrade, with excess mortality caused by waves of cold and heat. In the case of São Paulo, the effect of a cold spell is more intense than that observed with heat waves. In a cold city, the opposite occurs, i.e., heat waves have a greater impact on health. Those most affected are people whose mechanisms of adaptation are less ef-

ficient – children (respiratory disease) and the elderly (respiratory diseases in cold spells and cardiovascular diseases in heat waves). Social and economic factors also modify the effects of extreme temperatures. Lower income dwellings offer greater "permeability" to external temperature variations, while the reduced vegetation rates in the poorest regions of the city increase the local daily temperature range and are responsible for the greater impact of thermal extremes among the poorest members of a community.

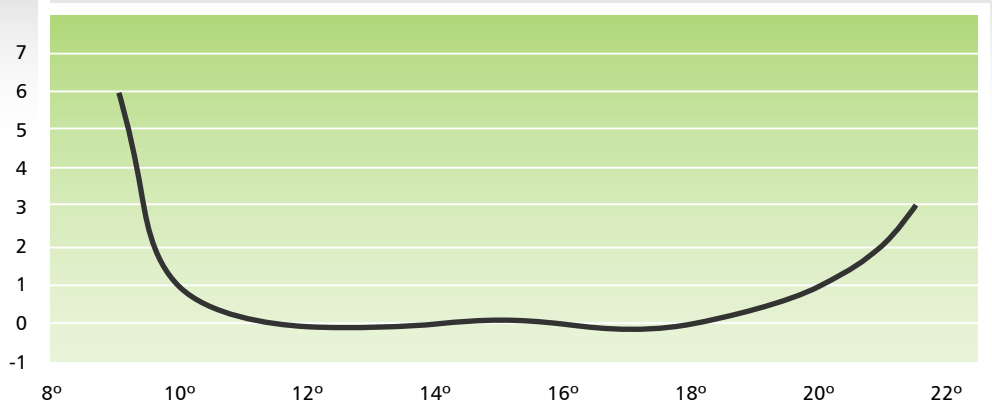
Some other aspects of the relationship between global warming and disease – such as possible increased spread of infectious diseases transmitted by insects, and natural disasters such as floods and landslides caused by extreme rainfall – also significant, but are not addressed at this time due to space limitations.

Impact of fuel ethanol from the perspective of accidents during transportation and storage

Leakage of oil and its derivatives is a major source of environmental accidents, with notable environmental impacts. Hydrocarbons and metals present in petroleum can be consumed by people and produce adverse health effects by contaminating water on the surface or deep underground, or by entering the food chain cycle. The expected effects are reproductive disturbances, changes in bone marrow function (damaging the formation of red blood cells – anemia – and white blood cells – low immunity) and increased risks of developing cancer, especially leukemia and lymphomas. Ethanol's chemical structure and greater capacity to break down in the natural environment implies virtually zero risk of the changes just described. One

Figure 3

Representation of additional deaths attributable to extremes of minimum temperatures in the city of São Paulo *Additional deaths per day / Minimum temperature in centigrade*



downside of ethanol comes with fuel storage at filling stations, given the increased permeability of gasoline in soil when it is mixed with ethanol (McDowell et al, 2003). In other words, the addition of ethanol to gasoline increases the plume dispersion of gasoline in the soil, increasing the risk of surface water contamination in the case of a filling station leak. This situation deserves greater attention in terms of monitoring tank sealing at filling stations in urban areas. And what about the risk of inhalation?

Health effects of emissions by ethanol powered vehicles

We know intuitively that, regardless of the fuel used, automotive emissions contain compounds that have potential effects on human health. Inhalation offers a gateway for these compounds, given that an adult's lungs have an alveolar surface of around 70 square meters, and a barrier of cells with an average thickness of less than one thousandth of a millimeter is interposed between our internal environment (the inside of alveolar capillaries) and the external environment. As the air passes through approximately 30 centimeters of airways, it must be heated to 37° C, reach a relative humidity of around 90% and be filtered of microorganisms and ambient pollutants. The arrival in the alveolar territory of chemical compounds, both in the gaseous state or adhering to soot particles, gives rise to a possible local or systemic inflammatory response as these compounds access the bloodstream. It is therefore imperative that the analysis of health effects of emissions from ethanol-powered vehicles be conducted in comparison to the emission from vehicles powered by gasoline or diesel.

Laboratory studies with rodents in the 1980s showed that emissions from light vehicles running on ethanol were less toxic than those from gasoline-powered engines, in tests for both acute and chronic toxicity. Ethanol emissions produced lower levels of lung inflammation and of mutations. In these studies, the lower toxicity of ethanol emissions was attributed to the type of organic compounds emitted.

In the case of emissions from ethanol-powered engines, the organic compounds are almost entirely ethanol, accounting for 70%, and aldehydes, amounting to 10% (this fraction comprised 85% acetaldehyde and 14% formaldehyde). Gasoline engines on the other hand emit a whole family of volatile compounds and polycyclic aromatic hydrocarbons with high toxic and carcinogenic potential. This approach, while useful as an initial step, does not take into account photochemical processes that can occur in the so-called "real world", i.e. in the atmosphere of big cities. It is extremely important to analyze the formation of secondary pollutants, especially ozone and other compounds of the photochemical oxidants family formed by the interaction of primary pollutants with solar radiation.

An increased level of aldehydes (acetaldehyde and formaldehyde) in the atmosphere is undeniably one of the consequences of using fuel ethanol. On the other hand, formaldehyde is the most characteristic aldehyde in vehicular emissions from the use of petroleum and its byproducts, notably diesel. It is important to look in more detail here at the behavior and toxicity of atmospheric aldehydes, both from the perspective of their direct toxicity, as well as their potential for ozone formation.

Structure and metabolism of aldehydes

Aldehydes are highly reactive organic substances. They contain a carbonyl group (double bond between carbon and oxygen atoms), and have a high affinity with lipids, proteins and DNA (Comeap, 2000)[i]. Aldehydes can be divided into three classes based on their structure and reactivity with organic substrates (Comeap, 2000):

a Simple or saturated aldehydes: the metabolism of these aldehydes happens through the oxidation of their carboxylic acids (via aldehyde dehydrogenase) or by reducing the alcohol dehydrogenase. Links with thiol groups, as well as links with several proteins, including those that make up DNA, also occur and explain the carcinogenic potential of these aldehydes. The aldehydes of interest in this study – formaldehyde and acetaldehyde – are representatives of this category of aldehydes.

b Unsaturated α,β-aldehydes (for example, acrolein): these aldehydes bind to substrates such as glutathione or cysteine, and oxidize after these links. As in the previous case, this class of aldehydes can bind to amino groups of DNA, and may lead to the development of mutations.

c Halogenated or modified aldehydes (benzoaldehyde): the metabolism of these aldehydes depends on the nature of their functional group; some can be oxidized (for example, benzoaldehyde, furfural, malodialdehyde) while others are predominantly conjugated with glutathione, cysteine or serine.

Sources of aldehydes in the external environment of large cities

In outdoor areas of large cities, the different classes of aldehydes described above are produced by vehicle emissions, biomass burning or through photochemical reactions (Miller et al, 2001). In the atmosphere of large urban centers, the relative contribution to aldehydes production of direct emissions and photochemical processes will depend on the rate of emission from anthropogenic sources and climatic conditions.

The emission of aldehydes in urban environments is the result of incomplete oxidation of vehicle fuel, be that gasoline, gasohol, ethanol, natural gas or diesel (Abrantes et al, 2005; Durbin et al, 2007; Kado et al 2005; Martins et al 2008). In the polluted atmosphere of large cities, the main precursors of aldehydes are hydrocarbons, alcohols, ethers and aromatic compounds of anthropogenic origin, subjected to the action of ozone or OH, HO_2 and NO_3 radicals (Andrade et al, 2002).

