ed a much lower r^2 (.25). Multiple regressions of H^+ versus excess SO_4^{2-} and other parameters taken one at a time (for example, NO_3^- or NH_4^+) did not improve the correlation, but regression of H^+ versus excess SO_4^{2-} and Ca^{2+} yielded the equation

$$[H^+] = 6.1 + 0.54[SO_4^{2-}]_{xs} - 0.35[Ca^{2+}]_{xs}$$

$R^2 = .75$, where the subscript xs stands for nonmarine-derived material. This two-variable model thus implies that the acidity of Florida rainfall can be accounted for in large part in terms of the interaction between H_2SO_4 and terrestrially derived basic calcium salts.

Historical *p*H data are lacking for Florida rainfall. However, *p*H values calculated by the ionic balance method (*2*) from Junge's data (*9*) for five locations in Florida indicate that rainfall at these sites had *p*H values above 5.6 in the mid-1950's (Table 1). Large increases have occurred in the concentrations of excess SO_4^{2-} and especially of NO_3^- in Florida rainfall since the mid-1950's. The average increase in the sum of these two ions over the state (23.5 μeq/liter) is more than adequate to account for the average increase in H^+ (14.8 to 17.3 μeq/liter). Moreover, present deposition values for SO_4^{2-} are up to four times higher (Fig. 2B) than values obtained at several sites in the early 1950's (*10*). The largest increases in the deposition of SO_4^{2-} have occurred in the northern part of the state. If fluxes of sea sulfur and biogenic sulfur have remained constant, the differences represent a substantial increase in anthropogenic excess SO_4^{2-}.

Although the degree of acidity in Florida rainfall is not as great as that presently occurring in the northeastern United States, our findings are of serious concern for several reasons. Because Florida receives 50 to 75 percent more rainfall annually than most of the Northeast, the differences in the annual deposition of H^+ and SO_4^{2-} are smaller than the *p*H-concentration data suggest. For example, 206 cm of rainfall resulted in the deposition of 500 eq of H^+ per hectare and 11.5 kg of sulfur per hectare at Jay in the period May 1978 to April 1979; analogous values for the same period at Lake Apopka are 136 cm of rain, 342 eq of H^+ per hectare, and 6.8 kg of sulfur per hectare. In comparison, precipitation at Hubbard Brook, New Hampshire, deposited an average of 970 eq of H^+ per hectare (*4*) and 12.7 kg of sulfur per hectare (*11*) over the 10-year period 1964–1965 to 1973–1974. Thus northern Florida receives about one third to one half the H^+ deposition and 50 to 90 percent of the excess SO_4^{2-} deposition of the heavily impacted Northeast.

Florida's highly weathered, sandy soils characteristically have low cation-exchange capacities (*12*). Thus soils in Florida are unable to provide much buffering capacity. Numerous soft-water lakes occur in the sandhill region of north and central Florida, and these lakes obtain most of their water directly from rainfall and from subsurface seepage through the sandy soils. Annual average *p*H values in a group of 12 such lakes (Trail Ridge lakes) located about 40 km east of Gainesville (Fig. 1A) are now 0.3 to 0.9 unit lower than the average *p*H values measured in the late 1950's and late 1960's (*6*). For example, Lake Brooklyn (Clay County) had a *p*H of 5.5 ($N = 6$) during 1957–1960, a *p*H of 5.0 ($N = 6$) during 1967–1972, and a *p*H of 4.9 ($N = 7$) during 1977–1979. In comparison, no significant long-term trends in *p*H have been observed in a group of eight soft-water lakes in Highlands County, south central Florida (Fig. 1A), in an area near the present southern limit of acid precipitation.

Because Florida's population still is expanding rapidly, the demands for electric power are increasing more rapidly in Florida than in most other states. Like most of the rest of the United States, Florida is shifting to coal as the energy source for new electric power-generating capacity. At present, six coal-fired plants [total capacity, 2086 MW (electric)] are operating in peninsular Florida, but 15 additional plants with a generating capacity of 6884 MW (electric) are planned for this region by 1987 (*13*). The effects of the added emissions on the acidity of Florida rainfall, and the consequent effects on Florida's vulnerable aquatic and terrestrial ecosystems, should be carefully examined in the coming years.

