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ABSTRACT

In this paper data from the National Health and Nutrition Examination Survey, 1976-1980, and from the U.S. Environmental Protection Agency's SAROAD system are used in an empirical analysis of the effects of carbon monoxide in the ambient air on blood pressure. There is evidence in these data of a positive effect of carbon monoxide exposure on diastolic and systolic blood pressure. This effect is stable and statistically important across a large number of alternative specifications, including those with additional criteria air pollutants. There is little evidence of relationships between the other criteria pollutants and blood pressure, which is consistent with epidemiological literature that identifies carbon monoxide as the primary threat to cardiovascular health among ambient air pollutants. The carbon monoxide effect on blood pressure implied by the regression results is small, but likely biased toward zero by measurement error.

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Carbon Monoxide in the Ambient Air and Blood Pressure: Evidence from NHANES II and the SAROAD System

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The effects of atmospheric air pollutants on human cardiovascular health are not well established. Evidence that exposure to ambient air pollution adversely affects cardiovascular health is strongest in the case of carbon monoxide (Hackney, 1975). There is evidence that carbon monoxide exposure increases myocardial ischemia in patients with coronary artery disease, aggravates angina pectoris, contributes to nonfatal and fatal myocardial infarction and sudden death from coronary artery disease, and plays a role in atherosclerotic disorders (Aronow, 1983). There is also some evidence of an association between carbon monoxide exposure and higher blood pressure. In their study of foundry workers, Hernberg, et al. (1976) found higher diastolic and systolic blood pressures in those occupations (furnacemen and casters) with the greatest carbon monoxide exposures. These differences were statistically significant. Shiotsuka, et al. (1984) reported that long term exposure to carbon monoxide enhanced the development of hypertension among Dahl rats bred for sensitivity to systemic hypertension induced by sodium chloride. Ahmad and Ahmad (1980) argued that the higher rates of hypertension among residents of Bombay and Delhi born in a rural Indian village as compared to residents of the same village with similar diet, smoking habits, and "genetic stock" could only be due to the high concentrations of carbon monoxide in the urban areas.[1]

In this paper we provide further evidence on the relationship between carbon monoxide in the ambient air and blood pressure. To do so we combine data from the National Health and Nutrition Examination Survey, 1976-1980, with data from the U.S. Environmental Protection Agency's SAROAD system. Our methodology is similar to Ostro and Anderson (1981), Ostro (1983), Portney and Mullahy (1983, 1986) and Gerking and Stanley (1984, 1986) in that we use health, socioeconomic, and demographic data on individuals in conjunction with ambient air pollution data from stationary monitors to assess the impact of air pollution on health. We find in this research that there is a statistically important relationship between carbon monoxide and blood pressure in the NHANES II-SAROAD data that is stable across a large number of alternative specifications.

I. The Data

The National Health and Nutrition Examination Survey, 1976-1980, is a national probability sample of the U.S. noninstitutionalized civilian population, ages 6 months through 74 years. It contains health, demographic, and socioeconomic information on approximately 20,000 individuals. Persons with low income, preschool children, and the elderly were over-sampled.[2] Each individual in the sample was given a detailed physical examination that emphasized potential health problems in the cardiovascular, respiratory, and neurological systems and included the measurement of systolic and diastolic blood pressure.[3] Medical history, dietary, and socioeconomic characteristics questionnaires were also administered to each subject.

We have augmented NHANES II with air pollution variables from the U.S. Environmental Protection Agency's Storage and Retrieval of Aeromatic Data System (SAROAD). This is an automated processing system used by EPA for the storage of data measuring concentrations of six criteria air pollutants: carbon monoxide, lead, sulfur dioxide, total suspended particulates, nitrogen dioxide, and ozone. These are pollutants for which National Ambient Air Quality Standards have been established as part of the Clean Air Act. Each subject in NHANES II residing in a census tract[4] was matched to all air pollution monitors within a five, ten, and twenty mile radius of the 1980 population centroid of his census tract.[5]