In the city of São Paulo, carbonyl directly emitted by vehicles predominates in the morning, with greater participation of photochemical processes in the afternoon (Miller et al, 2001). Usually in the morning, the concentration of acetaldehyde is higher than that of formaldehyde; this behavior is reversed in the afternoon after photochemical processes.

Aldehydes as ozone precursors

In addition to their direct toxicity, atmospheric aldehydes can contribute to the formation of ozone, one of the pollutants most associated with adverse effects for human health. The basic aspects of the photochemical reactions related to aldehydes and ozone are of great importance to understanding the consequences of using different fuels, and are presented in Annex 2 (aldehydes as precursors of ozone). Further details can be found in specific literature (Carter, 1994; Saldiva et al, 2005).

Effects of formaldehyde and acetaldehyde on human health

The vast majority of our knowledge about the effects of formaldehyde and acetaldehyde on human health comes from the occupational area or from a context of external environments. This information is of little value when one takes into account the scope of the current study, which has as its main objective the analysis of aldehydes in an ambient context. The detailed survey of the medical literature did not reveal the existence of population-based studies, relating ambient concentrations of formaldehyde and acetaldehyde to indicators of morbidity or mortality.

There are major problems with transposition of data from occupational studies to an ambient context. For a start, there are significant differences in the degree of concentration of aldehydes in the ambient study; this tends to be much greater in the working environment. On the other hand, the susceptibility of exposed populations tends to be different. There is much less frequency in the working environment of those individuals who are most prone to suffer greater adverse effects when exposed to ambient levels of atmospheric pollutants, such as the elderly, children or patients with severe asthma or cardiovascular diseases.

In this scenario, the estimated risk of the adverse effects of formaldehyde and acetaldehyde on indicators of morbidity – in other words, the induction or worsening of disease – presents as a limiting aspect the fact that symptoms or abnormalities observed in humans or experimental animals were observed in ambient concentrations much greater than those found in Brazilian cities.

The U.S. Environmental Protection Agency (EPA) has not defined a reference concentration for chronic inhalation of formaldehyde (Iris, 1990)[ii]. Based on studies with rodents, the EPA has identified cancer risk from exposure to formaldehyde through inhalation, establishing the unit of risk for inhalation at 1.3×10^{-5} per $\mu g/m^3$. This means that lifetime exposure to a concentration of 1 $\mu g/m^3$ leads to an additional 1.3 cancer cases per 100,000 inhabitants. The same situation occurs for acetaldehyde, which does not have a set safety standard for chronic inhalation, except for the risk of developing tumors (Iris, 1991). In the case of acetaldehyde, lifetime exposure to a concentration of 1 $\mu g/m^3$ leads to an excess of 2.2 x 10-6 per 1 $\mu g/m^3$. This means that lifetime exposure to a concentration of 1 $\mu g/m^3$ creates an additional 2.2 cancer cases per one million inhabitants.

Effects of ozone on human health

As noted earlier, aldehydes are important precursors of ozone formation. In this case, unlike the situation for formaldehyde and acetaldehyde, there is a solid mass of population-targeted information relating ambient variations of ozone to adverse health outcomes.

Studies using controlled inhalation, both in animals and in humans, indicate that ozone has the potential to cause adverse effects to human health, such as:

- Short-duration exposure produces inflammation of the respiratory tract, mainly in the upper airways and in the transition region between the respiratory bronchioles and alveoli;
- Studies in intoxication chambers demonstrate that the ozone levels present in large Brazilian cities (160 $\mu g/m^3$) are capable of inducing significant pulmonary inflammation in both humans and animals; this is established a few hours after the end of exposure;
- Ozone inhalation can induce a systemic inflammatory response that is characterized by the activation of serum levels of complement and acute-phase proteins;
- Ozone inhalation impairs pulmonary defenses through the functional impairment of the mucociliary system, reducing the activity of alveolar macrophages and impairing activation of circulating lymphocytes;
- Ambient levels of ozone cause increased bronchial reactivity;
- Repeated inhalation of ozone leads to a degree of adaptation by the recipient, through an increase in production of anti-oxidant substances by the respiratory tract. However, we must caution that this "adaptation" does not prevent the development of pulmonary inflammation, especially in terminal bronchiolar units;
- Some host factors modulate the magnitude of the response to ozone, such as age, respiratory co-morbidity, and genetic factors that modulate the synthesis of anti-oxidant substances through the respiratory tract.

a Effects of ozone on morbidity indicators

There is convincing evidence that ambient levels of ozone are associated with increased morbidity in the exposed population. The indicators of morbidity most consistently associated with ambient ozone variations are school absence, hospitalization for asthma and respiratory infections in emergency rooms, and episodes of worsening chronic obstructive pulmonary disease.

Romieu et al (1992) showed a 20% increase in infant day care absences due to respiratory infections in Mexico City, when ozone levels remained above 260 $\mu g/m^3$ for two consecutive days. A study of first grade students in 12 Californian cities found that an increase of 40 $\mu g/m^3$ of ozone was associated with an increase of 62.9% in general absences, 82.9% for respiratory diseases, and 45.1% for diseases of the lower respiratory tract (Gilliland et al, 2001). A Nevada study found a 13% increase in absence of first graders with eight-hour increments of 100 $\mu g/m^3$ in average ozone concentration (Chen et al, 2000). And a study

conducted with first grade Korean students showed that an increase of 32 $\mu g/m^3$ was related to an increase of 8% in absences (Park et al, 2002). These studies suggest that data for school absences can constitute an extremely sensitive instrument to detect acute effects of ozone on the infant population.

Monitoring the severity of asthma in children is another approach that has been used successfully to determine the adverse effects of ozone. A study in New Haven, Connecticut, showed that an increase of 100 $\mu g/m^3$ in hourly ozone levels was associated with increases of 35% in cases of wheezing and 47% in cases of respiratory symptoms (Gent et al, 2003). In a cohort study of 846 asthmatic children, an increase of 30 $\mu g/m^3$ was associated with morning respiratory symptoms (16%), followed by reduction of peak expiratory flow (Mortimer et al, 2000, 2002).

With regard to hospital admissions, the magnitude of the effects of ozone on the exposed population depends on the climate in the region where the study is conducted and on the type of indicator used. Given the large number of publications on this subject, **Table 3** summarizes the expected effects of different concentrations of ozone on hospital admissions.

b Effects of ozone on mortality

Contrary to the situation for particulate material, the relationship between ozone and mortality is less clear. This is because the magnitude of the effects was significantly affected by the specifications of the statistical models employed or the geographical location of the community being studied. However, studies conducted in various cities and more recent meta-analyses show that there are acute effects of variations in ozone and mortality of the exposed population, with an average coefficient of 0.256% in excess deaths for a 10 $\mu g/m^3$ increase of ozone. A summary of these studies can be seen in **Table 4**.

As for chronic effects, exposure to ozone has been linked to reduced lung function in children. However, the association with decreased life expectancy and increased risk for developing cancer has not yet been clarified.

Ambient concentrations of aldehydes in Brazilian cities

The research available in the scientific literature is somewhat disappointing, with a relatively small number of atmospheric aldehyde measurements. Taken together, these studies show that these measurements were a result of the initiative of research groups interested in the subject, rather than any systematic attempt at ambient monitoring of air quality for the purpose of safeguarding public health. This is a concern in a country such as ours, where mobile sources have high potential for emission of atmospheric aldehydes, given the wide use of ethanol, natural gas and diesel.

