SMOG: THE EAGER DESTROYER

Like so many other pollutants, smog, or ozone, is virtually everywhere. What happens when you inhale it is not pretty.

Within seconds of entering the lung, ozone burns through cells walls in lungs and airways. Tissues redden and swell.[1] Cellular fluid seeps into the lungs[2, 3, 4, 5, 6] and over time their elasticity drops.[7, 8, 9, 10, 11, 12] Macrophages, specialized white blood cells that are the body's first line of defense against bacteria, viruses, molds and other threats, rush to the lung's aid, but they are no match for the ozone. It stuns and kills them.[13, 14]

There are other serious health damages–development of asthma,[15] increased hospital visits and stays, for example—but there also is more, much more: ozone injures the plant equivalent of lungs, the stomata, so they grow neither as fast nor as big.[16] The amount of food that farmers can harvest falls. Trees—indeed, entire forests—especially in the mountains where gale force winds, sudden ice and snow storms cause forests to hover at the brink of death, can no longer survive.[17] Entire mountainsides begin to resemble a forest holocaust.

Figure 1 Ozone destroys organic matter—cells walls in the lung, for example—including the scents released by flowers to guide honeybees to them. In air not polluted by ozone, the scents can travel 4,000 feet, but today that has been reduced by 75 percent, and may contribute to the widespread decline in honeybee populations. (Source: Jim Kalisch, Department of Entomology, University of Nebraska-Lincoln)

Then, there's global warming. The pollutant that will be the major cause of global warming in the future is carbon dioxide. But after it, vying for second and third places are a handful of other pollutants, including ozone. Carbon dioxide and other pollutants designated as "greenhouse gases" under the international agreement to regulate them, the Kyoto Protocol, are fairly evenly distributed throughout the global atmosphere. Concentrations in, say Berlin, will be roughly equal to those in Baltimore or Burbank. This is not true of ozone or the other shorter-lived pollutants that also cause global warming. Because they are being created and destroyed constantly, levels can differ over a geographic scale of a few blocks or a time frame of a few hours. Nevertheless, wherever and whenever they are found, they cause warming.

Reducing ozone and these other shorter-lived pollutants will, therefore, not only save lives and avoid illnesses, but also curb global warming. And, because their lifetimes are measured in days or weeks, not centuries, the health and cooling benefits accrue very quickly. Want to cool the planet an infinitesimal amount with two weeks? Park the car, so it is not emitting pollutants that form ozone.

The proof that reducing ozone pollution will also lessen illness and visits to doctors and hospitals has been demonstrated: In Atlanta, George, for example, drivers there were asked to park their cars and ride the bus to cut air pollution during the summer Olympics of 1996. Atlantans did, causing ozone levels to all, and when smog levels dropped, so did doctor and hospital visits, insurance claims for medicines and many other measures of sickness.[18] In northern cities, when summer changes to winter and ozone levels fall, so do deaths.[19] And when children who have been breathing dirty air move someplace where it is cleaner, their health improves.[20] In one study, lowered smog concentrations reduced the percentage of children hospitalized for respiratory problems by 77 percent."[21]

These are sensible results, because once weakened by the ozone, the body's immune system is less able to fend off infections causing hospital admissions and emergency department visits to increase.[22, 23, 24] When ozone levels increase 20 parts per billion—a common daily variation—school absences due to general illness increase 62.9 percent, while those for respiratory sickness jump 82.9 percent.[25] Children at summer camp lose the ability to breathe normally, even when the air is supposedly "clean," based on standards set by the government at the time. These losses continue for up to a week after leaving camp.[26]

To this point, the damages inflicted by ozone might have been reversed by fleeing inside or to a place where the air is cleaner. Soon, that is no longer an option. Ozone begins to kill the cells with cilia, tiny whips that move mucus and dead germs out of the lungs. As the tall, slender ciliated cells die, they are replaced by cells that are thick, stiff and non-ciliated, reducing the lungs' flexibility—and with that, the ability to breathe normally.[27, 28] Scars and lesions, not unlike those found in smokers, form in the airways.[29]

For those who live in virtually any city in the world, smog is in every breath. Few people appreciate, however, just how dangerous ozone is.

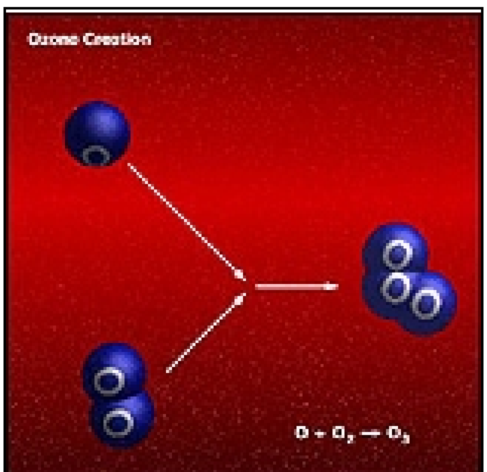

Figure 2 One oxygen atom joins with two others to form ozone, which quickly destroys whatever it contacts.

Every city and metropolitan area in the United States–indeed, with rare exceptions, every city in the world-- is plagued by high levels of ozone. In 1989, the exposed population in the U.S. was 67 million, but in warmer years like 1988, this can more than double to 135 million.[30] Some ozone is formed naturally, but the reaction between hydrocarbons (Hcs) and oxides of nitrogen (NOx), both emitted in large quantities by cars and trucks, is the principal source of the pollutant today. The reactions that form ozone are accelerated by higher temperatures, so as they increase with global warming, so will smog levels. One analysis of the effect of higher temperatures due to global warming concluded that ozone would increase 3 to 10 percent in various regions of California, and even with aggressive reduction if emissions of ozone precursors might be ineffective at bringing smog levels down in some regions, especially San Francisco.[31]

Ozone not only causes some diseases--asthma, for example–[32] it also reduces crop yields,[33] kills forests[34] and, both directly and indirectly, causes global warming. It also is associated with death in humans.[35]

There are hundreds of different hydrocarbons in the air, emitted by sources ranging from gasoline stations and printing shops to pine trees and shrubs. Each of these reacts to form a different kind of oxidant, which is a pollutant that destroys organic matter, not unlike chlorine bleach. The principal oxidant, however, is ozone, so it commonly used as shorthand for the complex mixture of poisons.[1]

Ozone so effective at destroying organic matter, whether it is dirt in a shirt or cells in a human lung, it is widely used to disinfect drinking water. In Los Angeles, for example, the$146 million filtration plant treats up to 600 million gallons of water each day, and is the second largest ozone-generating plant in the world.[36] In many hospitals, doctors and nurses use ozone to disinfect their scalpels, tongs and other medical instruments.[37]

According to the U.S. Department of Energy, "By any measure ozone is extremely toxic—one of the most toxic substances known."[38] The nearly invisible gas is, like oxides of nitrogen, formed from the Earth's own atmosphere.

The oxygen that is about 20 percent of the air is composed of two atoms tightly joined together in a nearly unbreakable bond. However, when the nuclear radiation from the sun completes its 90 million mile trip to the Earth, it is traveling so fast and with such immense force that it shatters the oxygen molecule into two atoms. Each of the atoms quickly attaches itself to an oxygen molecule, forming ozone, which has three atoms.

The bond between the single oxygen atom and the two-oxygen molecule is very weak, so ozone is extremely unstable. It is figuratively not unlike a child learning to ride a training bicycle. Just as the child is likely to fall off if the training bike hits something—a street curb, say—so, too, will the third oxygen atom break away when the ozone molecule bumps into something else. The single atom, now free, almost instantly reacts with and "oxidizes"—in other words, destroys—the other substance.

