

TOXICITY EVALUATION OF GASOLINE EXHAUST EMISSIONS.

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INTRODUCTION

The health effects of gasoline exhaust was examined previously in two animal toxicology studies (Brightwell et al., 1986a, 1986b, 1989; Heinrich et al., 1986). These studies were part of larger efforts examining the health effect of gasoline or diesel exhausts. The Brightwell study examined the systemic and pulmonary toxicity and carcinogenicity in rats and hamsters of engine-out (uncatalyzed) and tailpipe (catalyzed) exhaust emissions from engines burning unleaded gasoline. Two studies by Heinrich examined the subchronic and chronic toxicity and carcinogenicity in rats and hamsters of engine-out exhaust emission using leaded gasoline.

The results of Brightwell and Heinrich studies provide no evidence that gasoline engine exhaust is an animal carcinogen. The strongest dose-response and time-response relationships observed in these studies were linked to carbon monoxide (CO) exposure. The authors of these reports speculated that the respiratory and histopathological changes of the lung were associated with nitrogen oxides (NO_x), CO or lead particulate. Although some uncertainty exists in this analysis, the lesions that were observed are typical of these toxicants. It is well known that CO has marked effects on hematological and cardiac indices (USEPA, 1991). Nitrogen oxides are known to adversely affect the respiratory tract (USEPA, 1993). Thus, although Brightwell and Heinrich speculated on these findings, this hypothesis appears reasonable based on the known toxicity of CO and NO_x.

Automotive gasoline composition has changed since the time these studies were conducted. Oxygenated winter fuel (oxyfuel) and reformulated gasolines (RFG) were introduced in areas of CO or CO and ozone nonattainment, respectively, in the 1990s. These fuels differ from regular gasoline by the addition of an oxygenate to promote a more efficient combustion of fuel. Oxygen content of RFG and oxyfuels are 2.0 and 2.7 wt. percent, respectively resulting in 8 to 17 volume percent of gasoline depending on the oxygenate. With the introduction of these fuels into the marketplace, there has been concern regarding the toxicity of the combustion emissions of these fuels.

Components of automotive engine exhaust have been characterized. The Auto/Oil Air Quality Improvement Research Program (AQIRP) developed an extensive database on the level and composition of exhaust and evaporative emissions from up to 20 well-maintained model year 1989 cars and light trucks operated on industry average gasoline (RFA), and gasoline blended with the oxygenates methyl-tertiary-butyl ether (MTBE), ethanol (EtOH) and ethyl-tertiary butyl ether (ETBE). From the AQIRP speciation data, extrapolations can be made to determine if the toxicity of gasoline exhaust emissions has changed, and if further animal toxicology testing will to characterize gasoline exhaust toxicology will provide additional information from that which can be inferred from the above studies.

METHODS

Two sets of data from AQIRP were used for this paper. AQIRP Pilot Study includes both engine-out and tail-pipe exhaust measurements of 156 hydrocarbon and oxygenated species sampled and composited over three phases of the FTP driving cycle. The Phase I Working Data Set includes exhaust tailpipe measurements of 156 hydrocarbon and oxygenated species, sampled and composited over three phases of the FTP driving cycle. Detailed descriptions of the data in these two data sets are presented in technical papers contained in SAE Publication SP-920, *Auto/Oil Air Quality Improvement Research Program*, February 1992.

AQIRP data (g/mile) can be used to estimate concentrations in an inhalation toxicology study (ppm) by developing a molar ratio between a particular hydrocarbon species and CO. As CO will be the limiting exhaust component, hydrocarbon/CO molar ratio can be

multiplied by the fixed concentration of CO to produce the estimated exposure concentration of hydrocarbon. The conversion is illustrated in the equation below.

$$\left(\frac{([HC_{exh}] + HC \text{ mol. wt.})}{([CO_{exh}] + CO \text{ mol. wt.})} \right) \times [CO_{exp \text{ chamb}}] = [HC_{exp \text{ chamb}}]$$

Where $[HC_{exh}]$ is the measured concentration in mg/mile of a hydrocarbon species from Auto/Oil; $HC \text{ mol. wt.}$ is the molecular weight for a given hydrocarbon species; $[CO_{exh}]$ is the measured concentration in mg/mile of carbon monoxide from Auto/Oil; $CO \text{ mol. wt.}$ is the molecular weight of carbon monoxide; $[CO_{exp \text{ chamb}}]$ is the expected chamber concentration of carbon monoxide set at 200 ppm; and $[HC_{exp \text{ chamb}}]$ is the expected animal exposure chamber concentration of a particular hydrocarbon species in ppm.

