Possible Consequences of Nearby Supernova Explosions for Atmospheric Ozone and Terrestrial Life

Abstract. *Hard x-ray pulses or increased cosmic radiation originating in nearby supernova explosions may be capable of temporarily removing most of the earth's atmospheric ozone cover even when direct radiation effects at the earth's surface are negligible. Consequently, terrestrial life may be subject to relatively huge solar ultraviolet fluxes every few hundred million years.*

I present here a possible mechanism for nearby supernova explosions affecting terrestrial life based upon the following circumstances. Factor A: A very small amount of O_3 [equivalent to a layer only 3 to 4 mm thick at normal temperature and pressure (NTP), that is, 20°C and 1 atm] shields the earth's surface from intense solar ultraviolet radiation. Factor B: Small quantities of stratospheric nitric oxides (mainly NO or NO_2) may catalytically destroy significant amounts of O_3; NO_x concentrations of only a few parts per billion (ppb) may reduce the total O_3 column abundances by a factor of 2 or so (*1*, *2*). Large quantities of NO can be produced in the stratosphere by hot air from jet engines, nuclear explosions, or ionizing radiation. Factor C: Nearby supernova explosions may enormously increase stratospheric ionizing radiation when supernova-produced cosmic rays or hard x-rays reach the earth. The amount of radiation needed to produce enough NO to reduce the O_3 abundance significantly and to allow solar ultraviolet light to reach the earth's surface is estimated to be very much less than that proposed as affecting terrestrial life directly by increased radiation doses (*3*). Factor D: Various forms of life (especially DNA) are especially sensitive to strong ultraviolet radiation ($<$ 3200 Å) even at depths of a few meters of water (*4*, *5*). I elaborate below on each of these main points.

Factor A. Nitric oxides can catalytically destroy O_3, primarily through the reactions

$$NO + O_3 \rightarrow NO_2 + O_2$$

followed by

$$NO_2 + O \rightarrow NO + O_2$$

Johnston (*1*) and Crutzen (*2*) have suggested that NO_x in the stratosphere accounts for the failure of the observed O_3 to reach the abundance calculated from the well-known Chapman cycle describing the various interactions among O_2, O, O_3, and ultraviolet light (*6*). A relative NO_x abundance of the order of a few parts per billion would seem sufficient to explain the reduction by a factor of 2 or so from the calculated O_3 abundance.

There is some direct supportive evidence that stratospheric NO_x is a dominant factor in controlling the O_3 concentration: (i) Concentrations of NO plus NO_2 above 20 km appear to be of the right order of magnitude. (ii) Nuclear explosions should have dumped huge amounts of NO into the stratosphere (*7*). The 6 to 10 percent rise in the O_3 abundance in the Northern Hemisphere in the decade since the end of nuclear testing in 1962 has been attributed to NO_x "diffusing" out of the stratosphere (*8*) (where its residence time is 2 to 6 years). However, there is disagreement on whether the expected O_3 decrease during 1961–1962 was detected (*9*). (iii) It has been claimed that the effects of the relatively small amount of varying stratospheric NO associated with cosmic-ray modulation during 11-year sunspot cycles give rough agreement with long time-scale O_3 behavior (*10*).

The sensitivity of the O_3 abundance to additional NO_x is not well known. For altitude-independent mixing ratio models an idealization of the chemical kinetics which also gives a rough fit to numerical calculations is

$$2[O_3]/[O_3]_0 = (16 + 9X^2)^{1/2} - 3X$$

where X is the ratio of $[NO_x]$ to present ambient $[NO_x]_0$ and $[O_3]/[O_3]_0$ is the ratio of the associated O_3 abundances. In this dependence of the O_3 ratio on X it is assumed that: (i) the oxidation of NO_2 by O gives the limiting reaction rate for the cycle of catalytic O_3 destruction by NO_x, (ii) the ratio $[O]/[O_3]$ is a constant, and (iii) the present $[NO_x]_0$ reduces $[O_3]$ to one-half the value it would have had without any NO_x present.