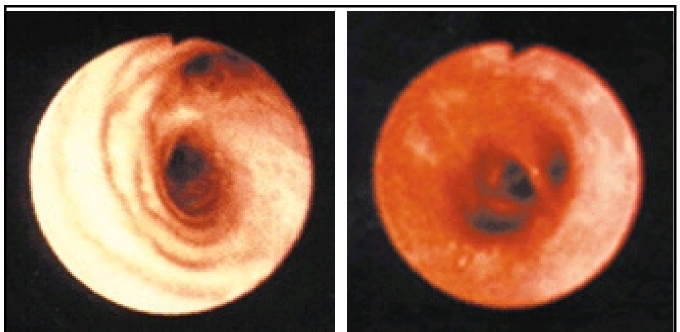

Figure 3 Typical ozone "sunburn" of lung lining, with a healthy lung airway (left) and an inflamed lung airway (right).

If the ozone happens to be in drinking water, it will kill bacteria. If it's in a hospital, blood and other residue on a scalpel will be oxidized. If the ozone is in our lungs, it almost instantly burns holes through a cell walls, and fluid begins to leak out.

Thus, whether the target is bacteria, dirt and other contaminants, they are chemically oxidized in much the way fire burns up paper or chlorine-based bleach zaps that dingy "ring around

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[1] Ozone is the pollutant measured for regulatory purposes, but there are many oxidants. See Finlayson-Pitts, B.J. & Pitts, J.N. *Chemistry of the Upper and Lower Atmosphere* (Elsevier, 1999).

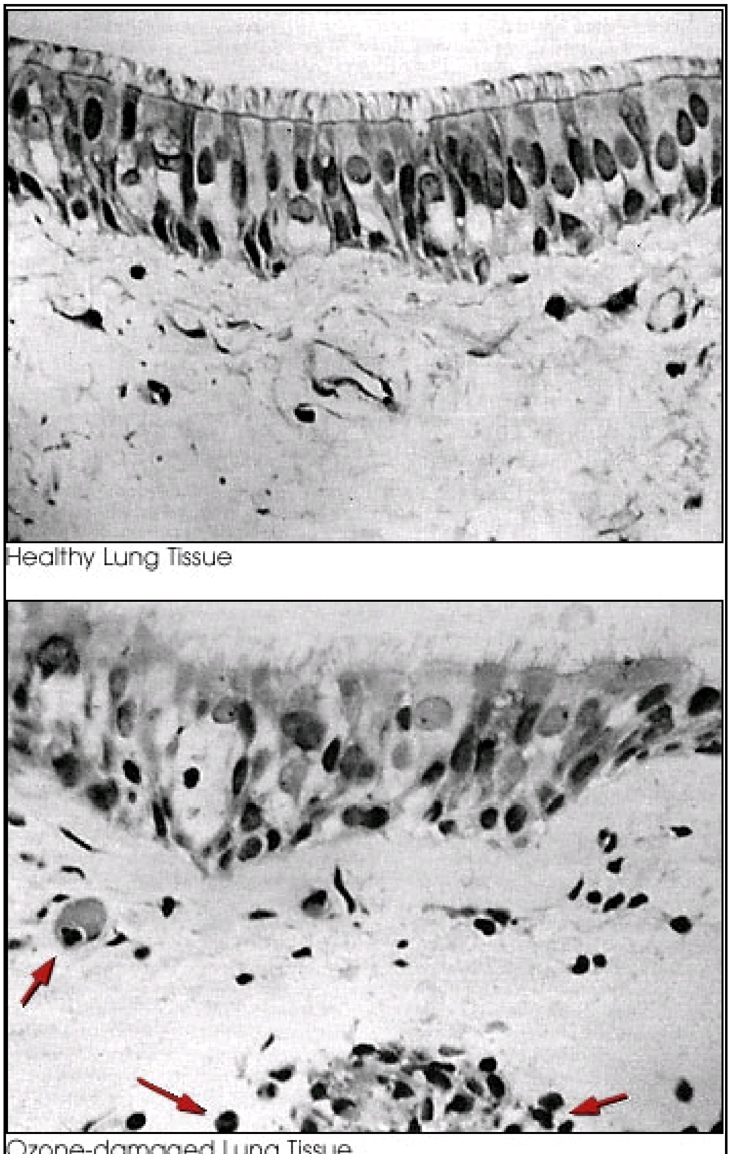

Figure 4 Microscopic views of human lung tissue (epithelium, or lining) show damage resulting from exposure to relatively low levels of ozone. In the control image (upper) from the lung of a person exposed only to air, the tiny cilia that clear the lungs of mucus appear along the top of the image in a neat and regular row. In the lung exposed to 20 ppb of ozone—a common day-to-day variation—added to the air for four hours during moderate exercise, many cilia appear missing and others appear misshapen. Arrows point to tiny bodies called neutrophils in the ozone-exposed subject. The presence of neutrophils indicates inflammation. Magnification: x400. (Micrographs courtesy of the American Thoracic Society, from American Review of Respiratory Diseases, Vol. 148, 1993, Robert Aris et al., pp. 1368—69.)

the collar." Not surprisingly, however, breathing the atmospheric equivalent of bleach isn't so good for the lungs.

Some people say there is "good" ozone—because about 90 percent of it is between about 6 to 20 miles overhead in the stratosphere, where it blocks out enough of the sun's radiation to make life on the surface habitable. Then, there's the remaining 10 percent at the level where humans, plants and animals live, and they are injured by ozone, which some call the "bad" kind.

Smog's damage starts with the first breath. It stimulates the nerve endings triggering pain, rapid breathing and shortness of breath. For many years, regulators thought the pain brought on by ozone caused these changes—but not so. When people are given pain killers, the hurt is gone but the abnormal breathing isn't. So the two—pain on the one hand and abnormal breathing on the other—are independent of one another.[39]

When the human body is being attacked, whether by bacteria or air pollution, it mounts a counter attack. In the case of ozone, specialized white blood cells that are the body's first line of defense against bacteria, viruses, molds and other threats, rush to the lung's aid, but they are no match for the ozone. It stuns and kills them.[40, 41]

With the body's immune system having been weakened by the ozone, the threat of bacterial infections increases. Even scarier, ozone begins to kill the ciliated cells, which have tiny whips that move mucus and dead germs out of the lungs. They are replaced by non-ciliated cells, which are so thick and stiff that they reduce the flexibility of the lungs and airways Over time, people become unable to inhale and exhale normally.[42, 43] (See Figure 13.)[44]

At ozone levels that prevail through much of the year in California and most other cities during warmer weather, healthy, non-smoking young men who exercise can't breathe normally. Breathing becomes rapid, shallow and painful.[45]


Figure 5 Los Angeles smog.

Ozone is so prevalent that even seemingly unpolluted rural or wilderness areas are shrouded.. In the Virginia mountains of Shenandoah National Park, for example, ozone concentrations are among the highest in the country,[46] and frequently exceed the law's health based standards.[47] Much the same is true in Acadia National Park, whose rocky cliffs hug the frigid Atlantic waters off Maine.[48] In the Great Smoky Mountains National Park in Tennessee and North Carolina ozone levels rival those of Los Angeles, while in California's Sequoia and Kings Canyon National Parks, ozone concentrations exceeded human-health standards on 61 summer days in 2001.[49] During summer, ozone levels in these parks exceed the California Health Standard at an average of one out of every three days at low-elevation sites and one out of every five days at mid-elevation sites.[50]

Indeed, although ozone concentrations tend to be highest in and downwind of cities, virtually the entire United States—excepting only portions of the Pacific Northwest—is blanketed by the pollutant at levels that have been demonstrated to reduce lung function.[51] For example, in tests, of 58 farm workers at Abbottsford and Matsqui, Canada, about 42 miles (70 kilometers) southeast of Vancouver, researchers found that as ozone concentration rose, the ability to breathe normally fell, even though average concentrations were only one-third of the U.S. standard. Ozone levels in this study were, in other words, at levels that prevail virtually constantly during the warm weather periods throughout much of North America, Europe and Asia. Even more alarming, these deficits were still present the following morning.[52]

THE STEADILY RISING TIDE

Finding injuries at very low levels is bad news, because so-called "background" concentrations of smog—those found on farms, in forests and at mountaintops—are rising.

