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

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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 NOx levels by altering the HC/NOX 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 emissions.) 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 turbohydraulic transmission coupled to an "eddy current" absorption dynamometer. In each study, the engine system was run continuously for 7...
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 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 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

| 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

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

|                      | Shipment 1* | Shipment 2* |
|----------------------|-------------|-------------|
| Date delivered       | 3/30/73     | 10/29/73    |
| Quantity, gal        | 2000        | 1200        |
| 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         |

* 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          |
|----------------------|-----------------|-----------------|-----------------|
| Engine               | Ref. only       | Ref. only       | Ref. +sulfur    |
| 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 | 9,300          | 12,640          |
| Catalyst miles       | 1,545           | 1,601           | 1,495           |
| Total fuel, lb       | 9.08            | 9.40            | 9.02            |
| Exhaust oxygen, %    | N.A.            | 4.2             | 4.7             |
| 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 |
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, 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

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**Table 4. Characterization of exhaust emissions.**

| Pollutant component | Analytic method | Where determined* |
|---------------------|----------------|------------------|
| Carbon monoxide     | Nondispersive  | EPM, EC          |
| (CO)                | Infrared spectroscopy |          |
| Total hydrocarbons  | Flame ionization spectroscopy | EPM, EC      |
| (THC), as CH₄       | Chemiluminescence spectroscopy; colorimetry with Saltzman reagent | EPM, EC |
| Nitrogen oxides     | Gas chromatography | EC               |
| (NO₂, includes NO and NO₃) |          |                  |
| C₃ to C₆ hydrocarbons (several compounds) | Gas chromatography | EC                |
| C₆ to C₁₀ aromatic hydrocarbons (several compounds) | MBTH according to Hauser | EC                |
| Aldehydes, total    | Filtration gravimetry | EC               |
| Particulates, total mass | Stage impaction (Anderson) | EC               |
| Particulate size distribution | Photoelectronic (Royco) | EC               |
| Aerodynamic         | Infrared and ultraviolet spectroscopy | EC               |
| Photonomeric        | Chemiluminescence spectroscopy | EC               |
| Ozone, "oxidant"    |                |                  |

* EPM = exhaust or primary exhaust; air mixture; EC = exposure chamber.

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

| Pollutant         | 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     |
| NO₂, 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    |
| NO₃, 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 (C₄-C₆), ppm | NI     | 1.30   | 0.61   | 0.44   |
|                   | I      | 1.32   | 0.58   | 0.39   |
| Olefins (C₆-C₈), 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/m³| NI     | 0.69   | 0.96   | 6.53   |
|                   | I      | 3.19   | 1.09   | 5.85   |

* NI = nonirradiated exhaust; I = irradiated exhaust.
Table 6. Survival of suckling rats following exposure to whole automobile exhaust.

| Survival, % | TAME | TAME | TAME |
|-------------|------|------|------|
|             | I    | J    | K    |
| Clean air control (CA) | 100  | 98   | 100  |
| Nonirradiated exhaust (NI) | 23   | 100  | 100  |
| Irradiated 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                       | -5.7   | -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 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.

| Parameter                  | TAME I | TAME J | TAME K |
|----------------------------|--------|--------|--------|
| Total serum protein, g-%   | 6.0    | 5.8    | 6.2    |
| CA                         | 6.3    | 6.1    | 6.2    |
| NI                         | 6.8    | 6.0    | 6.2    |
| I                          | 6.3    |        |        |
| Alkaline phosphatase, U    | 79.1   | 80.9   |        |
| CA                         | 54.2   | 91.4   | 94.5   |
| NI                         | 40.8   | 83.4   | 89.8   |
| I                          |        |        |        |
| CO                         |        |        |        |
| SGOT, R-F units            | 161.6  | 169.7  |        |
| CA                         | 185.3  | 174.4  | 161.8  |
| NI                         | 195.7  | 174.8  | 162.8  |
| I                          |        |        |        |
| CO                         |        |        |        |
| SGPT, R-F units            | 48.5   | 40.0   |        |
| CA                         | 60.5   | 40.7   | 47.2   |
| NI                         | 53.7   | 42.0   | 46.4   |
| I                          |        |        |        |
| CO                         |        |        |        |
| BUN, mg-%                  | 23.8   | 22.5   |        |
| CA                         | 21.0   | 21.8   | 19.1   |
| NI                         | 28.3   | 21.4   | 19.4   |
| I                          |        |        |        |
| CO                         |        |        |        |

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

| Parameter                  | TAME I | TAME J | TAME K |
|----------------------------|--------|--------|--------|
| Platelets/mm³x10^-4        | 0.95   | 0.93   | 1.02   |
| CA                         | 1.07   | 0.97   | 1.02   |
| NI                         | 1.10   | 0.92   | 0.98   |
| I                          |        |        |        |
| CO                         |        |        |        |
| Fibrinogen, mg/dl          | 170    | 175    | 177    |
| CA                         | 165    | 175    | 177    |
| NI                         | 220    | 170    | 175    |
| I                          |        |        |        |
| CO                         |        |        |        |
| Prothrombin time, sec      | 12.5   | 12.3   |        |
| CA                         | 12.9   | 12.1   | 12.2   |
| NI                         | 12.6   | 12.3   | 12.2   |
| I                          |        |        |        |
| CO                         |        |        |        |
| Partial thromboplastin     | 19.9   | 19.9   |        |
| time, sec                  | 21.7   | 19.3   | 19.1   |
| CA                         | 22.4   | 19.2   | 18.7   |
| NI                         |        |        |        |
| I                          |        |        |        |
| CO                         |        |        |        |

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 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 subacute 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 NOx 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 NOx 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/NOx ratios less than 3:1, no free oxidant is formed (2). These same HC/NOx ratios have a similar effect on NOx levels due to the overall NO–NOx reaction systems. In TAME I, the HC/NOx 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).