

Inhalation Toxicology of Automotive Emissions as Affected by an Oxidation Exhaust Catalyst

by D. K. Hysell,* W. Moore,* R. Hinners,* M. Malanchuk,*
R. Miller,* and J. F. Stara*

Preliminary data are given on the acute inhalation toxicology of automotive emissions as affected by an oxidation exhaust catalyst. The catalyst effectively reduced CO and HC in the exhaust which apparently had an effect (at least in a closed exposure system) on oxidant and NO_x levels by altering the HC/NO_x ratio. There was a resultant reduction in biological effects due to the exposure. The catalyst altered the type of particulate to one which probably contained sulfuric acid as a major component. No evidence was present in these acute exposures to suggest a toxic response due to the higher sulfate emissions or possible catalyst attrition products. The effects of long-term exposure have not yet been investigated.

Introduction

This report presents data from a series of acute animal exposure studies which were designed to assess certain health hazards of automobile exhaust from engines equipped with or without oxidative catalytic converters. It is expected that these catalytic converters will be widely used by the automobile manufacturers in order to control exhaust emission levels of carbon monoxide (CO) and hydrocarbons (HC). The concern, of course, is that use of these devices might release some other noxious or toxic substances into the environment. Three studies are discussed in this report: TAME I, J, and K. (TAME is an acronym used by us which means toxicologic assessment of mobile emis-

sions.) TAME I was a study of the biological effects of whole exhaust from an automobile engine with no catalyst, which served as a reference or baseline study; TAME J was identical except for the addition of an oxidative catalyst to the exhaust train; TAME K had the catalyst plus additional organic sulfur compounds added to the fuel to maximize production of sulfate emissions.

Experimental Methods and Results

Exposure System

The exhaust emission generation system used in these studies consisted of a 1973 Chevrolet 350 CID production engine equipped with exhaust gas recirculation (EGR), air pump, and turbodramatic transmission coupled to an "eddy current" absorption dynamometer. In each study, the engine system was run continuously for 7

* U.S. Environmental Protection Agency, National Environmental Research Center, Environmental Toxicology Research Laboratory, Cincinnati, Ohio 45268.

days on a modified California control cycle (Table 1). The gasoline used in the Chevrolet engine as a reference fuel was American Oil Co. unleaded 91 octane test fuel, intermediate grade indolene clear (Table 2). Note that in TAME K, thiophene was added to the reference fuel to produce a high sulfur fuel (1000 ppm). Some of the engine operating conditions are summarized in Table 3.

In TAME J and K, the exhaust passed through a noble metal pelletized type oxidation catalyst (manufactured by Engelhard Co. to General Motors specifications) and a muffler before mixing with CBR filtered and conditioned air in a dilution tube. The diluted exhaust was piped to a large volume mixing chamber and then entered dynamic flow irradiation chambers lighted to simulate sunlight so that photochemical reactions might occur. The irradiated exhaust then entered

Table 1. Modified California cycle used in the fuel emission studies.

Mode	Speed, mph	Time, sec
Idle	0	20
Acceleration	0 to 30	14
Cruise	30	15
Deceleration	30 to 15	11
Cruise	15	15
Acceleration	15 to 49	29
Peak	49 to 50	1.5
Deceleration	50 to 0	31.5
Total		137

animal exposure chambers. Additionally, the system provided nonirradiated exhaust in the same concentration to other exposure chambers. In each study there were clean air atmospheres, and in TAME I there was a CO atmosphere for control animal exposures. The catalyst was removed from the system for the TAME I exposure.

Aerometry

The major components characterized in the exhaust emissions and methods used are summarized in Table 4. Particulate samples

Table 2. Comparison and product analysis of the gasoline used for exhaust emission studies.

	Shipment 1 ^a	Shipment 2 ^a
Date delivered	3/30/73	10/29/73
Quantity, gal	2000	1500
Octane number, research	91.4	91.3
Octane number, motor	82.9	82.5
Lead atm. abs., g/gal	0.01	0.01
Phosphorus, g/gal	0.002	0.00
Sulfur, wt-%	0.04	0.05
Aromatics, vol-%	25.4	23.5
Olefins, vol-%	11.8	9.9
Gum, existent, mg/100 cc	0.8	1.0
Gravity, °API	61.4	61.5
Oxidation stability, min	600+	600+
Ried vapor pressure, lb	9.1	9.0

^a Shipment #1 used for studies I and J. Shipment #2 used for study K with thiophene added to produce 0.10% by weight sulfur.

Table 3. Comparison of engine operating conditions.