Factor B. Ionizing radiation has been estimated to produce free N atoms at a rate greater than one N atom per ion pair (*11*); slightly more than half of these atoms (N*) are in excited metastable states ^2D (2.37 ev) and ^2P (3.56 ev). Almost all N* produces NO via

$$N^* + O_2 \rightarrow NO + O$$

before quenching. At the low stratospheric temperatures (~ 230°K) this reaction with ground state N is suppressed sufficiently so that the destructive reaction

$$N + NO \rightarrow N_2 + O$$

proceeds at about the same rate as the reaction between N and O_2 when [NO] ~ 10 ppb. Therefore, the net NO production per ion pair in air in which all NO_x is NO is about $10(10 + y)^{-1}$, where y is the NO abundance in parts per billion. If, on the other hand, NO_x is mainly NO_2, we have the reactions:

$$N + NO_2 \rightarrow N_2 + O_2$$

$$N + NO_2 \rightarrow N_2O + O$$

$$N + NO_2 \rightarrow N_2 + O + O$$

or

$$N + NO_2 \rightarrow NO + NO$$

The reaction rates are such that the net number of NO molecules formed per ion pair in air at 230°K with NO_2 as the main ambient oxide of nitrogen is approximately $(80 + y')(60 + 3y')^{-1}$, where y' is the NO_2 abundance in parts per billion. In our present stratosphere the NO_x abundance seems less than 10 ppb so that ionizing radiation would be expected to produce about one NO molecule per ion pair. But in the presence of ionization sufficient to increase NO_x greatly, the efficiency for NO production will drop in a way which depends upon the NO_x species present.

Factor C. At stratospheric altitudes above 15 km and latitudes above 60°, normal cosmic-ray ionization produces about 3×10^7 NO molecule cm^{-2} sec^{-1} and an average of 10^7 NO molecule cm^{-2} sec^{-1} after lateral diffusion of the molecules over the whole hemisphere in a time period on the order of a year (*12*). The total stratospheric NO_x input from other sources is not well known: estimates generally range from 10^{15} to 10^{16} NO molecule cm^{-2} $year^{-1}$. Therefore, present cosmic rays are expected to contribute between 3 and 30 percent of the total stratospheric NO. This incident cosmic-ray flux is 9×10^4 erg cm^{-2} $year^{-1}$ at the top of the atmosphere and gives a radiation dose of 30×10^{-3} roentgen at the bottom of the atmosphere. The cosmic-ray flux would be significantly increased by a substantial diminution of the earth's magnetic field during periods of reversing field and, probably to a much greater extent, by nearby supernovae (*13*).