Table 5 shows available data for measurements of atmospheric concentrations of formaldehyde and acetaldehyde in some Brazilian cities.

Figures 4 and 5 show temporal variation of formaldehyde and acetaldehyde measurements in cities where data could be found in the literature. The compiled data does not permit a clear trace of the changes over the past 20 years. Moreover, the most recent data is from 2003, and so does not reflect the impact of flex-fuel vehicle growth, as well as the high rate of conversion of vehicles to natural gas.

Table 3

Estimate of the expected increase in hospital admissions for respiratory diseases compared to changes in the ambient ozone level

Expected increase of hospital admissions due to respiratory illness	O_3 concentration ($\mu g/m^3$)	
	1-hour average	8-hour Average
5%	30	25
10%	60	50
20%	120	100

Table 4

Summary of representative studies that link acute variations of ozone with mortality

Location of study	Findings	Reference
95 North American cities	20 $\mu g/m^3$ of ozone was associated with increments of 0.52% in total mortality and 0.64% in cardio-respiratory mortality.	Bell et al, 2004
23 European cities	An increase of 10 $\mu g/m^3$ was associated with an increase of 0.33% in overall mortality, including 0.45% of cardiovascular mortality and 1.13% in respiratory mortality.	Gryparis et al, 2004
Meta-analysis of studies conducted in seven European cities	An increase of 10 $\mu g/m^3$ was associated with an increase of 0.3% in overall mortality and 0.4% of mortality from cardiovascular diseases.	Anderson et al, 2004
14 U.S. cities	AAn increase of 20 $\mu g/m^3$ in the hourly ozone average was associated with an increase of 0.23% in respiratory mortality.	Schwartz, 2005
Meta-analysis of 39 time-series studies in the United States	An increase of 10 $\mu g/m^3$ was associated with an increase of 1.1% in mortality from cardiovascular disease.	Bell et al, 2005
Meta-analysis of 43 studies conducted in different parts of the world, with seven additional North American studies	An increase of 20-$\mu g/m^3$ in the hourly ozone average was associated with an increase of 0.39% in overall mortality.	Ito et al, 2005
Meta-analysis of 28 North American studies	Increase of 0.21% in overall mortality for an increase of 10 $\mu g/m^3$ in the average concentration of ozone.	Levy et al, 2005

As can be seen in **Table 5** and **Figures 4** and **5**, there is a larger set of data for the cities of Rio de Janeiro and São Paulo. Consolidating the measurements for these two cities, the ambient values of acetaldehyde and formaldehyde are those shown in **Table 5**. Generally speaking, the formaldehyde/acetaldehyde ratio in these two cities is about 0.5. Moreover, the concentrations of formaldehyde and acetaldehyde in Rio de Janeiro and São Paulo are much higher than those observed in other cities around the world, even those of similar size.

Estimated health effects of aldehydes

As mentioned earlier, there are no Brazilian or international studies linking ambient variations of formaldehyde and acetaldehyde to population morbidity indicators. What has been established is a numeric indicator for the risk of developing cancer, especially in the respiratory tract, as a result of ambient concentrations of these aldehydes.

Table 5 **Descriptive statistics of the measurements available in the literature on ambient levels (in $\mu g/m^3$) of formaldehyde and acetaldehyde in the atmosphere of Brazilian cities**

City		Formaldehyde	Acetaldehyde
São Paulo	N	17	17
	Average	11.7	24.3
	Median	8.8	18.8
	Minimum	1.6	5.0
	Maximum	28.8	54.8
	DP	8.1	16.6
Rio de Janeiro	N	8	8
	Average	11.7	26.2
	Median	8.9	10.7
	Minimum	2.3	3.4
	Maximum	33.0	86.3
	DP	9.7	31.6
Londrina	N	4	4
	Average	5.7	4.7
	Median	5.7	3.8
	Minimum	1.2	0.8
	Maximum	9.9	10.2
	DP	3.6	4.2
Porto Alegre	N	3	3
	Average	11.5	14.9
	Median	9.0	6.9
	Minimum	5.7	6.3
	Maximum	19.6	31.7
	DP	7.3	14.5
Salvador	N	3	3
	Average	15.5	19.0
	Median	13.7	11.3
	Minimum	3.6	6.3
	Maximum	29.1	39.6
	DP	12.8	17.9

Source: Monteiro et al, 2001; Andrade et al, 2002; Pinto et al; 2007, Martins et al, 2006.

According to census projections, the adult population (aged 20 years and over) in the metropolitan region of São Paulo is 12,674,944 inhabitants. Considering the unitary risk of developing cancer estimated for formaldehyde (1.3×10^{-5} cases per $\mu g/m^3$), and the average concentration of formaldehyde obtained from measurements in the literature (**Table 5**), we can estimate that approximately 1,928 and 678 cancer cases are due to

Figure 4

Average values and corresponding standard errors of formaldehyde concentrations measured in Brazilian cities between the years 1985 and 2003. *Formaldehyde (µg/m³)*

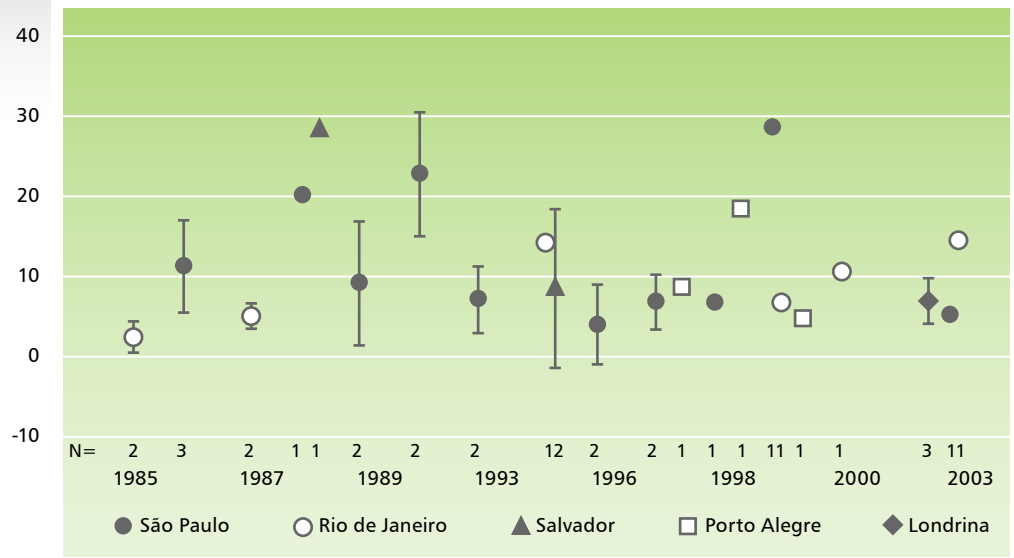
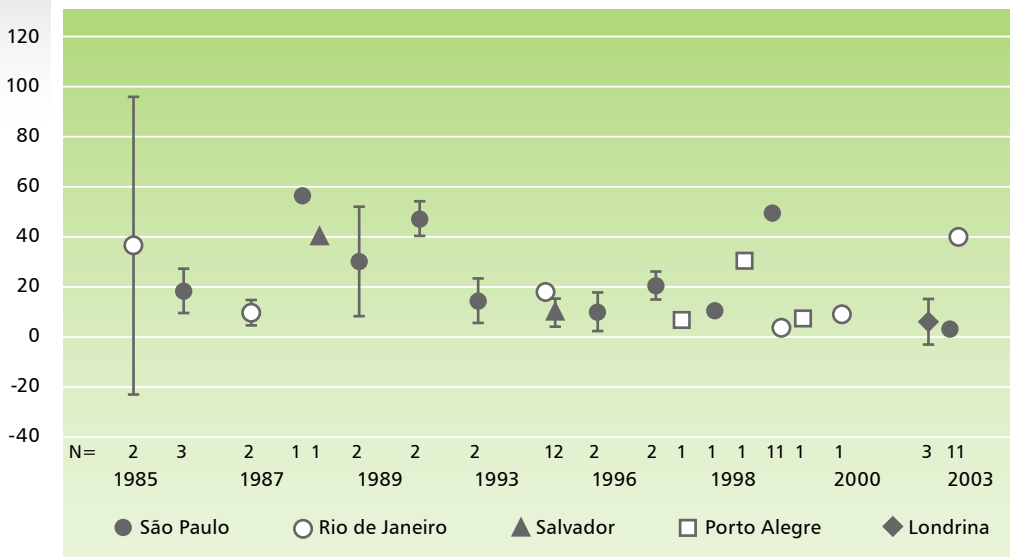