The raw data in SAROAD take the form of hourly concentrations of carbon monoxide, sulfur dioxide, nitrogen dioxide, and weekly concentrations of lead and total suspended particulates. Ozone concentrations are recorded hourly, but only in the "ozone season," which varies among regions. For each monitoring station that satisfied the EPA standard for completeness of data in a given year (at least 75 percent of all potential readings present), we computed annual hourly arithmetic averages of carbon monoxide, sulfur dioxide, and nitrogen dioxide, annual arithmetic averages of daily maximums of ozone concentrations, and annual weekly arithmetic averages of lead and particulate concentrations. These computations were made for the examination year and for each of the three previous years. Then six pollutant-specific annual averages for each subject were obtained as simple averages of the monitor averages. Finally, the pollutant-specific four-year average for the individual was calculated as a simple average of the four annual averages. If data were missing for one or two years, a three- or two-year average was computed. If an annual average was available for a single year alone, that figure was used. The six pollutant-specific "four-year" averages are the measures employed in the multiple regressions presented below. This algorithm will then smooth out unusually high or low short term fluctuations in monitor readings due to technical failure or other random events. It also allows for the importance of monitor proximity to vary by pollutant and recognizes there may be tradeoffs between information gained by including individuals more than five or more than ten miles from a monitor and precision lost by including individuals too far from monitors to have meaningful readings for a particular pollutant.

Nevertheless, it is apparent that substantial measurement error problems remain when assigning air pollution data to individuals from air pollution monitors within a certain distance of the census tract of residence. These problems result from the substantial variation in the levels of some pollutants within narrow geographic areas[6] and the substantial variation in the levels of some pollutants indoors and outdoors in a given location.[7] Thus individuals in the same census tract can have different exposures depending on their particular geographic location in the tract, and on the amount of time spent indoors and outdoors. Additional confounding factors are the amount of time spent away from home (e.g. at work) and the years of residence in the census tract, an issue to the extent longer term or cumulative exposure is of interest. Because econometric studies in air pollution epidemiology traditionally employ a health production framework and focus on an ambient air pollution variable as a health input, measurement error associated with the pollution exposure variable can be a serious problem to the extent it biases toward zero the pollution coefficient and its t statistic. It is possible that the findings of negligible health effects for some criteria air pollutants in the research previously cited are a statistical artifact resulting from measurement error in the exposure variables.

II. Empirical Results

In this paper we also employ a health production framework to examine the effects of carbon monoxide on blood pressure. Systolic and diastolic blood pressure are the health outcome variables.[8] Following the blood pressure regression literature, independent variables in the analysis include age, the square of age, race, sex, education, body mass, family income, cigarette smoking, and alcohol use, in addition to the six air pollution variables.[9] Variable definitions and summary statistics are presented in Tables 1 and 2. Blood pressure levels are age dependent and the analysis was done over three age groups: 12-17, 18-39, and 40-74 years. The older age group is emphasized here because this is the tradition of previous studies (high blood pressure is usually confined to this age group) and because the number of observations are larger for this group.

Ordinary least squares regression results for systolic and diastolic blood pressure for 40-74 year olds in NHANES II within 20 miles of a SAROAD monitor are presented in Table 3. Each air pollutant is considered separately, that is no more than one pollutant appears in any regression. These results probably yield artificially high t statistics for the pollution variables because collinearity between pollutants is not allowed to operate and because adjustments for sample design, which usually lowers t statistics, have not been carried out.[10] The carbon monoxide coefficients are

Table 1

Definitions of Variables

Variable	Definition
Diastolic blood pressure	Lowest arterial blood pressure in millimeters of mercury (mm. Hg) of a cardiac cycle occurring during an expansion of the heart; average of readings from the two measures taken in the sitting position
Systolic blood pressure	Highest arterial blood pressure in mm. Hg of a cardiac cycle occurring during a contraction of the heart; average of readings from the two measures taken in the sitting position
Age	Age of individual in years
Age squared	Square of age
Female	Dichotomous variable that identifies females
Black	Dichotomous variable that identifies blacks
Other	Dichotomous variable that identifies individuals not black or white
Education	Highest grade of school completed
Income	Family income in dollars
Weight/(Height)^2	Ratio of weight in pounds to the square of height in inches
Cigarettes	Average number of cigarettes smoked per day
Alcohol	Frequency of consumption of alcoholic beverages (beer, wine, and distilled spirits) per week during the past three months
Carbon monoxide[a]	Four-year average carbon monoxide level; milligrams per cubic meter
Lead[a]	Four-year average lead level; micrograms per cubic meter
Sulfur dioxide[a]	Four-year average sulfur dioxide level; micrograms per cubic meter