During the history of life on earth supernova explosions within 50 light-

years probably occurred at intervals of a few hundred million years or less (*3, 13*). It is thought probable that some, and perhaps all, galactic cosmic rays originate in such explosions. Ionizing radiation can be deposited by them in our stratosphere in two ways: (i) by x-rays and γ-rays emitted at about the time of the explosion and (ii) by greatly increased cosmic-ray densities streaming along tangled magnetic field lines between the solar neighborhood and the supernova. Tucker and Terry (*14*) considered 10^{48} to 10^{50} ergs as a reasonable assumption for the γ-ray pulse emitted in a supernova explosion. Colgate (*15*) has calculated a flux of 5×10^{47} ergs. If, as has been widely suggested, the isotropic hard x-ray bursts (100 kev to 1 Mev) first detected by the Vela satellite system (*16*) are indeed from extragalactic supernovae, the energy burst (10^{-4} erg cm^{-2}) implies 10^{47} ergs of such hard x-rays emitted from a typical supernova 10^{7} light-years away. At normal incidence, most of these x-rays will penetrate our atmosphere to an altitude of 27 to 32 km before Compton scattering begins to convert their energy to that of ionizing electrons. At a distance of 50 light-years, 10^{47} ergs of this radiation will produce an average stratospheric column ionization of about 10^{17} ion pairs per square centimeter over a hemisphere; this is over 300 times more ionization than caused by normal cosmic rays in an entire year. The number of stratospheric NO molecules formed as a by-product of this ionization is, according to section B, $10^{18}(10 + \bar{y})^{-1}$, where $\bar{y}$ is essentially the new average mole fraction of ionization. If we assume that this ionization is distributed in proportion to the stratospheric density among the 5×10^{23} molecule cm^{-2} between altitudes of 25 and 35 km, then 2×10^{16} NO molecule cm^{-2} are formed there. This is about seven times greater than a typical estimate of the present stratospheric concentration of NO_x (3 ppb) between 20 and 35 km. With this NO_x abundance the relationship of section A gives $[O_3]/[O_3]_0 \sim 0.2$, an extremely rough estimate. The same x-ray burst at 15 light-years or a 10^{48}-erg burst at 50 light-years would give three times as much NO and leave perhaps 6 percent of the normal O_3 during the expected minimum 2-year residence time for NO in the stratosphere. Despite their effect in causing a large ozone suppression, the incident hard x-ray bursts give no significant surface radiation doses.

Typical estimates of the cosmic-ray particle production in a supernova explosion range from 10^{50} to 10^{51} ergs. If these particles stream out along tangled interstellar magnetic field lines, without important energy loss, then at distances of 50 light-years or less their average flux is enormously greater than that of the present cosmic-ray flux. Laster (*17*) has estimated that, at a distance d, streaming cosmic rays would be spread over an interval $\tau = d^2(c\lambda)^{-1}$, where λ is ~ 3 light-years and c is the velocity of light. At 50 light-years a 10^{50}-erg cosmic-ray burst would then give 40 times the present cosmic-ray flux for 80 years. (The corresponding radiation dose at the earth's surface would be 1 roentgen per year.) If the explosion yields 10^{51} ergs of cosmic rays, or if the distance is 30 light-years, the cosmic-ray flux is about 400 times its present value.

A hundredfold increase in cosmic-ray flux would produce 3×10^{16} ion pairs per square centimeter per year in the stratosphere. Because the field line path from a given supernova to the earth is so complicated and may not always exist, a large variation in this flux would be expected even for identical explosions and distances. Much of the NO produced by this flux is continually converted to NO_2, and the efficiency for the production of NO per ion pair is larger than if all the annual production were to occur within a few seconds, as is expected for the hard x-ray component. When the uncertain NO production efficiency is coupled with the magnitude variations in estimates both of the present stratospheric NO_x concentration and the cosmic-ray yield from supernovae, the increase in the stratospheric NO abundance from a supernova at 50 light-years may range anywhere from a factor of 2 to a factor of 50. From section A the corresponding O_3 abundance would be reduced to between 60 and 3 percent of the normal value for almost a century. Closer supernovae would, of course, greatly lower these values for shorter periods.

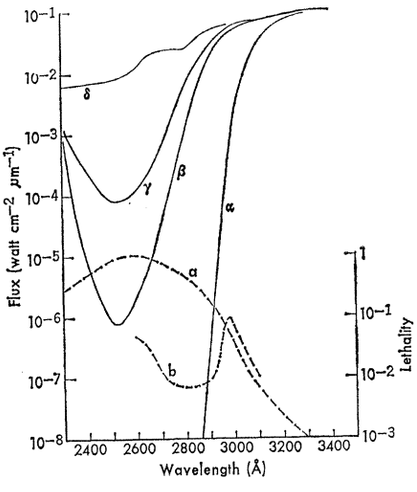

Fig. 1. Noontime incident solar ultraviolet flux on the earth: curve α, ultraviolet flux after passage through a typical stratospheric O_3 layer equivalent to 3 mm at NTP; curve β, flux if 10 percent O_3 (equivalent to 0.3 mm O_3 at NTP) survives; curve γ, flux at the equator if only 6 percent of the O_3 is left (equivalent to 0.15 mm O_3 at NTP); curve δ, incident flux at the top of the atmosphere; curve a, relative effectiveness of the ultraviolet flux at various wavelengths in killing bacterial cells (*Escherichia coli*) (*4, 5*); and curve b, relative effectiveness of the ultraviolet flux in producing erythema (sunburn) (*4*).