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Nickel Carbonyl: Decomposition in Air and Related Kinetic Studies

Abstract. *Nickel carbonyl [$Ni(CO)_4$] is a toxic gas used in the manufacture of metallic nickel which has been shown to be carcinogenic and teratogenic in laboratory studies. Its decomposition in air proceeds at a rate that is strongly dependent on the concentration of carbon monoxide (CO). In the absence of CO, the lifetime in air at 296°K and at atmospheric pressure is 60 ± 5 seconds. A mechanism consisting of equilibrium unimolecular decomposition to $Ni(CO)_3$ and CO, followed by reaction of the $Ni(CO)_3$ with molecular oxygen, is consistent with the observations.*

Nickel carbonyl [$Ni(CO)_4$] is used industrially in the Mond process for the manufacture of nickel, in nickel plating, and as an industrial catalyst (*1*). Although highly toxic, its effects on man remain somewhat controversial (*1–3*).

A knowledge of reactions of gaseous $Ni(CO)_4$ with other species that may be

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Table 1. Lifetime studies data for $Ni(CO)_4$, showing the concentration ranges and the measurement temperatures. The intercepts and slopes are determined from plots of the lifetime versus the dependent variable ([CO] or $[O_2]^{-1}$). The top six rows report the results of varying [CO] with $[O_2]$ held constant; the bottom three rows show the data from varying $[O_2]$ with [CO] held constant. The temperature reported is the average measured during each set of readings.

[CO] (ppm)	$[O_2]$ (ppm)	$[O_2]^{-1}$ (ppm^{-1})	Intercept (sec)	Slope (sec ppm^{-1})	Slope (sec ppm)	Temperature (°C)
0.50 to 22.9	10^6	10^{-6}	68.4	9.46		23.2
1.05 to 24.3	10^6	10^{-6}	54.8	8.27		23.5
0.14 to 4.3	2.1×10^5	4.76×10^{-6}	71.9	52.5		21.9
0.28 to 5.3	2.1×10^5	4.76×10^{-6}	62.3	36.9		23.2
0.14 to 5.3	2.1×10^5	4.76×10^{-6}	52.7	36.2		23.7
0.28 to 5.5	2.1×10^5	4.76×10^{-6}	53.1	33.1		24.2
4.63	10^5 to 10^6	10^{-5} to 10^{-6}	56.7		3.92×10^7	22.6
4.44	10^5 to 10^6	10^{-5} to 10^{-6}	58.2		3.53×10^7	23.4
1.94	10^5 to 10^6	10^{-5} to 10^{-6}	62.7		1.22×10^7	23.8

present in air, along with their respective rate constants, is important in evaluating situations of possible human exposure to the carbonyl. Accordingly, we measured the decay rate of mixing ratios of 0 to 100 parts per billion of $Ni(CO)_4$ in air at room temperature, using a newly developed chemiluminescent method (*4, 5*).

Although $Ni(CO)_4$ decay in air had not previously been studied, thermal (*6*) and photochemical decomposition (*7*), nucleophilic attack, and the rate of CO exchange have been reported (*8*). The basic mechanism is known to start with the decomposition

$$Ni(CO)_4 \underset{k_2}{\overset{k_1}{\rightleftarrows}} Ni(CO)_3 + CO \qquad (1)$$

According to Callear (*9*), heterogeneous oxidation follows

$$Ni(CO)_3 + O_2 \xrightarrow{k_3}$$

$$Ni(CO)O + CO + CO_2 \qquad (2)$$

Taken together, these earlier data (*6–8*) would imply a lifetime for $Ni(CO)_4$ in air of about 1 minute provided no CO were present. Any CO would dramatically retard the decay. However, until this work was done, our rough observation of a lifetime of 1/2 hour was, as far as we know, the only reported value (*4*).

For this study, standard mixtures of gaseous $Ni(CO)_4$ were diluted initially in a 700-liter Tedlar bag and later in an atmospheric pressure flow system. For these systems, we measured the lifetimes as a function of both [CO] and $[O_2]$ at room temperature. Plots of the lifetime versus [CO] at constant $[O_2]$ or versus $[O_2]^{-1}$ at constant [CO] yielded well-defined straight lines in the concentration ranges 0.5 part per million (ppm) to 25 ppm for CO and 10 to 100 percent for O_2, with a constant intercept (Table 1).

The mechanism given above accounts perfectly for this behavior. Application of the steady-state approximation for $[Ni(CO)_3]$ leads to $Ni(CO)_4$ decay kinetics in terms of the lifetime (τ)

$$\tau = 1/k_1 + k_2[CO]/k_1k_3[O_2]$$

where $\tau = -(\partial \ln[Ni(CO)_4]/\partial t)^{-1}$ The observed lifetime is linearly dependent on [CO] with an inverse dependence on $[O_2]$ and an intercept (k_1^{-1}) which measures the initial rate of carbonyl unimolecular decomposition. We obtain a value for k_1^{-1} of 60 ± 5 seconds at $296.5° \pm 1°K$. This gives a rate constant k_1 of 0.017 ± 0.002 sec^{-1}, in good agreement with the observations of Day *et al.* (*8*), who obtained 0.018 sec^{-1} at 298°K. Using values for the thermodynamics of reaction 1 suggested by Day *et al.* (*8*), one can derive the rate constants k_2 and k_3 (*10*).

