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October 19, 1990

BY MESSENGER

Ms. Mary T. Smith
Director
Field Operations and Support Division
EN-397F
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

RECEIVED
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EPA AIR DOCKET

Public Docket No. A-90-16

Dear Ms. Smith:

During the course of several recent meetings with EPA officials concerning Ethyl Corporation's ("Ethyl") waiver application for use of HiTEC® 3000 ("the Additive") in unleaded gasoline, two general questions related to public-health arose. First, does the inhalation of manganese present risks to public health different and more serious than those associated with the ingestion of manganese? Second, what would be the likely concentrations of ambient manganese in confined spaces (such as urban canyons, or parking garages), and any associated public health implications of such concentrations, if the waiver application was approved?

In this letter, we specifically address, on Ethyl's behalf, these two questions, and generally describe Ethyl's views on how

issues of public health relate to decisions under § 211(f)(4) of the Clean Air Act ("CAA" or "Act").[1/]

I. **Manganese: Inhalation vs. Ingestion**

During the comment period on the waiver application, EPA received a comment suggesting that inhaled manganese may affect the body differently than ingested manganese.[2/] No studies or other information was cited in support of this hypothesis. Ethyl nonetheless responded to this allegation in its comments on public health, asking Dr. Carl Schulz and Roth Associates to review the available information on inhaled versus ingested manganese. The results of that review showed no special concern would be associated with inhaled manganese.[3/]

Now that the EPA staff has also raised this issue, Ethyl has asked Dr. Schulz to explain in more detail his review of the available literature. Dr. Schulz's review includes occupational inhalation studies -- those studies that we understand are of

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[1/] In its prior submissions, Ethyl has already addressed generally each of these issues. In response to the questions raised by EPA, this letter simply draws the relevant material together for EPA's review, and provides a limited amount of supplemental materials.

[2/] See Memorandum from the Director, National Institute of Environmental Health Sciences to Deputy Ass't Administrator for Pesticides and Toxic Substances, docket entry IV-H-1 (June 7, 1990).

[3/] See Ethyl's Comments in Support of the Waiver Application for the HiTEC® 3000 Performance Additive (July 23, 1990) (hereinafter "Ethyl Comments") at Appendix 3.

most interest to the EPA staff. A copy of Dr. Schulz's statement is attached to this letter as Attachment 1.[4/]

This review establishes that manganese which is inhaled does not present a risk to public health any different or more serious than that associated with ingested manganese. In the words of Dr. Schulz, "there is no evidence that inhalation results in greater absorption of [manganese] or preferential distribution to the brain compared to oral exposure." Specifically, the literature indicates that:

- Only a very small portion of inhaled manganese is absorbed by the body;
- Homeostatic mechanisms control brain manganese levels regardless of whether the source of manganese is inhalation or ingestion; and
- Manganese does not accumulate in the brain over time.

Finally, the occupational studies referred to by EPA dealt with manganese concentrations orders of magnitude higher than those at issue here. As a result, any health effect observed in occupational inhalation studies would have been a product of exposure to very high doses of manganese (as high as 15,000 ug/m3), and not a product of manganese having been inhaled instead of ingested.

[4/] Ethyl also provided a copy of Dr. Schulz's statement to William Rosenberg, Assistant Administrator for Air and Radiation, and Erich Bretthauer, Assistant Administrator for Research and Development, at meetings at EPA's offices on October 2, 1990.

II. **Ambient Manganese Concentrations in Confined Spaces**

EPA also expressed interest in information concerning what the likely concentrations of ambient manganese would be in confined spaces (such as urban canyons, or parking garages) assuming use of the Additive in unleaded gasoline. In response, Ethyl requested independent experts to prepare separately estimates of the maximum short term manganese concentrations that would occur in representative, large urban canyons and enclosed parking garages, respectively. Systems Applications, Inc. (SAI) prepared the urban canyon estimate, while E.H. Pechan & Associates, Inc. ("Pechan Associates") prepared the estimate for enclosed parking garages. To predict the maximum short-term manganese concentrations, both organizations relied principally on a ratio of manganese to carbon monoxide (CO) tailpipe emissions.[5/] Copies

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[5/] Both organizations also used the results of manganese particulate tests completed by EPA on various Canadian, American, and Ethyl test fleet vehicles to predict maximum short-term manganese concentrations. While SAI and Pechan Associates used these manganese particulate test results in order to be conservative, Ethyl still has questions regarding the meaning of these test results.

For example, ECS Laboratories, a well-respected automotive testing firm, conducted manganese particulate tests earlier in this proceeding on Ethyl vehicles. This testing produced results for manganese approximately 20 times lower than those generated by EPA. This difference in test results is probably explained by the difference in the configuration of the test apparatus used by EPA and that used by ECS Laboratories. Of note, while EPA has developed a method for testing particulate emissions from diesel vehicles, there is no accepted method for testing particulate emissions from gasoline-powered vehicles.