Unfortunately, partly because the extent of the presumed suppression of O_3 by NO_x is not yet fully and reliably calculable and even more because of uncertainties in the parameters of supernova explosions, a more quantitative calculation of terrestrial O_3 suppression by nearby supernovae is not yet possible. Moreover, a large alteration in the photochemistry of our atmosphere may amplify other contributions to ultraviolet absorption in a manner not yet understood. However, it does appear plausible that a few times in the history of life on earth more than 90 percent of our O_3 umbrella may have disappeared for periods of a few years to a century. (The incident supernova energy flux required to accomplish this need be only equivalent to that of less than a minute of added sunshine over a year.) Such a removal of our O_3 umbrella would permit the ultraviolet solar flux to reach the values shown in Fig. 1.

Factor D. The ultimate biological consequences for most terrestrial life in the presence of such a huge increase in ultraviolet radiation (< 3100 Å) (possibly lasting for many generations), is unknown (*5*). In man it would certainly give rise to a vast increase in the incidence of skin carcinoma. In animals with calcified internal skeletons the increased vitamin D production might be toxic. The death rate for exposed cells and the mutation rate would grow enormously. The efficiency for photons causing cell deaths is large only in the ultraviolet (Fig. 1): radiation of 2900 Å is 10^2 times more effective than radiation at 3250 Å and 10^4 times more effective than radiation

at 5500 Å in killing bacteria. This lethal efficiency approximates that for absorption by DNA. Airborne viruses or those carried by small insects may be greatly altered by ultraviolet radiation. Host populations for these viruses could be greatly affected either by the production of numerous new mutants or by the return of such viruses after many generations of suppression by the intense ultraviolet radiation. Water, per se, is not a particularly good shield against that part of ultraviolet spectrum involved (in pure water the absorption coefficient at 2900 Å is only 7×10^{-3} cm^{-1}) (*4*). Since a high proportion of animal species is nocturnal, the effects of increased solar ultraviolet flux might be manifest in a differential depletion of diurnal species. There seems to be no compelling fossil evidence for past "biological cataclysms" (*18*), but the effects of ultraviolet deluges on past and future evolution may be more subtle.

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Nicotine Inhibition of the Metabolism of 3,4-Benzopyrene, a Carcinogen in Tobacco Smoke

Abstract. *A decreased rate of biliary excretion of radioactive metabolites of 3,4-benzopyrene was observed in rats given a single dose of nicotine. Prior treatment of rats with nicotine decreased benzopyrene hydroxylase activity in homogenates of liver, lung, and small intestine. The addition of nicotine to incubated tissues also decreased benzopyrene hydroxylase activity. These findings show that nicotine inhibits the metabolism of 3,4-benzopyrene in vivo and in vitro.*

In tobacco the alkaloid nicotine shares a physical and possibly a metabolic relation to the carcinogenic polycyclic hydrocarbon 3,4-benzopyrene (BP). Nicotine and BP are both constituents of inhaled tobacco smoke (*1*), and nicotine is reported to alter the activity of hepatic microsomal enzymes that metabolize many foreign chemicals including drugs and carcinogens (*2–4*). Thus, nicotine may also influence microsomal enzyme systems required for the metabolism of BP. Although BP is a potent carcinogen in tobacco smoke (*5*), information is limited concerning its metabolic interactions with nicotine. Only a single report was found showing that a large, single dose of nicotine (40 mg/kg) given intraperitoneally enhanced BP hydroxylase activity in rat liver microsomes measured in vitro 24 hours after treatment with nicotine (*3*).