	TAME I	TAME J	TAME K
Fuel	Ref. only	Ref. only	Ref. +sulfur
Engine	1973 Chev., no catalyst	1973 Chev., with catalyst	1973 Chev., with catalyst
Engine time, hr	255-425	444-615	675-941
Study time, hr	170	171	166
Engine miles	8,500	12,300	16,820
Cumulative catalyst time, hr		465	632
Catalyst miles		9,300	12,640
Total fuel, lb	1,545	1,601	1,495
Fuel, lb/hr	9.08	9.40	9.02
Exhaust oxygen, %	N.A.	4.2	4.7
Air fuel ratio			
Cycling	14.4 cycling	—	—
Idle	12.4 idle		
Oil consumption, qt	1/2	1/4	1/4
Dilution ratio	9.6/1	8.7/1	9.5/1
Dilution air flow (average), SCFM	305	310	324
Dilution tube temperature (average), °F	101	114	101

Table 4. Characterization of exhaust emissions.

Pollutant component	Analytic method	Where determined ^a
Carbon monoxide (CO)	Nondispersive Infrared spectroscopy	EPM, EC
Total hydrocarbons (THC), as CH ₄	Flame ionization spectroscopy	EPM, EC
Nitrogen oxides (NO _x , includes NO and NO ₂)	Chemiluminescence spectroscopy; colorimetry with Saltzman reagent	EPM, EC
C ₁ to C ₆ hydrocarbons (several compounds)	Gas chromatography	EC
C ₆ to C ₁₀ aromatic hydrocarbons (several compounds)	Gas chromatography	EC
Aldehydes, total	MBTH according to Hauser	EC
Particulates, total mass	Filtration gravimetry	EC
Particulate size distribution		
Aerodynamic	Stage impaction (Anderson)	EC
Photonometric	Photoelectronic (Royco)	EC
Particulate composition	Infrared and ultraviolet spectrophotometry	EC
Ozone, "oxidant"	Chemiluminescence spectroscopy	EC

^a EPM = exhaust or primary exhaust: air mixture; EC = exposure chamber.

were collected on pure quartz fiber filters. Bubbler and impinger samples of the atmospheres were used for collecting ammonia and sulfur based gases.

The concentration in the exposure chambers of the various emission components are shown in Table V. The incorporation of the oxidation catalyst into the exhaust system resulted in a large reduction in CO, total HC, and various individual organic compounds. In TAME I, the photochemical reactions in the irradiated atmospheres were very pronounced as evidenced by the presence of ozone, the low value for NO, and the high value for particulate. The color and weight stability suggested the particulate to be organic in nature. In TAME J and K, the particulate was strongly acidic, was liquid in nature, lost significant weight on standing,

Table 5. Engine exhaust emission values for selected components in the animal exposure chambers. ^a

	TAME I	TAME J	TAME K
Exhaust dilution ratio	9.6/1	8.7/1	9.5/1
CO, ppm			
NI	651	46	40
I	559	41	38
THC, ppm			
NI	110	22	18
I	95	22	18
NO _x , ppm			
NI	11.9	12.9	12.6
I	5.1	12.6	11.2
NO, ppm			
NI	6.7	11.1	10.8
I	0.5	9.6	9.7
NO ₂ , ppm			
NI	5.2	1.8	1.8
I	4.6	3.0	1.5
Aldehydes, ppm			
NI	10.20	0.08	0.18
I	14.62	0.10	0.11
Aliphatics (C ₄ -C ₆), ppm			
NI	1.30	0.61	0.44
I	1.32	0.58	0.39
Olefins (C ₂ -C ₄), ppm			
NI	13.24	0.89	0.91
I	9.23	0.79	0.82
Acetylene, ppm			
NI	3.28	0.03	0.04
I	3.06	0.03	0.04
Ozone, ppm			
NI	0.0	—	—
I	0.4	—	—
Particulate, mg/m ³			
NI	0.69	0.96	6.53
I	3.19	1.09	5.85

^a NI = nonirradiated exhaust; I = irradiated exhaust.

and contained sulfate as a primary constituent. All of this suggested sulfuric acid as the major particulate component.

Biologic Systems and Effects

Infant Mortality and Body Weight Determinations: Groups of 10 lactating female outbred albino rats and their 2-week old offspring (10 suckling rats/litter) were exposed to each of the treatment atmospheres for 7 days. Animals were weighed at the beginning and end of the study. Infant mortality was noted on a daily basis. As may be seen from Table 6, there was a prominent effect on infant mortality in those animals exposed to exhaust in TAME I. This was obviously not a CO effect alone, but rather due to the combination of biologically active

Table 6. Survival of suckling rats following exposure to whole automobile exhaust.