Table 2

Summary Statistics[a]

Variable	Mean	Standard Deviation
Diastolic blood pressure	82.2	12.0
Systolic blood pressure	136.0	22.0
Age	59.3	9.4
Female	.52	.50
Black	.16	.37
Other	.03	.18
Education	11.0	3.8
Income	14,794	8,838
Weight/(Height)^2	.04	.01
Cigarettes	6.44	12.34
Alcohol	2.68	3.63
Carbon Monoxide	.88	1.44
Lead	1.88	.20
Sulfur Dioxide	39.74	15.8
Total Suspended Particulates	68.07	19.79
Nitrogen Dioxide	77.84	22.56
Ozone	.06	.01

[a]Means and standard deviations from carbon monoxide twenty mile sample, 40-74 year olds in NHANES II, with the exception of the last five pollution variables. The total suspended particulates mean and standard deviation are from the total suspended particulates ten mile sample. The means and standard deviations of the remaining pollutants are from their respective twenty mile samples.

Table 3

Results of Regressing Systolic and Diastolic Blood Pressure of 40-74 Year Olds in NHANES II within 20 Miles of SAROAD Monitors Against Criteria Air Pollutants and Other Variables

Independent Variables	Carbon Monoxide		Sulfur Dioxide		Nitrogen Dioxide	
	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic
Intercept	21.995 (2.50)	57.378 (3.70)	26.696 (3.03)	67.910 (4.36)	-0.227 (-0.01)	31.611 (1.36)
Age	1.371 (4.47)	0.651 (1.20)	1.271 (4.13)	0.418 (0.76)	2.345 (4.95)	1.613 (2.00)
Age Squared	-0.012 (-4.50)	0.0006 (0.14)	-0.011 (-4.18)	0.002 (0.53)	-0.206 (-5.02)	-0.007 (-1.08)
Female	-2.458 (-5.07)	-0.686 (-0.80)	-2.846 (-5.80)	-0.990 (-1.14)	-3.562 (-4.73)	-1.140 (-0.89)
Black	3.818 (5.72)	3.901 (3.30)	2.707 (4.18)	2.905 (2.53)	3.559 (3.74)	4.465 (2.76)
Other	4.108 (3.10)	6.725 (2.86)	0.145 (0.07)	0.539 (0.15)	1.064 (0.47)	2.188 (0.57)
Education	0.033 (0.48)	-0.124 (-1.01)	0.021 (0.30)	-0.148 (-1.116)	0.019 (0.18)	-0.088 (-0.50)
Income	-0.00001 -.046	-0.00007 -1.42	-0.00002 -0.92	-0.00005 -0.97	-0.00006 -1.39	-0.00008 -1.07
Weight/ Height^2	581.087 (16.37)	964.192 (15.35)	579.167 (16.62)	883.911 (14.35)	593.531 (10.85)	933.387 (10.04)
Cigarettes	-0.053 (-2.71)	0.017 (0.49)	-0.044 (-2.30)	-0.008 (-0.25)	-0.083 (-2.64)	-0.026 (-0.49)
Alcohol	0.279 (4.23)	0.420 (3.58)	0.267 (3.98)	0.411 (3.46)	0.210 (2.13)	0.210 (1.25)
Pollutant	0.621 (3.85)	0.832 (2.91)	0.031 (2.27)	0.071 (2.88)	-0.016 (-0.98)	0.027 (0.97)
R^2	.1503	.1990	.1409	.1782	.1720	.2064
F	38.73	54.04	36.52	48.05	20.19	25.04
N	2350	2350	2387	2387	1017	1018

[a] t statistics in parentheses.

Table 3 (concluded)