When BP is metabolized by hepatic enzymes the end products are hydroxylated metabolites (*6*). An active epoxide intermediate, however, is also formed (*7*); it binds readily to DNA (*8*) and is implicated in the formation of tumors at target organs. Thus, an important aspect of carcinogenesis is the formation of a reactive epoxide of BP, the concentration of which might be influenced by other chemical agents: for example, nicotine, which may affect the microsomal enzyme activity required for the metabolism of BP. The accumulation of evidence that heavy, prolonged cigarette smoking in humans plays a role in the etiology of cancer of the lung emphasizes the need for more extensive studies of the metabolic interactions between a known carcinogen and other chemical agents in tobacco smoke.

We investigated the effects of a single dose of nicotine on hepatic and nonhepatic metabolism of the carcinogen BP in vivo and in vitro. Since BP is metabolized by hepatic microsomes and appears in the bile almost entirely as metabolic products (*9*), we first studied the effects of nicotine on the biliary excretion of BP metabolites. Sprague-Dawley male rats (220 ± 10 g) were used and treated with a single dose of nicotine bitartrate (25 mg/kg; nicotine base, 8.13 mg/kg) administered intraperitoneally in a volume of 0.5 ml per 100 g of body weight. The corresponding controls were given an equivalent amount of saline. The animals were anesthetized with ether 24 hours after the preliminary nicotine treatment, and the common bile duct was cannulated (*10*). Bile was collected in tared scintillation vials during a 15-minute control period. In the standard procedure, BP (2.5 mg/kg) containing tracer quantities of ^{14}C-labeled BP (0.8 μc/kg) was injected into the right femoral vein after the control period, after which bile samples were collected for six additional 15-minute periods. The ^{14}C in the bile (20 μl) samples was counted in a Packard Tri-Carb liquid scintillation counter with a Triton fluor (*11*).

For the metabolism of BP in vitro in a separate experiment rats were killed 24 hours after treatment with nicotine or saline, and homogenates of liver, lung, and small intestine were made (3 percent in ice-cold 0.25*M* sucrose). Tissue BP hydroxylase activity was determined by measuring the fluorescence of hydroxylated metabolites of BP (*12*). The homogenates (0.5 ml) of liver,

Table 1. Effect of treatment with nicotine on tissue BP hydroxylase activity. Rats were given nicotine bitartrate (25 mg/kg) intraperitoneally, and the enzyme activity of each tissue was determined 24 hours later. The final incubation mixture had a volume of 3.1 ml which was composed of: 0.5 ml of glucose-6-phosphate dehydrogenase (5 Kornberg units), 0.2 ml of 0.03*M* glucose 6-phosphate, 0.1 ml of nicotinamide adenine dinucleotide phosphate solution (4 mg/ml), 0.1 ml of nicotinamide adenine dinucleotide solution (4 mg/ml), 0.2 ml of 0.01*M* adenosine triphosphate, 0.2 ml of 0.6*M* nicotinamide, 0.1 ml of 2.0*M* KCl, 0.1 ml of 0.1*M* $MgCl_2$, 0.1*M* KH_2PO_4–K_2HPO_4 buffer at *p*H 7.4, 0.5 ml of tissue homogenate, and 0.1 ml of BP (50 μg). The results are expressed as nanograms of 3-hydroxybenzopyrene per milligram of tissue per 20 minutes. Each value represents the mean ± S.E. of data from five or six rats.

Treatment	BP hydroxylase activity*		
	Lung	Liver	Small intestine
None	14.55 ± 1.3	2.64 ± .42	0.92 ± .25
Nicotine*	8.65 ± 0.7	1.46 ± .15	0.25 ± .03

* Nicotine produced a significant reduction in all three tissues ($P < .05$).