According to the U.S. National Oceanic and Atmospheric Administration, "Over the last one hundred years the ozone concentrations near the ground in the northern middle latitudes have more than doubled. Several sources support a lower-tropospheric ozone increase of greater than 1 percent per year since the end of the nineteenth century."[53]

What this means is that smog is moving closer and closer to being a pollutant that can't be escaped, no matter your wealth or distances from a city.

At the same, a raft of studies show that smog isn't some minor problem that makes children and adults merely uncomfortable. On the contrary, smog must now be included on the short list of possible causes of the huge global increase in asthma. It now seems certain that over the long term ozone actually changes the size, shape and function of lungs, making them stiffer and smaller and causing either asthma itself, or something that looks like asthma.

There are three key studies that point to this conclusion:

Rhesus Monkeys

Rats and many other laboratory animals are born with lungs that are fully developed or nearly so. However, humans and other primates, such as rhesus monkeys, are born with respiratory systems that still are maturing.

At the University of California at Davis, rhesus monkeys of various ages were given doses of ozone designed to simulate what happens in Los Angeles, New York City and other areas with dirty air. They breathed ozone-polluted air for five days, then nine days of normal air. This was repeated over and over again every two weeks for five months.

During the tests young monkeys had all the symptoms of asthma in humans: changes in the size, shape and composition of lungs; sudden inability to breathe normally and loss of a key protective chemical, glutathione, from lung fluids and cells. Most alarming, the developing lungs stopped dividing after 9 times, instead of continuing to the normal number of 13.[54]

When changes like this happen, they show up in tests of breathing—how much air can be exhaled, etc. So, if the changes in the young monkeys were also happening in children, tests of breathing would show it. And they do.

Freshman from Polluted Areas Compared to those from Cleaner Cities

One specific breathing test, FEV_{25-75} or forced expiratory volume, measures how much air leaves the lung. If it is lower than it should be, it means that the parts of the lung just before oxygen and carbon dioxide are exchanged are blocked—another of asthma's symptoms.

Scientists at the University of California at Berkeley selected 130 freshmen to test. All were lifelong residents of either the Los Angeles Basin, where smog is the worst in the nation, or the San Francisco Bay area, which is relatively clean. When the lifetime exposures to smog were calculated and compared to FEV_{25-75}, there was a "consistent" linkage between having breathed smog and the inability to breathe normally.[55] Another study, this one of 520 Yale University students, found the same thing: breathing smog seriously and almost certainly permanently injures human lungs.[56]

Children Living in Southern California

But wait—if the direct evidence of the monkey studies and the indirect evidence of the examination of college freshman is correct, children who live and play in smog should have more lung diseases than those raised in cleaner neighborhoods. And they do.

In Southern California, scientists conducted one of the most ambitious air pollution research programs in history, a ten-year, 12-community study of the impact of air pollution on lung health and growth in thousands of children recruited from areas with varying levels of air pollution.

It is now considered throughout the world as perhaps the most comprehensive and authoritative study ever conducted on the effects of air pollution on children.

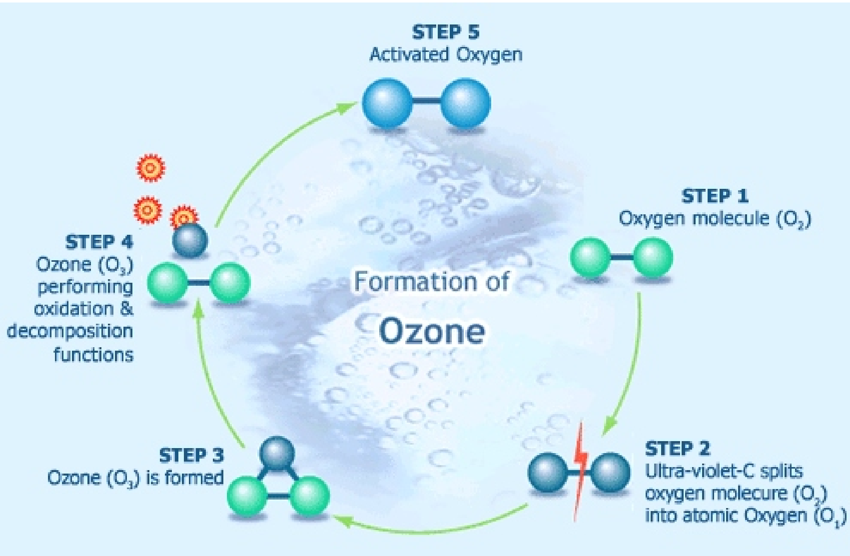

Figure 6 The cycle of ozone creation, followed by "oxidation and decomposition," which means, in plain terms, that it burns holes through your lungs and kills its cells. It does much the same to trees and other plants and, on top of all this, causes global warming as well.

Starting in 1991, researchers began tracking over 5,500 school age children, comparing their activities, illnesses, places of residence and a wide range of other things. What the study demonstrated should be a cause of alarm for every parent whose child lives in polluted cities—and most of the world's children do.

Asthma development was three times higher in children that lived in high ozone communities.[57] There was also a clear correlation between ozone levels and slowed lung growth, which was more pronounced in girls who were spending the most time outdoors.

The most profoundly alarming finding was the apparent link between exposure to ozone and the development of asthma.

Children who played three or more outdoor sports—which means they would breathe more air and the pollution in it, thus boosting the total amount of poison inhaled—and who lived in neighborhoods with higher ozone levels were three times more likely to develop asthma.[58]

Perhaps the worldwide increase in asthma has nothing to do with air pollution, including smog. Perhaps it is merely a coincidence that at the same time as an increase in background levels of ozone, there has been a jump in asthma rates. Perhaps it is also a coincidence that in places where air pollution is high, so are the asthma rates. Perhaps it is only chance that when one group of primates, rhesus monkeys, breath air pollution they develop asthma or something like it; and that when another group of primates, college freshman, have breathed air pollution for a lifetime, they, too, have breathing patterns like those of an asthmatic.

But then again, perhaps it is not a coincidence at all, but a simple reality: air pollution permanently injures infants and children, and it's getting worse, not better.

Taken together, these studies and those that have gone before, constitute a compelling, consistent and coherent body of evidence suggesting that long term exposure to ozone results in profound and permanent damage to the body's respiratory system, beginning at the earliest ages.

Injury to plants

Because ozone is so reactive, it has much the same effect on plant tissue as in humans. It enters the stomata, the tiny pores through which plants take up carbon dioxide, then once inside the leaf destroys cells.[59] The aggregate impact of these injuries is to substantially reduce the growth of a wide range of plants, including crops as well as trees. One group has concluded that increasing levels of tropospheric ozone under a business-as-usual scenario could cut global crop yields by nearly 40 percent worldwide by 2100.[60] Another group examined the indirect contribution that widespread ozone injury might have by reducing the ability of plants to take up and fix carbon dioxide. Carbon dioxide, an essential nutrient for plants, is removed by them and sequestered in their tissues. Ozone, however, by causing "significant suppression" of plant growth would lessen the ability of plants to sequester CO_2, indirectly causing global warming. Indeed, the indirect contribution to warming would greater than the direct impact.[61]

Figure 7 Potato leaf burned by ozone.