(continued...)

of the SAI and Pechan Associates analyses are enclosed as Attachments 2 and 3 to this letter, respectively.

5/ (...continued)

Both EPA and ECS Laboratories measured manganese particulate by venting tailpipe exhaust into a tunnel and diluting with clean air. ECS's tunnel was elevated horizontally approximately six to seven feet above ground level, and was connected to the automobile exhaust by means of a flexible, uninsulated hose. ECS informs us that this is a standard configuration used to conduct EPA diesel emission tests. The EPA tunnel, by contrast, was placed at ground level and connected to a test vehicle by means of an insulated pipe approximately six feet long. The EPA tunnel had a diameter of 10 inches, while the ECS Laboratories tunnel had a diameter of 18 inches. Since both EPA and ECS Laboratories used about the same volume of clean dilution air, the velocity of the air in the EPA tunnel was about 3.24 times that in the ECS Laboratories tunnel.

Because of these differences in exhaust connection systems and air velocity, Ethyl believes that the ECS Laboratories may have measured only the airborne component of particulate manganese emissions, while the EPA approach was more likely to measure total manganese emissions, including particulate manganese which would drop to the ground following emission from the tailpipe. In this regard, it should be noted that size alone does not determine whether a particle will remain airborne. No reasonable determination as to whether a particle will remain airborne can be made without consideration of the particle's aerodynamic characteristics, including size, shape, and density. Evaluation of these factors, while helpful to interpret the differing data, would involve time-consuming, highly sophisticated equipment and techniques, and would be further complicated by the very small amount of particulate to be analyzed.

Finally, it should be noted that Ethyl has conducted its exposure and risk analyses based on the conservative assumption that at least 30 percent of the manganese in the unleaded gasoline will be emitted to the ambient air. The results of EPA's testing do not make the 30 percent assumption any less conservative. Indeed, EPA's measurements of manganese emissions from the Ethyl fleet test cars are, on average, about two times lower than the 30 percent assumption.

In addition, Ethyl calculated, using different methods involving the use of lead and CO data, potential 24-hour, time-weighted average manganese exposures for the general population, assuming use of the Additive. The first method assumes that the ambient impacts of vehicular manganese emissions will closely parallel the ambient impacts of vehicular lead emissions, and relies on measured lead levels from personal samplers for taxi drivers and commuters during peak traffic periods. The second method assumes the impact of manganese emissions will mirror the impact of CO emissions, and is based on measured differences in ambient CO concentrations and concentrations inside vehicles.[6/] A copy of Ethyl's analysis is enclosed as Attachment 4 to this letter.

The results of all of these analyses show that even in confined spaces, peak ambient manganese concentrations will remain very low:

> Urban Canyons -- The predicted maximum 1-hour ambient manganese concentration in New York City would be 0.20 ug/m3. This prediction is conservative because it is based on data from 1983-1985, and CO

[6/] Ethyl believes use of CO measurements to predict manganese concentrations are not as reliable as extrapolating from historic lead data because particulates are removed from the atmosphere at a greater rate than CO. For this reason, thus, use of CO as a tracer is likely to overstate manganese particulate concentrations.

emissions from transportation sources decreased 19 percent from 1984 to 1988.[7/]

Parking Garages -- Depending upon whether a low or high manganese emission rate is applied, the maximum 5 minute ambient manganese concentration would range from 0.25 to 0.49 ug/m3 for a typical commuter parking garage scenario.[8/]

Commuter Exposure in Automobiles -- Based on an analogy to automotive lead emissions, the maximum ambient concentration of manganese in an automobile during peak traffic periods would be 0.5 ug/m3, and a typical 24-hour weighted-average exposure to manganese for a commuter would be approximately 0.079 ug/m3. Based on an analogy to CO emissions, the maximum ambient concentration of manganese in an automobile during peak traffic periods would be 0.2 ug/m3, and a typical 24 hour weighted-average exposures to manganese for a commuter would be approximately 0.054 ug/m3.[9/]

All of these predicted maximum, short-term manganese levels are well-below the conservative 1 ug/m3 annual threshold level deemed to be protective of even the most sensitive population groups by the World Health Organization.[10/] They are also far-below the levels deemed by the Agency to be protective of the public health -- 250 ug/m3 for 15 minutes and 125 ug/m3 for 8

7/ See Attachment 3 hereto.

8/ See Attachment 4 hereto.

9/ See Attachment 5 hereto.