	Survival, %		
	TAME I	TAME J	TAME K
Clean air control (CA)	100	98	100
Nonirradiated exhaust (NI)	23	100	100
Irridated exhaust (I)	0	100	100
Carbon monoxide control (CO)	96		

Table 7. Daily change in body weight in rats during exposure to whole automobile exhaust. *

	Change in body weight, g		
	TAME I	TAME J	TAME K
Lactating female rats			
CA	+2.2	+2.9	+0.8
NI	-6.7	-0.2	+2.2
I	-11.7	-1.3	+4.6
CO	+0.4		
Suckling rats			
CA	+2.0	+2.0	+2.1
NI	-0.6	+1.9	+2.6
I	—	+2.1	+3.3
CO	+1.3		

* CA=clean air control atmosphere; NI=nonirradiated exhaust atmosphere; I=irradiated exhaust atmosphere; CO=carbon monoxide (550 ppm) control atmosphere.

pollutants. A parallel effect was noted as far as body weight changes (Table 7) in both the adult and suckling animals. In TAME J and K, there were not pronounced treatment effects on either of these parameters.

Clinical Pathology Determinations:

Groups of 25 adult male outbred albino rats were exposed to each treatment atmosphere. Five animals per treatment were removed on days 2-6 of the study, anesthetized, and exsanguinated by abdominal aorta catheterization. The clinical laboratory determinations and treatment means are shown in Tables 8-11. Again, a treatment effect, if present, occurred only in TAME I in the exhaust exposure groups with the more prominent changes in the animals exposed to irradiated exhaust. The high CO levels had some effect on the hematologic parameters but was

Table 8. Treatment mean values for selected hematologic parameters in male rats.

	TAME I	TAME J	TAME K
RBC/mm ³ × 10 ⁻⁶			
CA	7.07	7.14	
NI	7.62	7.00	7.01
I	7.78	7.07	7.21
CO	7.44		
WBC/mm ³ × 10 ⁻³			
CA	9.1	9.3	
NI	11.9	9.0	9.4
I	12.0	8.7	8.8
CO	15.7		
Lymphocyte:neutrophil ratio			
CA	5.3	5.3	
NI	1.7	5.1	4.8
I	1.0	5.3	4.9
CO	2.3		
Hemoglobin (HB), g-%			
CA	14.9	14.6	
NI	16.5	14.7	14.5
I	16.7	14.5	14.7
CO	15.2		
Hematocrit (HCT), %			
CA	41.6	40.8	
NI	46.6	41.0	40.4
I	47.4	40.1	41.0
CO	43.2		

not totally responsible. It should be further noted that the high CO levels produced a rather striking increase in hemolysis resistant red blood cells which necessitated manual determinations of white blood cell counts. No treatment effect was noted in TAME J or K. Because of the historical association of platinum with allergic responses, it was particularly interesting to note no increase in eosinophils in TAME J or K.

Tissue Chemistry Determinations: Samples of lung, liver and kidney were collected from animals exposed to the exhaust atmospheres for determination of platinum (Pt) and palladium (Pd). The tissue samples were lyophilized and wet digested by using aqua regia, all nitric acid fumes being eliminated by other additions of hydrochloric acid and subsequent heating. The digested samples were transferred with hydrochloric acid and deionized water to a volumetric flask with an acid concentration of 10%. After the samples were treated with potassium iodide, the metals were concentrated by organic extraction with methyl isobutyl ketone. Aliquots (50 μl) were analyzed by using a Perkin-

Table 9. Treatment mean values for selected blood chemistry parameters in male rats.

	TAME I	TAME J	TAME K
Total serum protein, g-%			
CA	6.0	5.8	
NI	6.3	6.1	6.2
I	6.8	6.0	6.2
CO	6.3		
Alkaline phosphatase, U			
CA	79.1	80.9	
NI	54.2	91.4	94.5
I	40.8	83.4	89.8
CO	79.4		
SGOT, R-F units			
CA	161.6	169.7	
NI	185.3	174.4	161.8
I	196.7	174.8	162.8
CO	175.2		
SGPT, R-F units			
CA	48.5	40.0	
NI	60.5	40.7	47.2
I	53.7	42.0	46.4
CO	45.8		
BUN, mg-%			
CA	23.8	22.5	
NI	21.0	21.8	19.1
I	28.3	21.4	19.4
CO	20.0		

Table 10. Treatment mean values for selected coagulation tests in male rats.

	TAME I	TAME J	TAME K
Platelets/mm ³ × 10 ⁻⁶			
CA	0.95	0.93	
NI	1.07	0.97	1.02
I	1.10	0.92	.98
CO	1.08		
Fibrinogen, mg/dl			
CA	170	175	
NI	165	175	177
I	220	170	175
CO	160		
Prothrombin time, sec			
CA	12.5	12.2	
NI	12.9	12.1	12.2
I	12.6	12.3	12.2
CO	12.3		
Partial thromboplastin time, sec			
CA	19.9	19.9	
NI	21.7	19.3	19.1
I	22.4	19.2	18.7
CO	21.3		

Elmer 503 atomic absorption spectrophotometer equipped with a GHA 2000 graphite furnace.