The entire speciated exhaust component data sets were converted. As noted above the expected chamber concentration of CO was set at 200 ppm based on a large number of toxicity endpoints (developmental and systemic) following review of the health effects of CO. The expected exposure concentrations from the converted AQIRP data were compared to exposure concentrations from previous studies.

Toxicology databases were searched to determine No Observable Effect Levels (NOELs) for exhaust components derived from animal toxicity studies. NOELs represent exposure concentrations at which no effects were observed in an animal toxicity study. NOELs were compared to expected exposure concentrations from the converted AQIRP data. Only a few representative exhaust components are presented due to space limitations.

RESULTS

Based on a dose limiting concentration of 200 ppm for CO, the expected chamber concentrations of CO, CO_2 , NOx and total hydrocarbon can be extrapolated from AQIRP data using the equation listed above (Table 1). These data demonstrate that for a CO concentration of 200 ppm, total hydrocarbon concentrations will be approximately 70 ppm for RFA gasoline. These data are similar to data obtained in the Brightwell and Heinrich studies, which also are listed in this table.

Hydrocarbon speciation data from the Brightwell study can be directly compared to extrapolated data from AQIRP (Table 2). The extrapolated AQIRP data have been converted from ppm to mg/m^3 to make a direct comparison to Brightwell data easier. The extrapolated data is not directly comparable as the Brightwell data is the average and range of ten measurements on one vehicle over the course of two years, and AQIRP data is the average and range of two FTP tests for three vehicles. However, it is apparent that the expected concentrations for individual hydrocarbons is similar to what was actually measured in the Brightwell study.

Comparison of extrapolated animal exposure concentrations of hydrocarbon species for RFA and oxygenated fuel mixtures MTBE, EtOH or ETBE gasoline (Table 3) indicates that the addition of oxygenate to gasoline produces minor alterations in the composition of gasoline exhaust. As seen with the engine-out data, parent oxygenate was observed in the exhaust stream, at concentrations of approximately 1 ppm. Additionally, the concentration of aldehydes was increased with the addition of oxygenate, most notably formaldehyde and acetaldehyde. The remaining hydrocarbon species are not affected greatly by addition of oxygenate.

Extrapolated animal exposure concentrations of speciated exhaust components are compared to these components NOELs in Table 4. The comparison indicates that the exposure concentrations will be well below the observable effects level for all components.

DISCUSSION

Extrapolation of the AQIRP data indicate that total hydrocarbon concentrations will be low in animal exposures using RFA gasoline. These data also indicate the hydrocarbon exposure levels will be similar to the levels observed in the previous gasoline engine exhaust toxicology studies, where only CO and/or NOx effects were observed.

Small analytical differences in speciated exhaust components do exist between the extrapolated AQIRP data for gasoline and gasoline with MTBE and the speciated data measured in the Brightwell study. However, the composition of the exhaust atmosphere between the two data sets are fairly similar. This is not entirely surprising since the specifications for the fuel used in the Brightwell study are similar to the specifications for RFA used in AQIRP. Thus, results observed in the Brightwell study appear to be applicable to exhaust emissions generated from RFA and gasoline with MTBE. Further, although there are slight analytical differences the anticipated animal exposure atmospheres for the different reformulated fuels, exposures to exhaust components in toxicology studies on reformulated or oxygenated fuels will be fairly similar to the exposures in the Brightwell study. Thus, it can be concluded that addition of oxygenate does not dramatically alter the subsequent composition of an animal exposure to gasoline engine exhaust. Therefore, the Brightwell study results can be used to evaluate the health effects of engine exhaust from oxygenated gasolines.