Figure 8 Forest death, or "waldsterben," in Germany because of ozone. Similar scenes can be found throughout the eastern United States, along the spine of the Appalachian and Blue Ridge Mountains.

Ozone also destroys the scents released by flowers that guide honeybees to them. In unpolluted air, the scents can travel 4,000 feet, but destruction of up to 90 percent of the aroma by ozone can reduce that distance to 650 to 1,000 feet, reducing the honeybees' food supply and starving them. Plants, in turn, are not pollinated, reducing productivity.[62]

Ozone-caused warming

Ozone is created in the lower air by complex reactions between a variety of others pollutants, especially methane, oxides of nitrogen, hydrocarbons and some other pollutants.[63] As`mentioned earlier, almost all of these pollutants have extremely short lifetimes in the air, sometimes only minutes, so their concentrations vary markedly from place to place,[64] but in the aggregate they account for a substantial fraction of current warming. Ozone traps heat being radiated from the Earth's surface that otherwise would escape into space, increasing global temperature by 0.25 to 0.65 watts per square meter, according to the IPCC.[65]

In addition to its global impact, ozone also causes warming in the Arctic, especially in the non-summer seasons. During summer, sunlight and heat accelerate atmospheric reactions that destroy ozone, preventing it from persisting long enough to be transported from mid-latitudes to the Arctic. But in the cooler temperatures of the fall, winter, and spring, ozone pollution survives long enough to be blown into the frigid regions of Alaska and elsewhere in the Arctic, where it causes 0.4˚ C to 0.5˚ C degrees warming there.[66] One team of investigators concluded that traffic in Europe–cars, trucks, ships, trains and aircraft--alone could account for 12 percent of the ozone in the Arctic and remote maritime regions during July; and, in January, 15 percent in the Arctic and 8 percent elsewhere.[67]

Figure 9 Ozone causes warming globally, but has a disproportionate impact in the Arctic.

Other studies conclude that if traffic-related pollution in all regions of the world were to reach the same per capita levels as in Europe and in the United States, ozone levels in south Asia would essentially double, rising by 30 to 50 parts per billion. Warming due to ozone from vehicles would jump to 0.27 watts per square meter (W/m^2).[68] That compares to the IPCC 2007 estimate of ozone's contribution to global warming of 0.25 to 0.65 watts per square meter.[69] Some believe the IPCC's estimate is too conservative and estimate that ozone accounts for much more warming, perhaps about 15 percent of the total.[70]

Concentrations of free, or background, ozone have been increasing for at least one century. Modern air pollution control programs have lowered levels in and around some cities, but by no means all of them. In many other cities, however, and in the countryside, smog continues its steady climb. Some studies conclude that levels have approximately doubled,[71] while others have found a five-fold increase.[72] Increases have also been found in Bavaria, Germany,[73] Ahmedabad, India,[74] and the tropical Pacific region of South America[75] to name but a few. Levels are currently increasing faster than in the past and are projected to continue rising unless action is taken to reduce precursors.[76]

That said, reducing ozone is a fairly straightforward process. The technologies and practices to minimize, or even eliminate altogether, the emissions that cause ozone are well developed and have been for decades in some cases. Catalytic converters, installed on cars starting more than 40 years ago, can be loaded with additional precious metals, for example, in new models, thus reducing their emissions still further. Similar controls can be fitted to smokestacks and have been throughout much of Europe and Japan, as well as a few places in the United States. Electricity can be produced from sunshine, wind or even heat trapped in the earth, all with zero pollution.

Thus, although the bad news is that ozone seems to be almost everywhere, ways of reducing are as well. Adopting those technologies and practices not only could but, in fact would, start saving lives, reducing illness and cooling the planet with a matter of days.

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Asthma in exercising children exposed to ozone: a cohort study
Lancet 2002; 359; 386–391
From the Southern California Children's Study. Relevant numbers;
5762 children completed baseline questionnaires;
479 excluded because they were not at school when the questionnaire was administered;
883 excluded for a history of asthma;
312 excluded because of missing answers to "wheezing" questions;
26 excluded for chest illnesses such as cystic fibrosis:
527 excluded because they had less than one year of follow-up;
This left 3535 children with no initial history of asthma; 2752 of these had no history of wheezing; 1934 played sports; 273 played three or more team sports;
There were 46 low pollution communities (O_3 daytime mean 40.0 ppb); and 46 high pollution communities (O3 mean 59.6 ppb). PM_{10} twice as high in high ozone communities (43.3 vs 21.6) and $PM_{2.5}$ three times higher (21.4 vs 7.6). NO_2 three times higher in high ozone communities (29.2 vs 10.8 ppb).
It was shown that development of asthma prospectively was three times higher in children participating in more than 3 sports in high ozone communities, compared to children who did no sports in both communities or did fewer than 3

sports in high ozone communities. No differences in development of asthma if other pollutants were studied. Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

16. See, e.g., Agricultural Research Services, U.S. Department of Agriculture, "Effects of Ozone Air Pollution on Plants," http://www.ars.usda.gov/Main/docs.htm?docid=8453. Ozone penetrates stomata and destroys organic molecules in the plant tissue.

17. See H Sandermann, A. R. Wellburn & R.L. Heath (Eds), *Forest Decline and Ozone: A Comparison of Controlled Chamber and Field Experiments*, Springer Verlag, Berlin, 1997, explores the relationship between forest decline and ozone. It comprises a broad range of methods concerning field ecology, model ecosystem research, and basic physiological and biochemical research.

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Impact of changes in transportation and Commuting behaviors during the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma
JAMA 2001: 285; 897-905
Comparison of the 17 days of the Olympic Games (July 19-Aug 4) to a baseline period consisting of the 4 weeks before and 4 weeks after the Olympic Games. Peak one hour level of O3 fell to 50-100 ppb during the games from a predicted value of about 70-120 in the comparison periods. PM10 (24 hour level) was 20-45, compared to levels of 30-70; NO2 was only slightly lower running at about 30 ppb peak one hour level compared to values between 20-65. CO also slightly lower. Traffic density measurements showed decreases of 22% in weekday 1-hour morning peak traffic counts during the Olympic Games. Ozone levels fell slightly over the same period in three different places 60 km to 100 km from Atlanta; but these changes were only about one fifth of the drop in Atlanta.
Citywide acute care visits and hospitalizations for asthma were logged. Results showed no changes in nonasthma diagnoses; decreases of 41% in Medicaid claims file, 44% decreases in HMO database; 11% decreases in two emergency pediatric departments; and decreases of 19% in Georgia Hospital Discharge Database. Lack of change in other diagnostic categories indicates that children did not leave Atlanta over the period of the Olympic Games.

19. Gryparis, A. et. al. Acute Effects of Ozone on Mortality from the "Air Pollution and Health: A European Approach" Project. Am. J. of Respiratory and Critical Care Med. Vol 170. pp. 1080-1087, (2004).
In the Air Pollution and Health: A European Approach (APHEA2) project, the effects of ambient ozone concentrations on mortality were investigated. Data were collected on daily ozone concentrations, the daily number of deaths, confounders, and potential effect modifiers from 23 cities/areas for at least 3 years since 1990. Effect estimates were obtained for each city with city-specific models and were combined using second-stage regression models. No significant effects were observed during the cold half of the year. For the warm season, an increase in the 1-hour ozone concentration by 10 μg/m3 was associated with a 0.33% (95% confidence interval [CI], 0.17–0.52) increase in the total daily number of deaths, 0.45% (95% CI, 0.22–0.69) in the number of cardiovascular deaths, and 1.13% (95% CI, 0.62–1.48) in the number of respiratory deaths. The corresponding figures for the 8-hour ozone were similar. The associations with total mortality were independent of SO2 and particulate matter with aerodynamic diameter less than 10 μm (PM10) but were somewhat confounded by NO2 and CO. Individual city estimates were heterogeneous for total (a higher standardized mortality rate was associated with larger effects) and cardiovascular mortality (larger effects were observed in southern cities). The dose–response curve of ozone effects on total mortality during the summer did not deviate significantly from linearity.