Figure 5

Average values and corresponding standard errors of acetaldehyde concentration measured in Brazilian cities between the years 1985 to 2003. *Acetaldehyde (µg/m³)*



ambient concentrations of formaldehyde and acetaldehyde, respectively, in the metropolitan region of São Paulo. Considering that the average life of residents is about 70 years, and taking into account that population data was obtained for residents aged 20 years and over, then we can say that 52 new cases of cancer caused by concentrations of both aldehydes are observed in São Paulo each year.

Another possibility is to estimate the effects of aldehydes on health in the context of their potential for ozone formation. This type of approach has the advantage of offering a certain degree of support for the creation of vehicle emission standards directed to control ozone. Only one detailed study has been done in Brazil of vehicular emission factors for volatile organic compounds for vehicles in urban traffic conditions (a study of tunnels conducted by Martins et al, 2006). The results of this study are shown in **Table 6**. The same table also presents respective values of maximum incremental reactivity (MIR) for each of the compounds evaluated in the study, together with the estimated potential for ozone formation for each compound, defined as the product of emission factors (in g.km-1) multiplied by their respective MIR.

Volatile organic compounds have different levels of reactivity, which means they may have different potentials for forming ozone and other photochemical oxidants. These differences in the effects ozone formation are referred to as "VOC reactivity". The effect of the variation in VOC emission on ozone formation in a particular episode will depend on the magnitude of the variation of the emission. The MIR scale was developed by Carter (1994) and is based on averages for the increase in reactivity, calculated for various scenarios based on chamber studies and box type models. NOx concentrations have a considerable effect

Table 6 **Values of ambient concentrations of formaldehyde and acetaldehyde obtained in different cities of the world** *(in µg/m³)*

	Formaldehyde	Acetaldehyde
Los Angeles	1.8-13	1.8-16.5
Denver	2.8-4,8	1.,8-3
Atlanta	3.3	3.7
México	43.5	4.7-5.7
Copenhague	0.3-8	0.3-33
Paris	5-40	3.7-16.5
Grenoble	3.1-22	3.6-18
Roma	10.2-21.2	5.3-12.1
Londres	5.0-32.5	2.9-5.3
Leipzig	1.6-12.5	0.7-2.3
Urawa (Japão)	3.1-14.2	2.4-12.5
Algéria	5.2-27.1	2.6-10.3
Cairo	40	–
Hong-Kong	4.9	2.4

Source: according to data reported by Cecinato et al, 2002.

on VOC reactivities. In conditions of high NOx concentration, VOC reactivities are relatively insensitive to other conditions of the study scenario. However, in conditions of low NOx concentration, relative reactivities tend to be more sensitive to other ambient conditions.

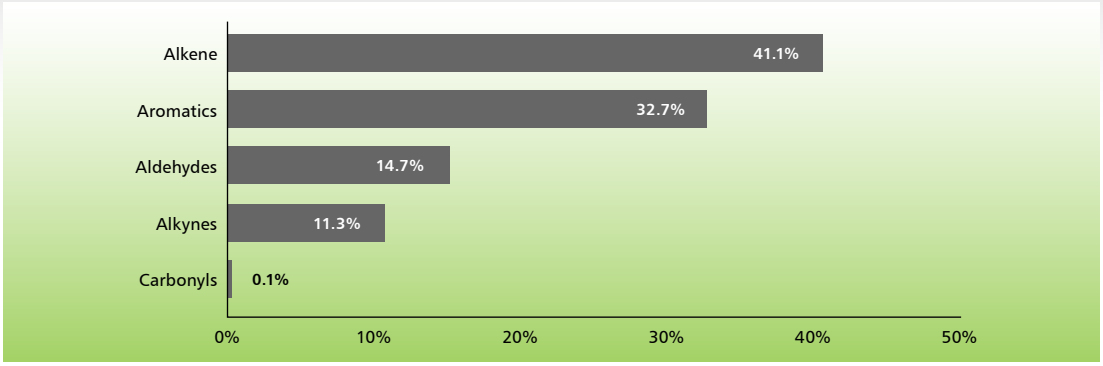
Potential for ozone formation can be represented graphically via the combination of different types of volatile organic compounds, as shown in **Figure 6**. Volatile organic compounds measured in this study represent a fraction of the total VOCs actually emitted. Thus, there is a significant part not included in the experiments. Therefore, the figures presented in **Figure 6** refer to the percentage of total VOCs measured in the interior of the tunnels.

It is important to note that the tunnels study was conducted in São Paulo in 2004, thus preceding the significant increase in flex-fuel vehicles observed in recent years. It is also important to point out that the measurements taken in the study by Martins et al (2006) represent a fraction of total volatile organic compounds emitted. In these conditions, the 14.7% potential for ozone formation attributed to aldehydes represents an over-estimation.

According to Cetesb data, annual averages for one-hour maximum ozone concentrations are around 90 $\mu g/m^3$ in São Paulo. As shown in **Figure 6**, all aldehydes together represent approximately 14.7% of the potential for ozone formation among the VOCs analyzed. The study by Grosjean et al (2002) shows that formaldehyde and acetaldehyde are the dominant aldehydes for ozone formation. If percentages attributable to aldehydes in ozone formation shown in **Figure 6** are applied to ambient concentrations measured in São Paulo, the annual averages for maximum one-hour ozone concentration would be 7.7 $\mu g/m^3$ produced by acetaldehyde and 5.6 $\mu g/m^3$ produced by formaldehyde. The estimated adverse events attributed under this scenario to direct and indirect effects of formaldehyde and acetaldehyde, for the metropolitan region of São Paulo, are presented in **Table 8**.

Figure 6 **Relative maximum potential for ozone formation of the classes of volatile organic compounds emitted by automotive sources**

Value determined by multiplying the emission rate (in g.km-1) by the respective MIR



Source: Andrade and cols, 2006.

Biofuels policy as a tool to promote public health

From what has been presented so far, we see that the process of fuel production generates pollutants that are associated with significant damage to health. Given the body of evidence about the adverse health impacts for sugar workers and the residents of towns surrounding the sugarcane plantations it is important to incorporate harvest mechanization into ethanol production, so avoiding the adverse impacts of emissions coming from straw burning (Ribeiro H., 2009).

When we look at the products of vehicular emission, acetaldehyde and formaldehyde represent a "new fact" in the use of ethanol as a light vehicle fuel, given their potential to form ozone. This is particularly important for the atmosphere in Brazilian cities that have high levels of NO_2, a situation implying that ozone formation becomes highly dependent on the increasing carbonyl concentration.