10/ See Ethyl Comments, supra, at Appendix 7, Attachments 2 and 3.

hours -- when the Agency decided not to regulate manganese as a hazardous pollutant in 1985.[11/]

Since the maximum, short-term ambient manganese concentrations that could occur with use of the Additive, including those associated with confined spaces, remain well-below ambient manganese levels deemed protective of the most sensitive population groups, there is no reasonable basis upon which to conclude that use of the Additive will adversely affect the public health.

III. **CAA § 211(f)(4) and Public Health**

Ethyl has previously explained that § 211(f)(4) requires a waiver applicant to demonstrate that use of a fuel additive "will not cause or contribute to a failure of any emission control device or system" to meet applicable emission standards. It does not expressly require a waiver applicant to address health issues unrelated to compliance with applicable emission standards.[12/]

This interpretation is fully consistent with EPA's prior decisions under § 211(f), applicable Agency guidance, and the legislative history of § 211(f). For example,

- In one of the first decisions under § 211(f)(4), the Agency stated in 1979 that "[s]ection 211(f)(4)[] is solely concerned with the emission standards which

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[11/] 50 Fed. Reg. 32,627, 32,628 (August 13, 1985).

[12/] See Ethyl Comments, supra, at 5-8.

apply to tailpipe emissions of HC, CO, and NOx and evaporative HC emissions;"[13]

- EPA guidance describing materials which must be submitted in waiver applications under § 211(f) does not require any health-related information;[14]

- The legislative history of § 211(f) indicates that this provision was not intended to address issues of public health. Instead, "[t]he committee expects the Administrator to require manufacturers to test registered additives insofar as they affect health and public welfare under sections (a), (b), and (c) of [§ 211]."[15]

Moreover, it should be noted that Congress expected EPA to develop rules for considering the public health and welfare implications associated with the use of fuels and fuel additives under §§ 211(b) and (e) of the Act.[16] In this regard, the Agency on August 7, 1990 issued an advance notice of proposed rulemaking (ANPRM) regarding how the public health and welfare impacts of

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[13] In Re Application for MTBE, Decision of the Administrator (December 26, 1978) at 4, n.5 (emphasis added).

[14] See 43 Fed. Reg. 11258 (1978).

[15] Senate Rep. No. 95-127, 95th Cong., 1st sess. 91-92 (1977), reprinted in, A Legislative History of the Clean Air Act of 1977, Comm. Print, Senate Comm. on Env't and Public Works (1978) at 1465-66.

[16] Section 211(b) provides that for purposes of registration of a fuel or fuel additive, the Administrator may require a manufacturer "to conduct tests to determine potential public health effects" of its product. 42 U.S.C. § 7545(b)(2). Section 211(e) requires EPA to promulgate regulations implementing its authority under § 211(b) for the testing of fuels and fuel additives. Id. at § 7545(e).

fuels and fuel additives might be evaluated under those provisions of the Act. See 55 Fed. Reg. 32,218.

This ANPRM is important for two reasons. First, the ANPRM is based solely on authority under §§ 211(b) and (e), not § 211(f). Since Congress enacted § 211(e) and § 211(f) at the same time, the clear implication is that it intended the Agency to evaluate matters pertaining to emission control performance under § 211(f) and other public health issues as necessary under §§ 211(b) and (e).[17]

Second, the recent ANPRM suggests that all designated fuels and fuel additives will be subject to examination on public health and welfare grounds once final regulations under §§ 211(b) and (e) are promulgated. See 55 Fed. Reg. at 32,219. Thus, while Ethyl believes that it has convincingly shown in this proceeding that use of the Additive will benefit the public health and welfare, a decision by the Agency to grant Ethyl's waiver application now will not preclude the Agency from revisiting public health issues in the future.

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[17] As Ethyl explained in its waiver application, public health issues unrelated to compliance with applicable emission standards are relevant in a § 211(f) proceeding, if at all, only by virtue of the general purposes clause of the Act. CAA § 101(b). Under this provision, the Agency would have to justify a decision to deny a waiver application on public health grounds. This is especially so in this case since (1) use of the Additive is predicted to reduce total pollutant emissions by about 1.8 billion pounds annually by 1999, and (2) the Agency has previously concluded that low concentrations of manganese present no public health concern. See 50 Fed. Reg. 32,627 (1985).

Finally, given the nature of the record in this proceeding, denial of Ethyl's application on public health grounds could have far-reaching policy consequences for other EPA programs. For example, if EPA denies the application based on unsubstantiated hypotheses regarding the public health effects of manganese, the same types of concerns could be used:

- To preclude approval of promising new fuels and fuel additives under § 211(f)(4);
- To challenge the continued use of existing fuels and fuel additives under §§ 211(b) and (e);
- To foreclose the use of ethanol, methanol, MTBE and other products as alternative fuels, or as components of reformulated fuels;[18] and
- To limit the Agency's discretion to list pollutants and to establish appropriate controls under either the current or an amended toxic air pollutant program.