We have also analyzed the CO produced from the decomposition of $Ni(CO)_4$ at a concentration of 50 to 100 ppm in air at 25°C. Methanation gas chromatography (*11*) indicates that 3.9 ± 0.5 molecules of CO were produced per molecule of $Ni(CO)_4$ decomposed, with no observable CO_2 production (< 1 percent of the CO produced). Other studies have suggested different end products (*9*) but were carried out at much higher $Ni(CO)_4$ concentrations.

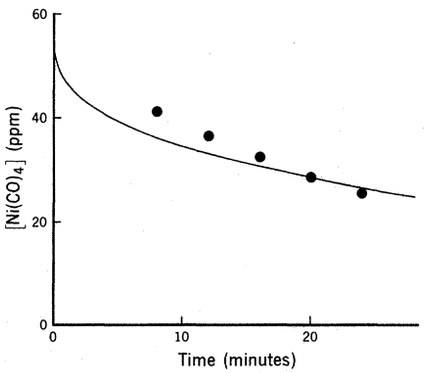

Fig. 1. Plot of $[Ni(CO)_4]$ versus time at a calculated initial $[Ni(CO)_4]$ of 52.5 ppm. The points are data obtained by syringe injection into a stirred 5-liter flask. The curve is the calculated decomposition for a homogeneous mixture of $Ni(CO)_4$ in air at the same initial $[Ni(CO)_4]$, based on the rate constants derived herein.

These results have a direct bearing on exposure to $Ni(CO)_4$ in air. In the absence of CO, part-per-million concentrations of $Ni(CO)_4$ will decay with a lifetime of about 1 minute, thus decreasing the risk of exposure to workers even in poorly ventilated areas. Since CO increases the lifetime by about a 1/2 minute for every part per million of added CO, the exposure hazard of $Ni(CO)_4$ in large excess of CO has to be treated more carefully, as does CO cylinder gas which contains at least part-per-million quantities of metal carbonyls (*12*).

The initial rapid decomposition of $Ni(CO)_4$ in an enclosed space such as that used by Sunderman *et al.* (*2*) in their biological studies introduces an extra variable. Calculations may be made on the basis of our measured constants, yielding plots of $[Ni(CO)_4]$ versus time for known initial $Ni(CO)_4$ concentrations in the range used in the biological studies. Such a plot (Fig. 1) shows a good agreement with our experimentally determined decay route and is consistent with the observations made by Sunderman *et al.* (*13*). Figure 1 demonstrates the decay-retarding effect of the initial CO released and shows that reaction products can quickly build up to a significant fraction of the initial concentration. These rapid increases could be an important factor in such biological studies.

It is possible that the pathological response arises from the dose of decay products, not the $Ni(CO)_4$. Experiments in which no decay products are present would resolve this possible question and might be particularly important in view of the controversy over the carcinogenic potential of $Ni(CO)_4$ in animals and in man.

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10. Using the thermodynamics from Day *et al.* (*8*), we obtain $k_{eq} = k_1/k_2 = 1.1 \times 10^{11}$ molecule cm^{-3}, with a probable uncertainty of about a factor of 2. Based on this, our value for the intercept k_1 (0.017 sec^{-1}) leads to a value for k_2 of 1.5×10^{-13} cm^3 $molecule^{-1}$ sec^{-1}, lower by a factor of about 7 than Callear's value (*9*). From the observed dependence on [CO] and $[O_2]^{-1}$, we obtain $k_2/k_3k_1 = 6.3 \pm 0.2 \times 10^6$ second, from which a value of 1.4×10^{-18} cm^3 $molecule^{-1}$ sec^{-1} can be derived for k_3. This is five orders of magnitude lower than Callear's value (*9*), which must be in error as a result of the complexity of the kinetic derivation in his flash photolysis system. The reproducibility and surface independence of our data imply that we are dealing with a purely homogeneous mechanism at these low concentrations.
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Aryl Hydrocarbon Hydroxylase Is Inhibited by Antibody to Rat Liver Cytochrome P-450

Abstract. *Antibody to the major purified cytochrome P-450 induced by 3-methylcholanthrene in rat liver strongly inhibits aryl hydrocarbon hydroxylase activity of uninduced and benz*[a]*anthracene-induced human monocytes and lymphocytes. Antibody to the cytochrome P-450 induced by phenobarbital has relatively little or no effect on the aryl hydrocarbon hydroxylase activity of the same human cells.*