The lower limits of detectability for total Pt and Pd in a gram of tissue were 0.2 and 0.1 µg, respectively. No Pt or Pd could be

Table 11. Treatment mean values for selected serum electrolytic constituents in male rats.

	TAME I	TAME J	TAME K
Na ⁺ , meq/l.			
CA	133.2	140.8	
NI	136.6	141.0	142.2
I	135.7	141.2	142.8
CO	142.6		
K ⁺ , meq/l.			
CA	4.8	5.3	
NI	5.0	5.5	5.3
I	5.2	5.4	5.6
CO	5.2		
Cl ⁻ , meq/l.			
CA	103.2	105.6	
NI	104.4	104.8	105.2
I	102.1	105.2	104.2
CO	106.2		
Ca ²⁺ , meq/l.			
CA	4.9	4.4	
NI	5.0	4.6	4.6
I	4.9	4.5	4.7
CO	4.7		

detected in any of the tissue samples from TAME I, J, or K.

Morphologic Pathology Determinations:

Tissues from adult outbred albino rats and adult male golden Syrian hamsters exposed to each treatment were collected and fixed in 10% formalin. Specimens of lung, liver, and kidney were processed, paraffin-embedded, sectioned, stained with hematoxylin and eosin, and examined microscopically for evidence of morphologic changes attributable to the exposure.

There were no treatment-related changes in TAME J or K. In TAME I, however, there were extensive pulmonary changes which were more severe in hamsters and most severe in those animals exposed to irradiated exhaust. In the nonirradiated exhaust group, the pulmonary changes were relatable to the levels of NO₂, with an increase in alveolar macrophages at the level of terminal bronchioles initially, followed by a proliferative phase with some apparent increase in epithelialization of respiratory ductules, and in thickness of alveolar septae. In the hamsters exposed to irradiated exhaust there was a very severe acute purulent bronchitis and bronchiolitis which progressed to a sub-acute purulent bronchopneumonia by the end of the study. Additionally, there were some

degenerative changes in renal and hepatic tissue by the end of the study in these animals. The only lesion which could be related solely to CO levels was extramedullary hematopoiesis in the liver of the rats after 4 days exposure.

Discussion

The initiating force behind use of the oxidative catalytic converter in the automobile exhaust train is the emission standards for CO and HC as set forth in the Clean Air Amendments of 1970 (1). The aerometry findings in this study would suggest that there is in fact a very marked reduction in CO and HC levels due to the use of the catalyst.

It was further expected that the oxidative catalysts would have minimal effect on NO_x levels, which again was corroborated by these studies. The catalyst did have an indirect effect in the exposure system used in these studies on levels of NO_2 and other oxidants (i.e., ozone) which constitute some of the more biologically active exhaust compounds relative to biological effects. The reasons for this relate to findings that at HC/ NO_x ratios less than 3:1, no free oxidant is formed (2). These same HC/ NO_x ratios have a similar effect on NO_2 levels due to the overall NO- NO_2 reaction systems. In TAME I, the

HC/ NO_x ratio was about 9:1; in TAME J and K the ratios were about 1.5-2:1. This helps explain the pronounced reduction in acute toxicity associated with the exposures, rather than the lower levels of CO alone.

As noted, the oxidation catalyst did have an effect on the type of particulate with an increase in the acidic fraction (probably sulfuric acid). There were no demonstrable acute biological effects in any of the animals studied which were attributable to these altered particulates. The study did not rule out possible chronic effects due to long-term exposure, either as a result of the increased sulfate emissions or attrition products of the noble metal oxidation catalyst. It is therefore imperative that long-term studies be initiated to provide this additional information.

REFERENCES

1. Clean Air Act (42 U.S.C. 1857 et seq.) includes the Clean Air Act of 1963 (P.L. 88-206), and amendments made by the Motor Vehicle Air Pollution Control Act, P.L. 89-272 (October 20, 1965), the Clean Air Act Amendments of 1966, P.L. 89-675 (October 15, 1966), the Air Quality Act of 1967, P.L. 90-148 (November 21, 1967), and the Clean Air Amendments of 1970, P.L. 91-604 (December 31, 1970).
2. Korth, M., Rose, A., and Stahman, R. Effects of hydrocarbon to oxides of nitrogen ratios in irradiated auto exhaust. *J. Air Pollution Control Assoc.* 14: 168 (1964).