Comparison of anticipated animal exposure levels and NOELs for exhaust components indicates no adverse effects are likely to be observed. Dilution of engine exhaust to reduce CO toxicity provides an inherent safety factor for the hydrocarbon component of exhaust. At 200 ppm CO it is unlikely that hydrocarbon effects will be observed as the anticipated exposure levels are well below the NOELs for these compounds. Doubling the CO concentration to 400 ppm will not sufficiently increase the hydrocarbon concentration above the NOEL for the individual hydrocarbons.

CONCLUSION

Although slight analytical differences exist in exhaust HC compositions for different oxygenated fuel blends, the likelihood of discerning differences in the toxicity of HC exhaust emissions from different gasoline blends is small.

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Compound	AQIRP	Brightwell	Heinrich
CO	200	224	305
Total Hydrocarbon	68	61	36.5 ¹
NO _x	32	49	23 ²
CO ₂	4200	6000	4000

¹ Non-methane hydrocarbon

² Measured as NO

Fuel Type	Brightwell	AQIRP Pilot Study	
	California	Baseline Gasoline	Gasoline with MTBE
Units	mg/m ³	mg/m ³	mg/m ³
Compound	Range (Average)	Range (Average)	Range (Average)
Methane	1.90 - 4.94 (2.96)	1.00 - 1.08 (1.03)	1.02 - 1.14 (1.09)
Benzene	0.45 - 2.26 (1.39)	1.27 - 1.53 (1.39)	1.13 - 1.26 (1.20)
Toluene	2.59 - 6.86 (4.1)	2.00 - 2.10 (0.82)	1.36 - 1.51 (1.45)
Formaldehyde	0.104 - 0.590 (0.308)	0.48 - 1.15 (0.82)	0.71 - 1.03 (0.91)
Acetaldehyde	0.073 - 0.297 (0.148)	0.20 - 0.35 (0.26)	0.20 - 0.29 (0.24)
MTBE			1.65 - 2.16 (1.95)

Hydrocarbon	Gasoline	Gasoline/MTBE	Gasoline/EtOH	Gasoline/ETBE
Methane	1.57	1.72	1.71	1.71
Toluene	0.71	0.75	0.68	0.72
Formaldehyde	0.55	0.49	0.41	1.35
Benzene	0.33	0.34	0.32	0.44
1,3-Butadiene	0.17	0.20	0.18	0.20
Acetaldehyde	0.14	0.15	0.29	0.43
E-Benzene	0.13	0.13	0.12	0.15
o-Xylene	0.09	0.09	0.08	0.10
ETBE	0.02	0.02	0.02	1.22
MTBE	0.00	1.24	0.00	0.00
EtOH	0.00	0.00	1.35	0.00

Table 4
Comparison of NOELs and Expected Chamber
Concentration for Selected Hydrocarbons

Compound	Chamber Conc. (ppm)	NOEL ¹ (ppm)	Endpoint	Species	NOEL to Exposure Ratio
Formaldehyde	0.580	15 mg/kg ²	Systemic	rat	
		74 mg/kg ²	Developmental	rat	
Acetaldehyde	0.145	150	Systemic	rat	1000
1,3-Butadiene	0.351	6.25 ³	Reproductive	mouse	18
Benzene	0.436	10	Developmental	rat	23
		300	Reproductive	rat	700
		30	Reproductive	mouse	70
		300	Neurological	mouse	700
Toluene	0.545	56	Neurological	rat	100
		500	Reproductive	rat	1000
		750	Developmental	rat	1400
<i>m</i> - and <i>p</i> -Xylene	0.407	99	Neurological	rat	240
		250	Developmental	rat	600
		1000	Reproductive	rat	2500
Ethylbenzene	0.188	100	Developmental	rat	500
		100	Systemic	rat	500
MTBE	0.542	1000	Reproductive	rat	1900
		1000	Developmental	mouse	1900
		400	Neurological	rat	200
Ethanol	1.35	20,000	Developmental	rat	15,000
ETBE	1.22	500	Neurological	rat	400

¹ No Observable Effect Level

² Oral Exposure

³ Low Observable Effect Level