Independent Variables	Total Suspended Particulates		Ozone		Lead	
	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic
Intercept	35.494 (5.01)	65.574 (5.15)	26.495 (2.90)	61.179 (3.74)	10.457 (0.63)	42.517 (1.47)
Age	1.102 (4.47)	0.688 (1.55)	1.411 (4.46)	.745 (1.31)	1.934 (3.31)	1.567 (1.54)
Age Squared	-0.009 (-4.65)	0.001 (0.04)	-0.012 (-4.55)	-0.0003 (-0.07)	-0.016 (-3.27)	-0.007 (-0.83)
Female	-2.515 (-6.51)	-0.371 (-0.53)	-2.850 (-5.71)	-1.402 (-1.56)	-2.005 (-2.12)	-0.478 (-0.29)
Black	3.115 (5.80)	3.690 (3.82)	2.973 (4.73)	3.540 (3.14)	0.285 (0.19)	1.374 (0.53)
Other	3.224 (2.49)	6.752 (2.90)	1.234 (0.63)	0.129 (0.03)	4.281 (1.38)	7.940 (1.46)
Education	-0.059 (-1.04)	-0.257 (-2.52)	0.042 (0.60)	-0.110 (-0.87)	-0.218 (-1.72)	-0.101 (-0.45)
Income	-0.00001 (-0.61)	-0.00005 (-1.27)	-0.00001 (-0.58)	-0.00004 (-0.81)	-0.00003 (-0.56)	-0.0001 (-1.24)
Weight/ $Height^2$	575.460 (20.83)	882.616 (17.78)	558.869 (16.04)	849.549 (13.61)	525.188 (7.86)	894.500 (7.67)
Cigarettes	-0.034 (-2.18)	0.026 (0.94)	-0.061 (-3.06)	-0.008 (-0.22)	-0.014 (-0.35)	0.062 (0.85)
Alcohol	0.226 (4.15)	0.294 (2.99)	0.279 (4.04)	0.342 (2.76)	0.219 (1.69)	0.168 (0.74)
Pollutant	-0.015 (-1.71)	-0.013 (-0.78)	34.818 (-1.92)	25.023 (0.77)	-1.323 (-0.56)	-8.219 (-2.01)
R^2	.1413	.1776	.1506	.1792	.1287	.2135
F	56.94	74.50	36.06	44.20	8.90	15.86
N	3739	3745	2176	2178	589	591

positive in the diastolic and systolic blood pressure models, with t values of 3.9 and 2.9, respectively.[11] The sulfur dioxide coefficients are also positive in the diastolic and systolic models, with t values of 2.3 and 2.9.

The remaining air pollution variables are negatively related to blood pressure in at least one of the blood pressure specifications and have generally low t statistics. In order to allow for more than one pollution variable in a blood pressure model consider first diastolic blood pressure regressions that include the independent variables of table 3 with the pollution variables added one at a time to form the following five pollution variable combinations: carbon monoxide (repeated from table 3); carbon monoxide and sulfur dioxide; carbon monoxide, sulfur dioxide, and total suspended particulates; these three pollutants plus ozone; and these four pollutants plus nitrogen dioxide.[12] The carbon monoxide regression coefficients and t values from these models are: .62, 3.9; .63, 3.5; .64, 3.5; .84, 4.1 and 1.75, 4.6. The carbon monoxide coefficients and t values in systolic blood pressure models with the same independent variables are .83, 2.9; .77, 2.5; .86, 2.7; 1.1, 3.0; and 1.1, 1.7. The coefficients in these relationships are obviously quite stable. In the second specification (carbon monoxide and sulfur dioxide are the pollution variables), the sulfur dioxide coefficients and t values fall by half in both the diastolic and systolic regressions in comparison to the sulfur dioxide results in table 3. The other pollution variables show no consistent relationship to diastolic blood pressure in multi-pollution variable specifications. The results of this empirical exploration are consistent with the general view that carbon monoxide is more deleterious to cardiovascular health than the other criteria air pollutants.[13]

Leamer's (1978) SEARCH algorithm can be used to further test the sensitivity of the carbon monoxide results to equation specification. In addition to other statistics, SEARCH generates the bounds of regression coefficients constrained to lie within 95% confidence ellipsoids. These bounds depend on which independent variables are designated as "free" or certain to appear in the model and which independent variables are designated as doubtful. These bounds can be quite different than those derived from simple sensitivity analysis that includes and then omits doubtful variables because the parameterization of the doubtful variables is not artificially restricted. Rather all linear combinations of the doubtful variables are considered in a "global sensitivity analysis."[13] The application of SEARCH to diastolic blood pressure models with the intercept, weight/(height)^2, sex, and carbon monoxide as free variables and with the other pollution variables and other independent variables as doubtful variables yields the following upper and lower bounds of the 95 percent confidence ellipsoids for the carbon monoxide variable: .81 and 1.04, .83 and 1.03, .78 and 1.06, .77 and 1.06, and 1.98 and 2.33. Five sets of bounds result because the doubtful pollution variables are added in the order indicated in the preceding paragraph. The bounds are relatively narrow and stable and indicate a positive effect of carbon monoxide on diastolic blood pressure. Application of SEARCH to systolic blood pressure on this manner also indicates coefficient stability. Using SEARCH in the same way for the other criteria pollutants (each pollutant allowed to be a free variable in the company of the other pollution variable combinations specified as doubtful) yields no instances where the upper and lower bounds are both positive, except in the case of sulfur dioxide. The bounds for the 95 percent confidence ellipsoid for this variable with carbon monoxide doubtful are .01 and .03. The other pollution variable combinations result in positive and negative 95 percent bounds for sulfur dioxide as a free variable.