20. Avol, E. et. al. Respiratory Effects of Relocating to Areas of Differing Air Pollution. Levels. Am. J. Respir. Crit. Care Med., Volume 164, Number 11, December 2001, 2067-2072.
Investigators studied 110 children (59 boys and 51 girls, who were 10 yr of age at enrollment and 15 yr of age at follow-up) who had moved from communities participating in a 10-yr prospective study of respiratory health (The Children's Health Study [CHS]) to determine whether changes in air quality caused by relocation were associated with changes in annual lung function growth rates. The subjects were given health questionnaires and underwent spirometry in their homes across six western states, according to a protocol identical to evaluations performed annually on the CHS cohort in school. Changes in annual average exposure to particulate matter with a mean diameter of 10 μm (PM10) were associated with differences in annual lung function growth rates for FEV1, maximal midexpiratory flow, and peak expiratory flow rate. As a group, subjects who had moved to areas of lower PM10 showed increased growth in lung function and subjects who moved to communities with a higher PM10 showed decreased growth in lung function. A stronger trend was found for subjects who had migrated at least 3 yr before the follow-up visit than for those who had moved in the previous 1 to 2 yr. We conclude that changes in air pollution exposure during adolescent growth years have a measurable and potentially important effect on lung function growth

and performance.

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26. Kinney, et al. A Critical Evaluation of Acute Ozone Epidemiology Results. Arch. Env. Health 43, 168–73, 1988.

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28. Gardner DE. Use of experimental airborne infections for monitoring altered host defenses. Environ Health Perspect 1982; 43:99–107.

29. "Executive Summary," *Air Quality Criteria for Ozone and Related Photochemical Oxidants,* United States Environmental Protection Agency, (Washington, DC: Office of Research and Development), February 1994.

[30]. Friedman RM. Catching our breath: Next steps for reducing urban ozone. Office of Technology Assessment, 1989. U.S. Government Printing Office, Washington, D.C. OTA-0-412.

31. Steiner, A.L. Influence of future climate and emissions on regional air quality in California. JG R, V. 111, D18303, doi:10.1029/2005JD006935, 2006 http://www.agu.org/pubs/crossref/2006/2005JD006935.shtml.
Using a chemical transport model simulating ozone concentrations in central California, we evaluate the effects of variables associated with future changes in climate and ozone precursor emissions, including (1) increasing temperature; (2) increasing atmospheric water vapor; (3) increasing biogenic VOC emissions due to temperature; (4) projected decreases in anthropogenic NOx, VOC, and CO emissions in California for 2050; and (5) the influence of changing ozone, CO, and methane at the western boundary. Climatic changes expected for temperature, atmospheric water vapor, and biogenic VOC emissions each individually cause a 1–5% increase in the daily peak ozone. Projected reductions in anthropogenic emissions of 10–50% in NOx and 50–70% in VOCs and CO have the greatest single effect, reducing ozone by 8–15% in urban areas. Changes to the chemical boundary conditions lead to ozone increases of 6% in the San Francisco Bay area and along the west coast but only 1–2% inland. Simulations combining climate effects predict that ozone will increase 3–10% in various regions of California. This increase is partly offset by projected future emissions reductions, and a combined climate and emissions simulation yields ozone reductions of 3–9% in the Central Valley and almost no net change in the San Francisco Bay area. We find that different portions of the model domain have widely varying sensitivity to climate parameters. In particular, the San Francisco Bay region is more strongly influenced by temperature changes than inland regions, indicating that air quality in this region may worsen under future climate regimes.

32. McConnell, R., Berhane, K., Gilliand, F., London, S.J., Islam, T., Gauderman, W.J., Avol, E., Margolis, H.G., & Peters, J.M.
Asthma in exercising children exposed to ozone: a cohort study
Lancet 2002; 359; 386–391
From the Southern California Children's Study. Relevant numbers;
5762 children completed baseline questionnaires;

479 excluded because they were not at school when the questionnaire was administered;
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527 excluded because they had less than one year of follow-up;
This left 3535 children with no initial history of asthma; 2752 of these had no history of wheezing; 1934 played sports; 273 played three or more team sports;
There were 46 low pollution communities (O_3 daytime mean 40.0 ppb); and 46 high pollution communities (O3 mean 59.6 ppb). PM_{10} twice as high in high ozone communities (43.3 vs 21.6) and $PM_{2.5}$ three times higher (21.4 vs 7.6). NO_2 three times higher in high ozone communities (29.2 vs 10.8 ppb).
It was shown that development of asthma prospectively was three times higher in children participating in more than 3 sports in high ozone communities, compared to children who did no sports in both communities or did fewer than 3 sports in high ozone communities. No differences in development of asthma if other pollutants were studied. Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

33. See, e.g., Agricultural Research Services, U.S. Department of Agriculture, “Effects of Ozone Air Pollution on Plants,” http://www.ars.usda.gov/Main/docs.htm?docid=8453. Ozone penetrates stomata and destroys organic molecules in the plant tissue.

34. See H Sandermann, A. R. Wellburn & R.L. Heath (Eds), *Forest Decline and Ozone: A Comparison of Controlled Chamber and Field Experiments*, Springer Verlag, Berlin, 1997, explores the relationship between forest decline and ozone. It comprises a broad range of methods concerning field ecology, model ecosystem research, and basic physiological and biochemical research.

35. See e.g. Bell, M.L. et. Al. The Exposure-Response Curve for Ozone and Risk of Mortality and the Adequacy of Current Ozone Regulations. Environmental Health Perspectives Volume 114, Number 4, April 2006.

36. Los Angeles Department of Water and Power, “Water Quality,” http://wsoweb.ladwp.com/Aqueduct/historyoflaa/waterquality.htm.

37. Murphy, L. Ozone - the Latest Advance in Sterilization of Medical Devices. Canadian Operating Room Nursing Journal, Jun 2006.
In 2003, a Canadian company developed a unique sterilization process employing ozone as the sterilizing agent. This technology is a safe, rapid and economical alternative to other low temperature sterilization modalities and may relieve some of the pressure experienced when instruments in short supply are in high demand. The article discusses the principles of the ozone sterilizer and the cycle and will explore the advantages of using this sterilization technology.

38. U.S. Department of Energy, “UV Lamps and Ozone,” http://www.newton.dep.anl.gov/askasci/chem03/chem03323.htm.

39. Schelegle, E.S., Eldridge, M.W., Cross, C.E., Walby, W.F., & Adams, W.C. Differential effects of airway anesthesia on ozone-induced pulmonary responses in human subjects Am J Respir Crit Care Med 163; 1121-1127; 2001. 0.30 ppm O_3 inhaled for 65 minutes by 22 ozone-sensitive healthy subjects. After 50 minutes, FEV1 was reduced 24%, breathing frequency was increased 40%, VT was decreased 31%, and the subjective symptom score increased. Inhalation of tetracaine aerosol (MMD = 3.52 microns) caused marked reductions in throat irritation, cough, shortness of breath, and pain on deep inspiration. But minor and inconsistent rectification of the FEV1 occurred, and respiratory rate not significantly different from effect of saline aerosol. Authors note: “Our data are consistent with afferent endings located within the large conducting airways of the tracheobronchial tree being primarily responsible for ozone-induced subjective symptoms and provides strong evidence that ozone-induced inhibition of maximal inspiratory effort is not dependent on conscious sensations of inspiratory discomfort”. Reviews evidence that C-fiber afferent endings initiate response. Note that partial reversal of effect by lidocaine reported by Hazucha, Bates & Bromberg
(J Appl Physiol 1989;67; 1535-1541) probably attributable to the time that separated the pulmonary function tests before and after lidocaine.

40. Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D,Koren HS. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. Am J Respir Cell Molec Biol 1991; 4:72–81.

41. Harder Sd, Harris DT, House D, Koren HS. Inhibition of human natural killer cell activity following in vitro exposure to ozone. Inhal Toxicol 1990; 2:161–73.

42. Van Louveren HS; Wagenaar S, Walvoort HC, Vos JG. Effect of ozone on the defense to a respiratory Listeria monocytogenes infection in the rat. Suppression of macrophage function on cellular immunity and aggravation of histopathology in lung and liver during infection. Toxicol Appl Pharmacol 1988; 94:374–393.

43. Gardner DE. Use of experimental airborne infections for monitoring altered host defenses. Environ Health Perspect 1982; 43:99–107.

44. "Executive Summary," *Air Quality Criteria for Ozone and Related Photochemical Oxidants,* United States Environmental Protection Agency, (Washington, DC: Office of Research and Development), February 1994.

45. McDonnell WF, Horstman DH, Hazucha MJ, Seal Jr E, Haak ED, Salaam SA, House DE. Pulmonary effects of ozone exposure during exercise: dose response characteristics. J Appl Physiol 1983; 5:1345–52.

46. Isaak Walton League, "Air Pollution in Our Parks: Shenandoah National Park Fact Sheet," http://www.iwla.org/reports/parkfssh.html. Ozone is also toxic to trees and other vegetation. A decline of 26 percent to 51 percent in the growth rate of eastern white pines in the Blue Ridge Mountains from the late 1950's to mid-1970's has been attributed to ozone pollution. In Shenandoah National Park, tulip poplar, green ash, sweet gum, black locust, Eastern hemlock, Table Mountain pine, pitch pine and Virginia pine seedlings have all demonstrated growth loss at ozone levels below minimum federal health standards. National Park Service, "Air Quality in the National Parks," http://www2.nature.nps.gov/ard/pubs/aqnps.htm.

47. National Park Service, "Shenandoah National Park Natural Resource Guide," http://www.shenandoah.national-park.com/nat.htm#air.

48. National Park Service, *Air Quality in the National Parks (2d Edition)*, Washington, D.C. (Sep. 2002), http://www2.nature.nps.gov/ard/pubs/aqnps.htm.

49. National Park Service, "List of High Ozone in Park Units - 2001 Season," http://www.aqd.nps.gov/ard/gas/exceed2001.htm.

50. National Park Service, "Ozone—the Invisible Poison," http://www.nps.gov/seki/ozone.htm.

51. See e.g. Brasseur GP, et. al. "Tropospheric Ozone and Climate: Past, Present and Future," in *Present and Future of Modeling Global Change: Toward Integrated Modeling,* Eds. T. Matsuno and H. Kida, TERRAPUB, 2001, http://www.terrapub.co.jp/e-library/toyota/pdf/063.pdf.

52. Brauer, M., Blair, J., & Vedal ,S. Effect of ambient ozone exposure on lung function in farm workers Am J Respir Crit Care Med 154, 981–987, 1996 Fraser Valley study. 58 workers. Farms were at Abbottsford and Matsqui, about 70 Km southeast of Vancouver. Workers spent all day in outside work. Ambient monitors for O_3 and SO_4. $PM_{2.5}$ mean was 11.4 micrograms/m3. Ozone daily maximum was mean of 40.3 ppb, with single highest value of 84 ppb. Ozone for the workshift time had a mean of 26 ppb, and a maximum of 54 ppb. FEV1 fell -3.3 ml and FVc fell -4.7 ml for each ppb increase in ozone. Deficits still apparent on following morning.

53. Climate Monitoring and Diagnostics Laboratory, National Oceanic and Atmospheric Administration, "Observed Ozone Changes," http://www.cmdl.noaa.gov/ozwv/dobson/papers/wmobro/observed.html. A.S.L. Associates has studies historic ozone levels for the U.S. National Acid Precipitation Program and the U.S. Environmental Protection Agency. According to A.S.L., "In the mid-1800s, surface ozone was the focus of many scientific studies to prove its existence, to discover its functions in the atmosphere, and to define its role in affecting the spread of epidemics. Ozone was commonly measured using the Schoenbein ozonoscope method. Schoenbein papers were coated with iodide; the reaction with ozone formed iodine. Ozone concentration was expressed as Schoenbein numbers based on coloration of Schoenbein's test paper. Starting in the mid-1800s, more than 300 stations recorded ozone exposures in countries such as Austria, Australia, Belgium, England, France, Germany, Russia, and the United States. Based on

data evaluated, some scientists have concluded that (1) the average daily maximum of the surface ozone partial pressure in the Great Lakes area of North America was approximately 0.019 ppm, and (2) the European measurements between the 1850s and 1900 were mostly in the range of approximately 0.017 ppm to 0.023." ppm.http://www.asl-associates.com/back.htm

54. Fanucchi, M.V., Wong, V., Hinds, D., Tarkington, B., Van Winkle, L.S., Evans, M.J., & Plopper, C.G. Repeated episodes of exposure to ozone alters postnatal development of distal conducting airways in infant rhesus monkeys. Am J Respir Crit Care Med 161: A615, 2000. 30 day old rhesus monkeys exposed to filtered air or ozone for 11 episodes, 5 days each, 0.5 ppm, 8 hr/day, followed by 9 days filtered air; sacrificed during the last fresh air exposure. Details of preparation of lung given. Findings were:
1. ozone decreased the number of branchings to the most proximal respiratory bronchiole (from 13 to about 9):
2. increased density and distribution of goblet cells:
Authors conclude: "We conclude that the periodic cycles of ozone exposure alters postnatal lung morphogenesis and epithelial differentiation in the distal lung of infant primates."

55. Kunzli, N., Lurmann, F., Segal, M., Ngo, L., Balmes, J., Tager, I.B. Association between lifetime ambientozone exposure and pulmonary function in college freshmen--Results of a pilot study. Environ. Res. 1997; 72:8–16.

56. Galizia, A., Kinney, P.L. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. Environ. Health Perspect 1999; 107:675–9.

57. McCONNELL, R., BERHANE, K., GILLILAND, F., LONDON, S.J., ISLAM, T., GAUDERMAN, W.J., AVOL, E., MARGOLIS, H.G., & PETERS, J.M.
Asthma in exercising children exposed to ozone: a cohort study
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Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

58. McCconnell, R., Berhane, K., Gilliland, F., London, S.J., Islam, T., Gauderman, W.J., Avol, E., Margolis, H.G., & Peters, J.M. Asthma in exercising children exposed to ozone: a cohort study Lancet 2002; 359; 386–391. From the Southern California Children's Study. Relevant numbers; 5762 children completed baseline questionnaires; 479 excluded because they were not at school when the questionnaire was administered; 883 excluded for a history of asthma; 312 excluded because of missing answers to "wheezing" questions; 26 excluded for chest illnesses such as cystic fibrosis: 527 excluded because they had less than one year of follow-up. This left 3535 children with no initial history of asthma; 2752 of these had no history of wheezing; 1934 played sports; 273 played three or more team sports. There were 46 low pollution communities (O_3 daytime mean 40.0 ppb); and 46 high pollution communities (O_3 mean 59.6 ppb). PM_{10} twice as high in high ozone communities (43.3 vs 21.6) and $PM_{2.5}$ three times higher (21.4 vs 7.6). NO_2 three times higher in high ozone communities (29.2 vs 10.8 ppb).
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Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

59. Agriculture and Agri-food Canada, Measuring plant response to ozone, http://www.agr.gc.ca/nlwis-snite/index_e.cfm?s1=pub&s2=ha_sa&page=73.