The use of ethanol as a fuel for heavy vehicles would also promote changes in the profile of emissions. With respect to aldehydes, the use of ethanol means exchanging formaldehyde emissions (a characteristic of diesel) for acetaldehyde (ethanol). More importantly, emissions of ethanol-fueled vehicles are much lower than those of the vehicles currently in use in Brazil, meaning that the emission of existing particulate matter from this automotive source would drop to virtually zero.

Each of the above cases deserves separate consideration. For the case of light vehicles that use ethanol as fuel, the increase in emissions of aldehydes is accompanied by a reduction of other volatile organic compounds associated with gasoline emissions. To simplify matters, we shall outline a situation of exchanging acetaldehyde for benzene, toluene and xylene, which are the volatile organic compounds most associated with petroleum derivatives. Considering the emission rates determined in tunnels (Martins & Andrade, 2008a) and presented in Table 7, we can infer that the potential for ozone formation of aromatic compounds (typical of petroleum products) is 6.3 times greater than that of acetaldehyde. Considering that aromatic compounds and olefins have ozone formation potential estimated from tunnel measurements of about 32.7% and 41.1%, respectively, and taking into account only the reduction of aromatic compounds, we can infer that using ethanol to replace gasoline would reduce the potential for ozone formation by about two times the current gasoline formulation. These results are consistent with those found in Martins and Andrade (2006) who – based on simulations with Eulerian photochemical models – obtained significant reductions in ozone

Table 8

Lethal effects

Estimate of the additional number of events that can be attributed to the direct effects (cases of cancer) and indirect effects (premature death from ozone formation) in the São Paulo metropolitan region, taking as a base estimates by Cetesb of direct emissions of the vehicle fleet and environmental measures of ozone.

São Paulo	Formaldehyde	Acetaldehyde	**Total**
Cancer	38	14	**52**
Premature death	120	169	**289**

Pollution in tunnels in São Paulo Table 7

Emission factors of volatile organic compounds emitted by vehicular sources, calculated from averages in tunnels in São Paulo, and the potential for ozone formation under maximum conditions defined as the product of multiplying the emissions factor by the respective MIR of each compound (gO_3/km)

Type	MIR	Emission (mg/Km)	Potential for ozone formation
toluene	2.7	134.5	363.15
1-butene	8.9	113.9	1013.71
n-pentane	1.04	87.9	91.42
cyclohexane	1.28	81.3	104.06
benzene	0.42	78.3	32.89
n-butane	1.02	74.9	76.4
M+p-xylene	7.4	62	458.8
n-hexane	0.98	60.1	58.9
1,2,4-trimethylbenzene	8.8	52.5	462
formaldehyde	7.2	48.4	348.48
acetaldehyde	5.5	45.7	251.35
o-xylene	6.5	44.4	288.6
n-heptane	0.81	41.1	33.29
1-ethyl-4-methylbenzene	8.8	32	281.6
ethylbenzene	2.7	31.1	83.97
n-octane	0.6	29.3	17.58
methylpentane	1.5	28.7	43.05
aldehydes >C2	6.3	24.9	156.87
n-nonane	0.54	22.6	12.2
isobutane	1.21	20.9	25.29
1,3,5-trimethylbenzene	10.1	20.8	210.08
1-pentene	6.2	19.6	121.52
3-methylhexane	1.4	19.5	27.3
1-ethyl-3-methylbenzene	2.7	19.3	52.11
cumene	6.5	17.9	116.35
1-ethyl-2-methylbenzene	8.8	16.4	144.32
decanoic acid	0.46	14	6.44
n-propylbenzene	2.1	12.2	25.62
methylcyclopentane	2.8	11.2	31.36
n-undecanoic acid	0.42	9.6	4.03
acetone	0.56	9.3	5.21
methylcyclohexane	1.8	9.2	16.56
1-methylethyl benzene	3	8.3	24.9
2,3-dimethylpentane	1.31	7.9	10.35
isoprene	9.1	7.6	69.16
2-butanone	1.02	6.9	7.04
1-hexene	4.4	6.8	29.92
n-dodecanoic acid	0.38	6.2	2.36
Styrene	2.2	5.7	12.54
2,2-dimethylbutane	0.82	4	3.28
2.4-dimethylpentane	1.5	3.7	5.55
2,3-dimethylhexane	1.31	3.3	4.32

Source: Martins et al, 2006.

production when considering a fictional scenario to replace all gasoline with ethanol in the light vehicle fleet. Various scenarios were considered for different gasoline formulas with reduced aromatics and olefins, but even so the use of ethanol had the effect of a greater reduction in ozone production.

Particulate matter also merits detailed analysis. An analysis of filters containing fine particulate matter, conducted over the past three years and associated with receptor modeling, indicates that diesel vehicle emissions account for about 25% of ambient concentrations of this pollutant in the cities of São Paulo and Rio de Janeiro. Since particulate matter emissions from heavy vehicles running on ethanol are practically nonexistent, the use of ethanol as fuel for heavy vehicles offers significant potential for reducing fine particulate matter, which is clearly associated with adverse health effects.

With respect to mortality, long-term studies conducted by Pope and colleagues (Pope et al, 2002) indicate that an increase of 10 $\mu g/m^3$ in the annual average of fine particulate matter leads to a 6% increase in general mortality.

As for morbidity, several epidemiological studies available in the literature relate morbid effects with both respiratory and cardio-vascular diseases for different age groups of the population, principally in terms of hospital admissions.

Given that we know the epidemiological coefficients for ozone and fine particulate matter, we can calculate the expected variations in health outcomes for changes in pollutant concentration levels by using **equation 12**:

$$[Events\ (MPolt)] = [exp\ (\beta*(MPolt)-1] * Total\ Events$$

where **Events** is the total number of morbid outcomes associated with ambient exposure;
MPolt is the average change in pollutant concentration;
exp is the exponential function;
$\boldsymbol{\beta}$ is the regression coefficient obtained through epidemiologic studies; and
Total Events is the total number of morbid outcomes in the period under review.

Estimated ambient concentrations in scenarios for fuel substitution

We need data on health outcomes in order to apply the function described in **equation 12**. Figures regarding mortality and hospital admissions under the public health system, as well as the coverage rate of the private health care system, can be obtained from DATASUS databases. The relationship between remuneration of hospital admissions paid for by the public system and those paid for by the private health system were obtained from the São Paulo Hospital das Clinicas. Ambient concentrations of ozone, the availability of ethanol and gasoline in the metropolitan region of São Paulo, and the composition of gasohol (gasoline blended with ethanol) are available on the website of Cetesb, the São Paulo State environmental agency, while the concentrations of inhalable fine particulate matter are at the stage of being published.

As ozone is not a pollutant emitted directly by vehicles, but results from the photochemical reaction of several so-called precursor gases that are emitted by vehicles, among other sources, we shall consider the study by Martins and Andrade (2008b). This study used modeling and simulation to estimate a reduction of 29 mg/m^3 in the ambient concentration of ozone if all gasoline were to be substituted by ethanol in the São Paulo vehicle fleet. Scenarios for the partial replacement of gasoline by ethanol therefore use a proportional reduction of this ozone concentration.

Thus, the partial replacement of gasoline with ethanol would reduce the direct vehicular emission of precursor gases, and be potentially capable of reducing the ozone concentration in the same proportion to the maximum reduction estimated in the above-mentioned study. Were ethanol to be used by the diesel fleet, this would promote a direct reduction in fine particle emissions.

Based on the above assumptions, we can estimate the reduction in expected deaths and hospital admissions, avoided by ozone reduction, due to ethanol use in the scenarios of 5%, 10%, 15% and 100% gasoline replacement, and the reduction in expected deaths and hospital admissions avoided by the reduction of fine particulate matter under scenarios of 5%, 10%, 15%, 50% and 100% ethanol use in the heavy vehicle fleet.