Human monocytes and lymphocytes metabolize the carcinogenic polycyclic hydrocarbon benzo[*a*]pyrene (BP) (*1*). This activity is mediated by aryl hydrocarbon hydroxylase (AHH), a cytochrome P-450–dependent mixed function oxidase system in mammalian liver (*2, 3*), lung (*3, 4*), and other tissues (*3, 5*). The AHH activity is induced in mammalian liver by a wide variety of xenobiotics, including polycyclic hydrocarbons and phenobarbital. Induction in liver by polycyclic hydrocarbons results also in the appearance of a form of the cytochrome different from the major form present in the untreated animal. This new form of cytochrome P-450 exhibits different substrate specificity and has different susceptibility to the inhibitor 7,8-benzoflavone than the uninduced enzyme (*6*). Treatment with phenobarbital causes an increase in another form of cytochrome P-450 (*7*). Several of these cytochromes P-450 have been purified from rat (*8, 9*), rabbit (*10*), and mouse (*11*) liver. The AHH of human monocytes and lymphocytes is induced by polycyclic hydrocarbons in cell culture (*12*). However, since AHH in these cells is low and only very small amounts of cells from individual donors are available, direct studies of cytochrome P-450 multiplicity in monocytes and lymphocytes have not been reported. In this study rabbit antibodies (IgG, immunoglobulin G) to the major purified forms of cytochrome P-450 from rats treated with 3-methylcholanthrene (MC-IgG) and from rats treated with phenobarbital (PB-IgG) (*8, 13*) were used to investigate the nature and diversity of AHH activity of human monocytes and lymphocytes.

Monocytes and lymphocytes were isolated from leukocyte-rich plateletpheresis residues obtained from blood of normal volunteers, cultured, and harvested as described previously (*14, 15*). For inhibition experiments, monocytes (2.5×10^6 to 5×10^6 cells) and lymphocytes (5×10^6 to 10×10^6 cells) were first incubated at room temperature in 0.5 ml of 50 m*M* potassium phosphate buffer (*p*H 7.55) either alone or with IgG from rabbits prior to immunization, MC-IgG, or PB-IgG. Antibody was used at 1 mg per 0.5 ml, except where indicated otherwise. After the preliminary incubation, 0.5 ml of solution containing the remaining assay ingredients was added to give final concentrations of: 50 m*M* phosphate buffer, *p*H 7.55, 4 m*M* $MgCl_2$, 25 m*M* nicotinamide, 0.7 mg of bovine serum albumin per milliliter, 0.8 m*M* reduced nicotinamide adenine dinucleotide phosphate (NADPH), and 1.0 m*M* reduced nicotinamide adenine dinucleotide

Table 1. Effect of immunoglobulin G (IgG) from unimmunized animals (designated preimmune IgG), MC-IgG, and PB-IgG on AHH activity of human monocytes and lymphocytes.

Donor/additions	AHH activity* (percent of control)†			
	Monocytes		Lymphocytes	
	Uninduced	BA-induced	Uninduced	BA-induced
Donor 1				
None	0.51 ± 0.12 (100)	5.70 ± 0.72 (100)		
Preimmune IgG	0.55 ± 0.04 (108)	6.24 ± 0.33 (109)		
MC-IgG	0.24 ± 0.04 (47)	3.54 ± 0.43 (62)		
Donor 2				
None	1.29 ± 0.18 (100)	18.78 ± 5.21 (100)	0.38 ± 0.08 (100)	1.33 ± 0.17 (100)
Preimmune IgG			0.26 ± 0.06 (68)	0.83 ± 0.03 (62)
MC-IgG	0.43 ± 0.15 (33)	7.97 ± 2.54 (42)	0.06 ± 0.04 (16)	0.17 ± 0.04 (13)
PB-IgG	1.52 ± 0.04 (118)	13.50 ± 2.12 (72)	0.18 ± 0.01 (47)	0.90 ± 0.06 (68)
Donor 3				
None	0.35 ± 0.11 (100)	7.85 ± 2.21 (100)	0.47 ± 0.12 (100)	1.38 ± 0.06 (100)
MC-IgG	0.10 ± 0.05 (29)	2.72 ± 0.87 (35)	0.03 ± 0.03 (6)	0.15 ± 0.05 (11)
PB-IgG	0.42 ± 0.11 (120)	7.60 ± 1.24 (97)	0.63 ± 0.16 (134)	1.09 ± 0.00 (79)

*AHH activity is expressed as units per milligram of protein; values represent the means ± standard deviation (four to eight determinations) except that where PB-IgG was used only two to four determinations were made. †The activity of each cell sample in the absence of IgG (no additions) is designated as 100 percent. The other numbers in parentheses show AHH activity in comparison to the control.

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