To evaluate the size of the carbon monoxide effect on blood pressure implied by the regression results consider the effects of a one standard deviation reduction in carbon monoxide (1.4 mg/m^3) on diastolic blood pressure. Since the carbon monoxide regression coefficient is approximately one, such a reduction in average carbon monoxide exposure would reduce diastolic blood pressure by about one and one-half units. This is a small change in blood pressure -- the mean and standard deviation of diastolic blood pressure for this age group are 82.2 and 12.0. However, this is not to say that such a reduction in carbon monoxide would have inconsequential public health consequences.

The Pooling Project results indicate that a one unit fall in diastolic blood pressure across 40-74 year olds males would reduce the number of major coronary events (first time heart attacks and strokes) by approximately two for every 1,000 men in this age group over an 8.6 year period (Pooling Project Research Group, 1978). This implies an annual reduction of about 10,000 major coronary events among males in this age group in the U.S. Also, the reader is reminded that the carbon monoxide regression coefficients are biased toward zero because of the previously described measurement error.

III. Summary and Conclusion

To summarize, there is evidence from the NHANES II and SAROAD data of a positive effect of carbon monoxide exposure on diastolic and systolic blood pressure. This effect is stable and statistically important[14] across a large number of alternative specifications, including those with additional criteria air pollutants. There is little evidence of relationships between the other criteria pollutants and blood pressure, which is consistent with epidemiological literature that identifies carbon monoxide as the primary threat to cardiovascular health among ambient air pollutants. The carbon monoxide effect on blood pressure implied by the regression results is small and this research must be considered exploratory because of the difficulties in accurately measuring carbon monoxide exposure with these data. However, because even small reductions in blood pressure might have important implications for cardiovascular health, it is important that future research establish the strength of the carbon monoxide-blood pressure relationship.

Footnotes

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[1]Ahmad and Ahmad (1980) provide two possible explanations for the association between carbon monoxide exposure and hypertension. The first of these is that "chronic stimulation of the cardiac chemoreceptor and adrenal glands by serotonin could contribute to the pathogenesis of hypertension" (p. 565). The idea here is that the human body responds to the risk of carbon monoxide exposure by increasing blood pressure as a protective measure. The second explanation is that the association of carbon monoxide with arteriosclerosis may produce higher blood pressure because a stiffening of the baroreceptor area may result in partial denervation.

[2]For a detailed description of NHANES II, see National Center for Health Statistics (1981). The exact sample size is 25,285. Of these subjects, 20,322 received the physical examination.

[3]Problems with the blood pressure data in NHANES II have recently been acknowledged by the National Center for Health Statistics (1986). In their

description of trends in blood pressure in the United States from 1960 to 1980, it is pointed out that the large proportion of diastolic blood pressure measures with an end digit of zero in NHANES II may be indicative of a lack of concentration or of hearing impairment among the examining physicians during the measurement of diastolic blood pressure. This is possible because the examining physicians in NHANES II were almost all retired practitioners and because hearing acuity diminishes with age. Systolic blood pressure measurement was thought not to be adversely affected by physician hearing loss or carelessness. The end digit zero problem characterized the latter part of the NHANES II period with blood pressure data from five primary sampling units in particular being of questionable quality (Coate and Fowles, 1987). Deleting data from these PSUs from the samples used below lowers slightly the coefficients and t values of the carbon monoxide variables in models reported below.

[4]All counties in SMSA's have census tracts. Whether or not a county outside an SMSA has census tracts is a local government decision. Approximately two-thirds of the NHANES II subjects resided in counties with census tracts.