Table 9 presents the impacts on the ambient concentration of inhalable fine particulate matter (MP2.5) for the diesel/ethanol scenarios, and ozone for the gasoline/ethanol scenarios.

As shown in **Table 9**, scenarios for ethanol use in place of diesel allow us to estimate a reduction in the ambient concentration of inhalable fine particulate matter of 2% to 25%, depending on the replacement scenario. When ethanol replaces gasoline, then depending on the replacement scenario we can expected a decrease of 2% to 30% in the ambient concentrations of ozone (due to precursors).

Estimated health costs avoided through environmental improvement

Establishing priorities for health prevention and management implies the need to estimate the cost of adverse effects of diseases. It is also important as an instrument for public management. Several approaches can be used to achieve this goal, the most direct one being to estimate the expenditure on direct investments in the health system and of expenditure lost due to the consequences of disease.

There are various approaches to determining environmental costs, and more specifically the public health costs arising from a particular environmental variation, which in the case of this study is a change in air quality.

The allocation of economic value to natural resources is based on principles of neoclassical economics, where the approach is to assign monetary values to social and environmental losses that result from ambient degradation. Thus, the idea of attributing economic value seeks to treat the social costs and benefits provided by the environment as an economic agent (Pearce, 1987).

The most accurate way to measure the impact of air pollution in a given region is to conduct epidemiological studies to establish dose-response functions that correlate morbidity and mortality in susceptible populations with ambient air concentrations.

Several methods have been used in various studies to put a value on health costs associated with ambient pollution. These methods can be grouped into two broad categories. The first approach – defensive expenditure – includes methods that measure only direct income loss (lost wages and additional costs). These do not include inconvenience, suffering, loss of leisure and other intangible impacts on individuals and on family well-being, and may well disregard or seriously underestimate the health costs of people who are not part of the labor market. This approach therefore indicates only the lower limit of the social costs of pollution and underestimates the total cost for individuals. The second broad approach – quota valuation – includes methods that attempt to capture the willingness of individuals to pay to avoid or reduce the risk of death or disease.

The cost of illness approach is applied to morbidity. Direct morbidity costs can be divided into two categories: medical expenses for treatment of ailments (cost of hospital care and out-patient treatment) and loss of wages during hospitalization, days missed at work and other times when activities are significantly restricted due to illness.

The preventive cost approach constitutes an attempt to infer the minimum amount that people are willing to pay to reduce health risks, and are calculated based on the amount that people living in polluted areas spend on preventive measures. For example, expenditure on bottled water to avoid water-borne diseases, or installation of air filters to prevent indoor air pollution.

Table 9

Scenarios for the reduction of particulate material

Expected behavior of a 2.5 PM concentration for scenarios of replacement of diesel by ethanol in the heavy vehicle fleet, and in the ozone concentration for scenarios for the substitution of gasoline by ethanol in the light vehicle fleet in the São Paulo Metropolitan Region.

Fuel substitution scenario	Environmental expectation for the pollutant concentration	Expected variation
5% of diesel by ethanol	Reduction in the 2.5 PM from direct emission	Up to 2%
10% of diesel by ethanol	Reduction in the 2.5 PM from direct emission	Up to 3%
15% of diesel by ethanol	Reduction in the 2.5 PM from direct emission	Up to 4%
50% of diesel by ethanol	Reduction in the 2.5 PM from direct emission	Up to 13%
100% of diesel by ethanol	Reduction in the 2.5 PM from direct emission	Up to 25%
5% of gasoline by ethanol	Potential reduction of ozone from precursor emissions	Up to 2%
10% of gasoline by ethanol	Potential reduction of ozone from precursor emissions	Up to 3%
15% of gasoline by ethanol	Potential reduction of ozone from precursor emissions	Up to 5%
100% of gasoline by ethanol	Potential reduction of ozone from precursor emissions	Up to 30%

The contingency value approach uses survey data to determine how much people are willing to pay to reduce the risk of premature death from disease. Studies on contingency valuation produced Values of a Statistical Life (VSL) that are relatively lower than the wage differential, ranging from US$1.2 million to US$9.7 million per statistical life (IEI, 1992; U.S. EPA, 1997).

The technique of calculating the economic value of health impacts used to make this estimate was based on evidence from epidemiological studies and economic theory, developed by the World Health Organization (WHO) and by Harvard University. It is called the Disability-Adjusted Life Year (DALY), implying the years of life lost or lived with disability (Murray and Lopez, 1996). This method is based on studies that associate an ambient factor (in this case pollution) with a health indicator (hospital admissions and mortality) to estimate how many years each adverse health event has impacted the population. In other words, how many years of life each affected person has lived with temporary or permanent disability – defined as health status less than perfect – and how many years of life were lost, relative to their life expectancy, by each person suffering premature death. The years of life indicator can be converted into a monetary base for the purpose of a cost-benefit evaluation (Miraglia, 2002).

Techniques to place an economic value on health impacts constitute a tool for evaluating projects and policies for public health and pollution control, lending support to the decision making process.

In this sense, estimating potential health costs that could be avoided by virtue of ambient improvement in air quality arising from the adoption of scenarios to use ethanol in place of gasoline in light vehicles, and in place of diesel in heavy vehicles, provide this analysis with an important parameter for benchmarking biofuels policy.

Mortality – annual avoided costs

Tabela 10 shows total annual deaths that would potentially be avoided through the use of ethanol in the various scenarios, due to improvements in ambient concentrations of ozone and fine particulate matter, together with the related value of avoided mortality costs. The calculation of value of deaths avoided by the reductions in concentrations of ozone and fine particulate matter were obtained from the average values of years of life lost due to ambient concentrations of air pollutants in São Paulo (Miraglia et al, 2005) applied to scenarios for potential mortality reductions (**Table 9**) and using the current rates of life expectancy of the population (IBGE, 2008).

As can be seen in **Table 10**, the potential for avoided mortality through the introduction of ethanol into the energy matrix can be translated into annual economic benefits ranging from US$1 million to US$133 million, respectively, for scenarios of ethanol replacing 5% of gasoline, and 100% of diesel. These values point to the magnitude of potential benefits that could come from the implementation of a biofuel policy for the Metropolitan Region of São Paulo, even under the conservative scenarios.

Morbidity – annual avoided costs

The estimate of morbidity detailed here takes into account only those costs associated with hospital admissions for diseases and population age groups that are most consistently associated with air pollution, namely, hospital admissions for respiratory diseases for children up to four years of age and adults over 40 years of age, and cardiovascular disease only for adults over 40 years of age. This estimate may therefore be considered conservative, by omitting other less frequent outcomes and other age groups, but it is in line with the criteria commonly used in this type of estimate.

Table 11 shows the benefit in terms of morbidity reduction that would arise as a result of using ethanol in place of gasoline and diesel. It also presents estimates of value for each fleet replacement scenario, using the methodology described.

Conservative estimates therefore indicate that using ethanol in the replacement scenarios described above would create a morbidity reduction that, translated into economic benefits, would range from US$0.6 million to $19.8 million annually for the scenarios of ethanol replacing 5% diesel and 100% of gasoline, respectively, and considering only the Metropolitan Region of São Paulo.

Final thoughts, and an analysis of uncertainties

This chapter assumes that impacts on human health should be part of the life cycle analysis of fuels. The exposure of entire populations to atmospheric emissions, both in the fuel production process as well as from vehicle emissions in major metropolitan areas, clearly points in that direction. In the case of ethanol, both positive and negative characteristics have been identified for its use as an alternative to fossil fuels.