60. Reilly, S. et. al. Global economic effects of changes in crops, pasture, and forests due to changing climate, carbon dioxide, and ozone. Energy Policy 35 (11) 5370–5383 doi:10.1016/j.enpol.2006.01.040 (November 2007).

61. Sitch, S. Et. Al. “Indirect radiative forcing of climate change through ozone effects on the land-carbon sink. Nature 448, 791–794 (16 August 2007) | doi:10.1038/nature06059.
Tropospheric ozone is known to damage plants, reducing plant primary productivity and crop yields, yet increasing atmospheric carbon dioxide concentrations are thought to stimulate plant primary productivity. Increased carbon dioxide and ozone levels can both lead to stomatal closure, reducing the uptake of either gas, and in turn limiting the damaging effect of ozone and the carbon dioxide fertilization of photosynthesis. Researchers estimated the impact of projected changes in ozone levels on the land-carbon sink using a global land carbon cycle model modified to include the effect of ozone deposition on photosynthesis and to account for interactions between ozone and carbon dioxide through stomatal closure. They found a “significant suppression” of the global land-carbon sink as increases in ozone concentrations affect plant productivity. In consequence, more carbon dioxide accumulates in the atmosphere. They suggest that the resulting indirect radiative forcing by ozone effects on plants could contribute more to global warming than the direct radiative forcing due to tropospheric ozone increases.

62. Juliet Eilperin, “Air Pollution Impedes Bees' Ability to Find Flowers,” The Washington Post, May 5, 2008; P. A3.

63. Fuglestvedt, J.S. Climatic forcing of nitrogen oxides through changes in tropospheric ozone and methane; global 3D model studies. Atmospheric Environment,Volume 33, Issue 6, March 1999, Pages 961–977.
A three-dimensional global chemical tracer model and a radiation transfer model were used to study the role of NO_X emissions in global warming. Through production of tropospheric ozone, NO_X emissions lead to positive radiative forcing and warming. But by affecting the concentration of OH radicals, NO_X also reduces the levels of CH_4, thereby giving negative forcing and cooling. The lifetime of NO_X varies from hours to days, giving large spatial variations in the levels of NO_X. Geographical regions representing different chemical and physical conditions were selected to project chemical and radiative effects of reducing NO_X emissions by 20% in each region. Due to nonlinearities in the O_3 chemistry as well as differences in convective activity, there are large geographical differences in the effect of NO_X on O_3 as well as variations in the annual profile of the changes. The effect of NO_X emissions on methane is also found to depend on the localization of the emissions. The calculated ozone and methane forcing are of similar magnitude but of opposite sign. The methane effect acts on a global scale with a delay of approximately a decade, while the ozone effect is of regional character and occurs during weeks.

64. Fuglestvedt, J.S. Climatic forcing of nitrogen oxides through changes in tropospheric ozone and methane; global 3D model studies. Atmospheric Environment,Volume 33, Issue 6, March 1999, Pages 961–977.
A three-dimensional global chemical tracer model and a radiation transfer model were used to study the role of NO_X emissions in global warming. Through production of tropospheric ozone, NO_X emissions lead to positive radiative forcing and warming. But by affecting the concentration of OH radicals, NO_X also reduces the levels of CH_4, thereby giving negative forcing and cooling. The lifetime of NO_X varies from hours to days, giving large spatial variations in the levels of NO_X. Geographical regions representing different chemical and physical conditions were selected to project chemical and radiative effects of reducing NO_X emissions by 20% in each region. Due to nonlinearities in the O_3 chemistry as well as differences in convective activity, there are large geographical differences in the effect of NO_X on O_3 as well as variations in the annual profile of the changes. The effect of NO_X emissions on methane is also found to depend on the localization of the emissions. The calculated ozone and methane forcing are of similar magnitude but of opposite sign. The methane effect acts on a global scale with a delay of approximately a decade, while the ozone effect is of regional character and occurs during weeks.

65. Houghton, J. T. et al. (eds) Climate Change 2007: The Science of Climate Change (Cambridge Univ. Press, 2007).

66. Shindell, D., et. Al. Role of tropospheric ozone increases in 20^{th}-century climate change. J. Geophys. Res., 111, D08302, doi:10.1029/2005JD006348.
To simulate the warming effect of ozone, researchers employed the NASA Goddard Institute for Space Studies (GISS) chemistry model, using the spatial and temporal distribution of precursor emissions of tropospheric ozone from 1890 to 1990, finding that tropospheric ozone has contributed to the greater 20th-century warming in the Northern Hemisphere extratropics compared with the tropics and in the tropics compared with the Southern

Hemisphere extratropics. Additionally, ozone increased more rapidly during the latter half of the century than the former, causing more rapid warming during that time. Other climate forcings do not substantially accelerate warming rates in the tropics relative to other regions, suggesting that tropospheric ozone increases related to industrialization in the developing world have contributed to the accelerated tropical warming. During boreal, or northern, summer, tropospheric ozone causes enhanced warming (>0.5˚C) over polluted northern continental regions. Finally, the Arctic climate response to tropospheric ozone increases is large during fall, winter, and spring when ozone's lifetime is comparatively long and pollution transported from midlatitudes is abundant. The model indicates that tropospheric ozone could have contributed about 0.3˚C annual average and about 0.4˚ C–0.5˚ C during winter and spring to the 20th-century Arctic warming. According to the authors, "pollution controls could thus substantially reduce the rapid rate of Arctic warming."

67. Matthes, S. et. al. Global impact of road traffic emissions on tropospheric ozone. Atmos. Chem. Phys., 7, 1707–1718, 2007.
Road traffic is one of the major anthropogenic emission sectors for NO_X , CO and NMHCs (non-methane hydrocarbons). To assess the global impact of 1990 road traffic emissions on the atmosphere, investigators applied ECHAM4/CBM, a general circulation model coupled to a chemistry module, which includes higher hydrocarbons. This improved on previous global modeling studies, which concentrated on road traffic NO_X and CO emissions only. Including NMHC emissions from road traffic that NMHC emissions from road traffic play a key role for the impact on ozone. They are responsible for (indirect) long-range transport of NO_X from road traffic via the formation of PAN, which is not found in a simulation without NMHC emissions from road traffic. Long-range transport of NMHC induced PAN impacts on the ozone distribution in Northern Hemisphere regions far away from the sources, especially in arctic and remote maritime regions. In July total road traffic emissions (NO_X , CO and NMHCs) contribute to the zonally averaged ozone distribution by more than 12% near the surface in the Northern Hemisphere midlatitudes and arctic latitudes. In January, road traffic emissions contribute near the surface in northern and southern extratropics more than 8%. Sensitivity studies for regional emission show that effective transport of road traffic emissions occurs mainly in the free troposphere. In tropical latitudes of America up to an altitude of 200 hPa, global road traffic emissions contribute about 8% to the ozone concentration. In arctic latitudes NMHC emissions from road transport are responsible for about 90% of PAN increase from road transport, leading to a contribution to ozone concentrations of up to 15%.