[5]Because of the large number of total suspended particulate monitors, readings for this pollutant were never taken from monitors more than ten miles from the population centroid of the subjects' census tract. Note also that at a given monitor location data may be recorded for only a single pollutant.

[6]Gerking and Stanley (1984) found ozone to be the air pollutant most uniformly spread across the St. Louis metropolitan area. Pairwise Pearson

correlation coefficients of hourly readings among 25 monitors in the St. Louis metropolitan area ranged from .33 to .90. However, the highest station specific mean ozone reading was about double the lowest station specific mean reading. Carbon monoxide levels are apparently subject to greater geographic variation. Pairwise Pearson correlations among the 25 monitors in the St. Louis study ranged from .0005 to .90. Nitrogen dioxide correlations ranged from negative values to .62. Sulfur dioxide correlations reached .26 with a substantial number of negative correlations.

[7]Exposure levels of the criteria air pollutants are generally much smaller indoors than outdoors at a given location.

[8]There are other health outcome data available in NHANES II that might be related to ambient pollution exposure, in particular annual school loss and work loss days from chronic heart and respiratory disease, but these variables have very low response rates and come from health history questionnaires which are subject to recall error. Also exploratory regressions with these health outcome variables with the independent variables described below yielded insignificant F statistics.

[9]See, for example, Fortmann et al. (1983), Gordon and Kannel (1983), and Pirkle et al. (1985). Gordon and Kannel find in the Framingham data a negative relationship between own cigarette smoking, which might be considered one form of carbon monoxide exposure, and blood pressure. They attribute the finding to the lower weight of smokers, although the relationship apparently holds up in some instances after controlling for weight. Our regression results, presented below, also show a negative and significant relationship between cigarette smoking and blood pressure in

some specifications, with body mass held constant. The inclusion of both family income and education in the set of regressors is consistent with the economics literature on the determinants of health (for example, Grossman, 1972, 1975; Gerking and Stanley, 1986; Portney and Mullahy, 1986). In this literature, family income is viewed as a measure of command over real resources, while education is viewed as a measure of efficiency in health production and tastes. The simple correlation coefficient between these two variables of .25 is low enought so that problems associated with multicollinearity do not arise when both are used as regressors.

[10]Adjustments for sample design lower t statistics because they incorporate a positive (in most cases) correlation among the regression disturbance terms of individuals who reside in the same primary sampling units. Adjustments for sample design have not been carried out because the sampling weights provided with the NHANES II data are not appropriate for the various subsamples considered in this paper.

[11]Carbon monoxide coefficients were nearly identical to those reported in table 3 when the diastolic and systolic blood pressure models were estimated over separate samples of males and females. For the 12-17 year olds and 18-39 year olds carbon monoxide coefficients are positive and t values exceed three in the case of diastolic blood pressure and one in the case of systolic blood pressure.

[12]The ordering of the last three pollutants was fixed so as to maximize the number of observations in each regression. The carbon monoxide-sulfur dioxide ordering was based on their statistical significance as indicated by the t values in table 2. The number of observations for the six

regressions in the order listed in the text are: 2,342; 1,975; 1,932; 1,582; 826. Because observations with missing data are deleted, the number of observations falls as pollutants are added because data completeness requires a subject to be within twenty miles of a monitor for each pollutant included in the regression. The results for the regression with lead and the other five pollutants are not presented because of the small sample size (N less than 150).

[13]The carbon monoxide results are not consistent with the blood pressure-carboxyhemoglobin relationship in the NHANES II data. Carboxyhemoglobin concentrations were determined for a half sample of NHANES II participants from laboratory analysis of blood samples. These levels are very sensitive to the cigarette smoking status of NHANES II subjects (the simple correlation between carboxyhemoglobin levels and daily cigarette smoking is .66) but are negatively related to blood pressure even among non smokers. The problem with using carboxyhemoglobin concentrations, however, as even a short term indicator of carbon monoxide exposure, is the precipitous fall in this measure that occurs with the decline or cessation of carbon monoxide exposure. Carboxyhemoglobin concentrations in NHANES II subjects would primarily reflect recent smoking behavior or carbon monoxide exposure at the examination site or in the examination trailers.

[14]The t values of the carbon monoxide coefficients are well above two in most specifications but these values likely overstate statistical significance because adjustments for sample design have not been carried out (See note 10).

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