Table 10 **Potential annual variation in mortality by scenarios of ethanol addition in the metropolitan region of São Paulo and its economic assessment**

Scenario for substitution of fuels	Annual Mortality		
	Diagnosis	Quantity	US$ million
5% of diesel by ethanol	Reduction	37	6.63
10% of diesel by ethanol	Reduction	75	13.45
15% of diesel by ethanol	Reduction	112	20.08
50% of diesel by ethanol	Reduction	373	66.89
100% of diesel by ethanol	Reduction	745	133.60
5% of gasoline by ethanol	Reduction	6	1.07
10% of gasoline by ethanol	Reduction	13	2.33
15% of gasoline by ethanol	Reduction	19	3.40
100% of gasoline by ethanol	Reduction	130	23.31

The main negative aspects in the ethanol production process are the process of burning off straw during harvest, and the question of water balance during the plant's growing cycle, together with the problems of mono-culture and land use. Fortunately, the industry can significantly reduce this trend via self regulation. The assessment of the current impacts of sugarcane straw burning is based on the few studies that have been conducted in São Paulo. The absence of an efficient monitoring network in rural areas complicates an in-depth analysis of this issue. Burning sugarcane straw also undermines bio-ethanol's efficiency in the balance of greenhouse gases. Considering these two points – local effects of pollutants and global effects on climate – the conclusion must be that there is no environmental or human health argument to justify straw burning prior to harvest.

With regards to the effects of vehicle emissions, ethanol has advantages over both gasoline and diesel. This is evidenced by the favorable balance in terms of global climate change, and also as a factor in reducing tropospheric ozone production (when used as a substitute for gasoline) and aerosol (substituting diesel). Given the quality of petroleum derivative fuels sold in Brazil today, ethanol constitutes an alternative within the list of measures and possible improvements in terms of air quality and reduced health impacts of air pollution. One of the most significant aspects, in our view, is using ethanol in bus fleets in major urban centers.

The impact of ethanol on ozone production is one of the key points in the discussion about the health effects of ethanol emissions. Given the current formulation of Brazilian gasoline, current vehicle technology, and the scenario of high concentrations of nitrogen oxides in Brazilian cities, our projections are that fuel ethanol reduces tropospheric ozone formation. In this case, there are some degrees of uncertainty. The most significant limitation here is the lack of historical and consolidated data for ambient concentrations of aldehydes in the regions under study. The available data relate to sampling periods with different sampling times and were usually performed in isolated spots. It is regrettable that Brazil, where the last three decades have seen significant changes in the matrix of automotive fuels, has paid so little attention to ambient measurement of aldehydes. Another limiting aspect of the study is the shortage of data for auto-

Table 11 **Annual potential variation of morbidity under scenarios for the addition of ethanol in the São Paulo Metropolitan Region, and respective economic values**

Scenario for substitution of fuels	Annual morbidity Hospitalizations (SUS and private)		
	Diagnosis	Quantity	US$ million
5% of diesel by ethanol	Reduction	224	0.63
10% of diesel by ethanol	Reduction	450	1.26
15% of diesel by ethanol	Reduction	675	1.89
50% of diesel by ethanol	Reduction	2,270	6.38
100% of diesel by ethanol	Reduction	4,588	12.86
5% of gasoline by ethanol	Reduction	398	0.98
10% of gasoline by ethanol	Reduction	795	1.96
15% of gasoline by ethanol	Reduction	1,193	2.95
100% of gasoline by ethanol	Reduction	8,002	19.79

motive emissions based on measurements in the field, such as the tunnels experiments mentioned earlier. Significant changes in fleet profile – the introduction of flex-fuel vehicles and the significant conversion of part of the fleet to vehicles running on natural gas – could not be considered in this study, a step that would have allowed health risks to be broken down by sectors of the vehicle fleet.

The two above-mentioned factors prevent the creation of photochemical models with the accuracy needed to assess the contribution of different fuels to the production of aldehydes and ozone. It is therefore necessary to obtain this key information to reduce the uncertainty of the estimates and, as a consequence, to support the development of consistent public policies in the areas of air pollution and public health in urban centers.

There is also a lack of studies on the effects of biofuels use on the emissions of heavy vehicles, with respect to the behavior of the engine and the filter system for particulate matter. There are many studies regarding changes in emissions of nitrogen oxides and fine particles due to the use of biofuels in heavy vehicle engines.

The calculation of the economic value of environmental benefits translated into health indicators shows a favorable scenario for implementing such a change in the current energy matrix, increasing resources for other investments which should prioritize the health of populations exposed to air pollutants, as well as rail transportation.

Concept of adverse or harmful effect on human health

Annex 1

The most widely adopted definition used to describe an adverse health effect is the one approved by the American Thoracic Society (1995). This defines a health impact as "a significant medical event, characterized by one or more of the following factors: **1)** interference with the normal activity of the affected individuals; **2)** episodic respiratory illness; **3)** incapacitating illness; **4)** permanent respiratory disease; **5)** progressive respiratory dysfunction."

In 2000, in light of new scientific knowledge, the American Thoracic Society expanded the scope of its previous definition, incorporating the following events: biomarkers, quality of life, physiological effects, symptoms, increased demand for medical care and finally, mortality (American Thoracic Society, 2000). More recently, in 2004, the American Cardiology Society (Brook et al, 2004) published a document acknowledging air pollution as a risk factor for aggravating cardiovascular diseases, in particular acute myocardial infarction, congestive heart failure, and the development of arrhythmias.

Studies with data from the American Cancer Society (Pope et al, 2002) include lung cancer as an indicator of the effects of air pollution. Finally, reproductive alterations such as low birth weight, miscarriage and abnormal sex ratio at birth have also been incorporated into the set of indicators of significant adverse effects of air pollution.

As mentioned above, several adverse effects of air pollution on human health can be identified. Some of them are acutely apparent, manifesting just hours or days after exposure, while others become evident only after long periods of exposure. Both the acute and chronic effects may exhibit different levels of severity, ranging from vague discomfort to death – the outcome of greatest severity. Some examples may help to better clarify these ideas. Increases in air pollution will cause cognitive alterations or non-specific irritability in a large fraction of the population. A smaller proportion of exposed individuals will present increases in plasma markers and lung inflammation, indicating the presence of subclinical inflammation. In an even smaller proportion of the population, this inflammation may cause functional alterations such as increased blood pressure, mild disturbance of autonomic heart control or a reduction in indicators of lung function. At a greater level of severity, chronic users of medication to control respiratory and cardiac diseases (for example, asthma and hypertension) will need larger amounts of drugs to control their conditions. There will be those who, unable to control the changes by themselves, consult a doctor or, in more serious cases, will receive treatment at first aid posts or hospitals. Finally, a fraction of those affected will die the same day, or a few days later, due to the effects of the pollution to which they were exposed (**Figure 2**).

Most studies that use severe outcomes, such as respiratory hospitalizations and mortality, to assess acute pollution effects are likely to have human health and air pollution related coefficients that undermine the real effects, since events that affect quality of life, such as impaired control of chronic diseases, are not accounted for by the lack of mandatory reporting of such issues.

Annex 1 Concept of adverse or harmful effect on human health

Long-term studies that monitor population groups for extended periods of time recognized that pollution effects could only be detected after years of exposure. Similar to the effects of cigarettes, which only manifest after years of tobacco consumption, pollution presents, to a lesser extent, some of the same chronic effects. Table 2 shows the relationship of some chronic effects of air pollution.