68. Niemeir, U. et. al. Global impact of road traffic on atmospheric chemical composition and on ozone climate forcing. Journal of Geophysical Research, Vol. 111, D09301,doi:10.1029/2005JD006407, 2006.
Automobile emissions are known to contribute to local air pollution and to photochemical smog in urban areas. The impact of road traffic on the chemical composition of the troposphere at the global scale and on climate forcing is less well quantified. Calculations performed with the chemical transport MOZART-2 model show that the concentrations of ozone and its precursors (NO_X, CO,and hydrocarbons) are considerably enhanced in most regions of the Northern Hemisphere in response to current surface traffic. During summertime in the Northern Hemisphere, road traffic has increased the zonally averaged ozone concentration by more than 10% in the boundary layer and in the extratropics by approximately 6% at 500 hPa and 2.5% at 300 hPa. The summertime surface ozone concentrations have increased by typically 1–5 ppbv in the remote regions and by 5–20 ppbv in industrialized regions of the Northern Hemisphere. The corresponding ozone-related radiative forcing is 0.05 Wm-2. In order to assess the sensitivity of potential changes in road traffic intensity, two additional model cases were considered, in which traffic-related emissions in all regions of the world were assumed to be on a per capita basis the same as in Europe and in the United States, respectively. In the second and most dramatic case, the surface ozone concentration increases by 30–50 ppbv (50–100%) in south Asia as compared to the present situation. Under this assumption, the global radiative forcing due to traffic-generated ozone reaches 0.27 Wm-2.

69. Houghton, J. T. et al. (eds) Climate Change 2007: The Science of Climate Change (Cambridge Univ. Press, 2007).

70. de F. Forster, P.M. et al. Further Estimates of Radiative Forcing Due to Tropospheric Ozone Changes. Geophys. Res. Lett., 23(23), 3321–3324, Nov. 15, 1996.
Estimates made by two 2-D (latitude-height) chemical transport models show large uncertainty, but continue to support the case that tropospheric ozone changes make a substantial contribution (about 15%) to the total greenhouse gas radiative forcing.

71. Volz, A. & Kley, D. Evaluation of the Montsouris series of ozone measurements made in the nineteenth century. Nature 332, 240–242 (17 March 1988); doi:10.1038/332240a0
Questions regarding pre-industrial or 'background' ozone concentrations have led to the search for data from the

early days of ozone monitoring, during the second half of the last century. Unfortunately, most measurements were then made using Schönbein test paper, giving only semi-quantitative information due to poor standardization and the influence of humidity and wind speed on its sensitivity. Volz and Kley reinvestigated a set of ozone measurements gathered at the Observatoire de Montsouris, located on the outskirts of Paris, where a quantitative method was established in 1876 and used continuously for 34 years. The evaluation of the technique, together with the analysis of nearly 3,000 of the original daily measurements that previously remained unnoticed in a statistical bulletin of the City of Paris, provides conclusive evidence that ozone levels in central Europe 100 years ago averaged 10 p.p.b. and exhibited a seasonal variation, with a maximum during the spring months. Comparisons with modern data show that ozone levels in rural areas have more than doubled over the past century and that the tropospheric ozone budget is now strongly influenced by photochemical production due to increased levels of NO_X.

72. Nolle, M. Et. Al. A study of historical surface ozone measurements (1884–1900) on the island of Gozo in the central Mediterranean. Atmospheric Environment Volume 39, Issue 30, September 2005, Pages 5608–5618.
Surface ozone measurements using the Schönbein method were made from 1884–1900 on the rural island of Gozo (Malta). To assess the relative seasonal changes of the ozone concentration researchers, in combination with historical meteorological measurements (temperature, relative humidity, wind speed and direction), employed a new approach to humidity correction of the historical Schönbein measurements using boundary-layer considerations of the old and a nearby modern measuring site. The humidity-corrected Schönbein measurements from Gozo indicate—contrary to current ozone concentrations on Gozo—a clear annual ozone minimum in the summer. Average ozone-mixing ratios in the central Mediterranean could have increased by a factor of five since that time. However, due to the shortcomings of the Schönbein method, quantitative conclusions must be interpreted with care.

73. Schmidt, M. Evidence of a 50-year increase in tropospheric ozone in Upper Bavaria. Ann. Geophys., 12, 1197–1206, 1994
In a series of ozone-sonde soundings at the Hohenpeißenberg observatory, starting in 1967, the most striking features are increases of \sim2.2% per year in all tropospheric heights up to 8 km during the past 24 years. These facts have recently been published and discussed by several authors. In this paper, we present some evidence for the increase of tropospheric ozone concentrations during the past 50 years 1940–1990 in the territory of the northern edge of the Bavarian Alps, including the Hohenpeißenberg data. In December 1940 and August 1942, probably the first exact wet-chemical vertical soundings of ozone up to 9 km height were made by an aircraft in the region mentioned. These results were published in the earlier literature. We have converted the results of the flights on 4 days in December 1940 and on 6 days in August 1942 to modern units and have compared them with the Hohenpeißenberg ozone-sonde data of the December and August months. We also compared the data at the ground with the August results of Paris-Montsouris 1886–1898. Our results show an increase of ozone concentration at all tropospheric heights in Upper Bavaria during the past 50 years, compared with the Montsouris data in August during the past 105 years. In the recently published papers, the increases since 1967 were approximated linearly. Our results, extended to the past, show non-linear trends, with steeper increases since 1975–1979. Possible reasons for these findings are discussed. Quite recently (in case of the December months since 1986/87, the August months since 1990), the ozone mixing ratios at and above Hohenpeißenberg seem to have decreased.

74. Naja, M. Changes in Surface Ozone Amount and Its Diurnal and Seasonal Patterns, from 1954–55 to 1991–93, Measured at Ahmedabad, India. S. Lal, Geophys. Res. Lett., 23(1), 81–84, Jan. 1, 1996.
Despite the crucial role of ozone as a greenhouse gas and in the production of OH radicals, there are few systematic, long-term measurements in the tropics. The measurements presented here show a linear increase of 1.45% per year in average ozone between the two periods analyzed; background concentrations increased by 0.49% per year.

75. Jiang, Y. & Yung, Y.L. Concentrations of Tropospheric Ozone from 1979 to 1992 over Tropical Pacific South America from TOMS Data. Science, 272(5262), 714–716, May 3, 1996.

Satellite measurements indicate that tropospheric ozone increased by 1.48 ± 0.40 percent per year or 0.21 ± 0.06 Dobson unit over South America and the surrounding oceans. An increase in biomass burning in the Southern Hemisphere can account for this trend.

76. Hough, A.M & Derwent, R.G. Changes in the global concentration of tropospheric ozone due to human activities. Nature 344, 645–648 (12 April 1990); doi:10.1038/344645a0.
Evidence from records of ground-level measurements demonstrate that the average tropospheric concentration of ozone in the Northern Hemisphere has increased. In particular, the comparison of recent observations with those made at the Montsouris laboratory in Paris between 1876 and 1910, suggests that the surface concentration of ozone at mid to high latitudes has more than doubled in the past 100 years. This has potential implications for a wide range

of environmental issues both because of the direct effects of elevated concentrations of ozone on man and ecosystems and because ozone is a radiatively active gas which could contribute significantly to global warming if its concentration were to increase. Used a global tropospheric model to simulate the chemistry of the pre-industrial atmosphere and that of the present day. The model results for surface ozone concentrations in the pre-industrial atmosphere agree well with the Montsouris data, and the calculated concentrations for the present day agree with recent observations of a wide range of chemical species. Estimates of the future growth in emissions of nitrogen oxides (NO_X) were used to make similar calculations for the year 2020. On the basis of these estimates, the global tropospheric concentration of ozone will continue to increase at a rate faster than during the past 100 years. The potential for further increases in tropospheric ozone needs to be taken into account when assessing the impact of air pollution emissions and the adequacy of measures to control them.