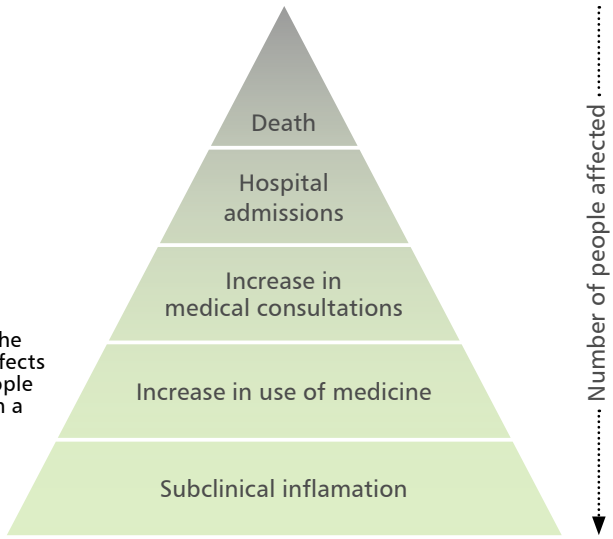


Figure 2 Diagrammatic representation of the relationship between the severity of pollution effects and the number of people affected by pollution in a given community.

Source: adapted from American Thoracic Society, 2000.

Table 2 Some of the secondary outcomes most consistently reported in the literature as relating to chronic exposure to air pollutants

Increased respiratory symptoms	Worsening of arterial atherosclerotic
Reduced lung function	Increased frequency of abortions
Reduction of birth weight	Higher incidence of pulmonary neoplasms
Greater incidence of obstructive pulmonary disease	Loss of years of life due to cardio-respiratory diseases

Source: adapted from WHO, 2006.

Aldehydes as ozone precursors — Annex 2

The general simplified equations governing photochemical atmospheric pollution can be summarized as follows:

NO_2 is dissociated by the action of ultraviolet rays forming NO and atomic oxygen;

(1) $$NO_2 + h\nu\ (\lambda \leq 430\ nm) \rightarrow NO + O$$

The oxygen atom combines with one molecule of oxygen to form ozone;

(2) $$O + O_2 \rightarrow O_3$$

Ozone is decomposed by the reaction with NO, forming NO_2 and one oxygen molecule;

(3) $$NO + O_3 \rightarrow NO_2 + O_2$$

The process described in reactions 1 to 3 is photostationary. In other words, the balance of ozone production is close to zero. However, the atmosphere of major cities favors the disruption of photostationary cycle, allowing the generation of significant amounts of ozone, as clearly shown by environmental measurements taken in Brazilian urban centers. The reaction of NO with peroxides is the greatest cause of photochemical imbalance, as shown in reaction 4;

(4) $$NO + RO_2\cdot \rightarrow NO_2 + RO$$

Atmospheric peroxides are formed by the oxidation of several organic compounds, as shown in the following equations, which illustrate possible outcomes of the oxidation of an alkene;

(5) $$RCHCHR + O \rightarrow RCH_2\cdot + RCO\cdot$$ (generation of free radicals)

(6) $$RCH_2\cdot + O_2 \rightarrow RCH_2O_2$$ (generation of peroxides)

(7) $$RCH_2O\cdot + O_2 \rightarrow RCHO + HO_2\cdot$$ (generation of aldehydes)

(8) $$RCH_2O_2\cdot + NO_2 \rightarrow RCH_2O_2NO_2$$ (generation of organic nitrates)

The group of above-mentioned reactions exemplifies some of the possibilities by which volatile organic compounds (VOCs), such as aldehydes, can be generated or interfere with the stationary photochemical equilibrium, allowing the formation of ozone. The process can be summarized as follows: in the absence of VOCs, the amount of ozone formed in the troposphere is very low; the presence of VOCs can consume NO or convert NO to NO2, creating the real possibility of the formation of ozone, according to the following general formulas:

(9) $$VOC + OH\cdot \rightarrow RO_2\cdot + intermediate\ products$$

(10) $$RO_2\cdot + NO \rightarrow NO_2 + free\ radicals$$

(11) $$radicals \rightarrow OH\cdot + intermediate\ products$$

Annex 2 Aldehydes as Ozone Precursors

Under the conditions set out above, the process of ozone formation is dependent on the amount of VOCs available in the troposphere, as well as the amount of OH radicals or other chemical species with which VOCs can interact (Carter, 1994). The influence of VOCs on ozone formation depends on the amount of NOx available. If NOx levels are sufficiently high, the amount of VOCs is the limiting factor for ozone formation. In these conditions, when NOx concentrations are high, the NOx inhibits the formation of ozone because the reaction of OH with NO_2 limits the formation of reactive species in the atmosphere. On the other hand, when NOx concentrations are low, ozone formation is dependent on the availability of NOx, causing the increase in the concentration of NOx to increase the rate of ozone formation.

These equations explain the sequence of ozone formation. However, at night and close to large sources of NO (for example high-traffic zones), ozone concentrations are reduced via processes of removal of O_3 by the reaction with NO [**equation 3**].

During the day this reaction is generally balanced by the photolysis of NO_2 [**equation 1**]. However in the vicinity of large emissions of NO the net result is the conversion of O3 to NO_2. In the vicinity of such sources, ozone is consumed and can become higher as the plume moves with the wind (plume aging). As there is no photolysis of NO_2 at night, this [**equation 3**] leads to ozone removal.

The classification between systems with saturation of NOx and sensitive to NOx (NOx-limited) is determined by the chemistry of hydroxyl radical (OH) and hydroperoxide (HO_2) and peroxyl radicals in organic RO_2 form.

The atmosphere manifests a system that is sensitive to NOx (NOx-limited) when peroxides and carboxylic acids represent the dominant radical sink. In this case, the ambient concentrations of HO_2 and RO_2 will be determined by the balance between free radical sources and reactions for the formation of peroxides and carboxylic acids.

Since the rate of peroxide formation is quadratic in HO_2, ambient concentrations of HO_2 and RO_2 vary only slightly in response to changes in NOx and VOC. The rate of ozone formation is determined by the reaction of HO_2 and RO_2 with NO. In polluted regions the rate of ozone formation is generally little affected by variations in VOC. In more remote areas, the rate of ozone formation also increases with the increase in concentration of VOCs.

NOx-saturated regimes (VOC-limited) occur when nitric acid is the dominant radical sink. In this case, ambient concentrations of OH will be determined by the balance between free radicals and the reaction of OH with NO_2. Since the rate of nitric acid formation increases with NO_2, the ambient OH decreases with the increase of NO_2. The rate of ozone formation is determined by the rate of VOC and CO reaction with OH. This rate increases with increasing VOC and decreases with increasing NOx.

Aldehydes as Ozone Precursors Annex 2

The division between regimes that are sensitive to NOx (NOx-limited) or to VOCs (VOC-limited) is closely related to the ratio of the sum of VOCs with NO_2, bearing in mind that the sums are weighted by the reactivity of the VOCs.

The ratio of free radicals to the rate of nitric acid formation is proportional to the ratio of the sum of all VOCs (weighted by reactivity with OH) with NO_2. When this ratio is high, the peroxides become the dominant radical sink and conditions are sensitive to NOx. When this ratio is low, nitric acid becomes the dominant radical sink and conditions are of NOx saturation. Ozone photolysis is the greatest source of the hydroxyl radical (OH) in the troposphere of remote regions, meaning that an increase in O_3 will produce more OH, resulting in a decrease in the lifetime of many trace species such as methane and hydrochlorofluorocarbons (HCFCs), which are species of great importance to physical and chemical processes in the stratosphere.

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Explanatory Notes

[i] Comeap (Committee on the Medical Effects of Air Pollution).
[ii] Iris (Integrated Risk Information System).