In short, neither the record here nor the applicable legal standard provide a basis for denying the waiver application on public health or welfare grounds. Indeed, for the reasons explained here and in Ethyl's earlier comments, consideration of public health and welfare fully supports the granting of Ethyl's

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[18] For example, both ethanol and methanol increase the emission of aldehydes (acetaldehyde and formaldehyde, respectively), which are known carcinogens.

HUNTON & WILLIAMS

waiver application. Ethyl requests, therefore, that the Agency promptly approve its waiver application.

Sincerely,

John J. Adams
F. William Brownell
Kevin L. Fast

Enclosures
cc: Public Docket A-90-16
William G. Rosenberg, Esq.
Erich W. Bretthauer
Dr. J. Clarence Davies

ATTACHMENT 1

COSAR Inc.

Consultants in Occupational and Environmental Toxicology

October 3, 1990

Neil Roth, Ph.D.
President
Roth Associates, Inc.
6115 Executive Blvd.
Rockville, MD 20852

Dear Neil:

Enclosed is a report which I wrote addressing the issue as to whether exposure to manganese compounds by inhalation will result in greater uptake and higher accumulation of manganese in the brain than will exposure to similar amounts of manganese by the oral route. As you can see, I have concluded that there is no reliable evidence that exposure to airborne manganese will result in significantly greater accumulation of manganese in the brain than exposure by any other route. With regard to the proposed use of MMT as a gasoline additive, the available evidence indicates that the extremely small increment in airborne manganese concentration that might result from such use would not result in any increased body burden of this element or in any adverse health effects.

Sincerely yours,

Carl O. Schulz

Carl O. Schulz, Ph.D.,
DABT
Consulting Toxicologist

P.O. Box 50334, Columbia, SC 29250 (803) 777-6584

Brain Uptake of Manganese Following Exposure by Inhalation

Carl O. Schulz, Ph.D., DABT

I have been asked to address the issue as to whether exposure to manganese (Mn) by inhalation results in greater uptake and higher brain levels of Mn than does exposure by the oral or dermal routes. Based on my experience and review of the available literature, I have concluded that there is no evidence that inhalation results in greater absorption of Mn or preferential distribution to the brain compared to oral exposure. Furthermore, there is no reliable evidence that Mn accumulates in the brain or that inhalation enhances accumulation. The available evidence indicates that the uptake and elimination of Mn following inhalation exposure is dependent upon the solubility and the particle size distribution of the Mn compounds in the respired air. Furthermore, homeostatic mechanisms that control the body burden (including brain levels) of Mn appear to operate independently of the route of exposure.

Airborne Mn is almost entirely in the particulate form and is predominantly insoluble oxides. Upon inhalation it behaves like all insoluble particulates. Depending on particle size, some of it will deposit in the upper airways where it can be expelled through sneezing and coughing or swallowed with mucous, entering the G.I. tract. Some small but unknown fraction may be absorbed through the mucous membranes lining the upper respiratory tract. Particles having a sufficiently small diameter will enter the lower airways and the alveoli. Some of these will deposit on the airway or alveolar walls while others will remain suspended and be exhaled. Particles depositing on the walls of the lower airways may be absorbed or may be cleared to the G.I. tract by mucociliary action. Particles depositing in the alveoli will eventually be absorbed into the systemic circulation. Thus, an unknown, but likely small, fraction of inhaled manganese is absorbed through the lungs with the remainder being exhaled or transferred to the G.I. tract. The distribution among these is determined by particle size. Mena et al. (1969) reported that a large percentage of the $MnCl_2O_2$ to which humans were exposed in their study was absorbed by the G.I. tract.

There is almost no information in the scientific literature on concentrations of Mn in the brains of humans who have been exposed to manganese or who exhibit signs and symptoms of Mn intoxication. What little information is available is contradictory. Banta and Markesbery (1977) reported that brain Mn levels were three times normal levels in a man suffering from Mn poisoning as a result of self-administration of drugs containing high levels of Mn. In a follow-up study, one of these authors and coworkers measured the brain Mn concentrations in 14 patients with Alzheimer's disease and in 33 non-demented individuals of various ages (Markesbery et al. 1984). They found no significant differences in Mn levels between AD patients and the controls and they found no increase of brain Mn levels with age, leading them to conclude that the brain has an efficient homeostatic mechanism regulating Mn concentrations. Yamada et al. (1986) measured the concentration of Mn in the brain of a 52-year old man suffering from chronic manganese poisoning and found no significant difference in either the concentration or the distribution of manganese in the brain compared to controls. Borit et al. (1975 as cited in Bleecker 1988) found elevated Mn levels in the brains of patients afflicted with a disease known as striatonigral degeneration. Taken together, the human evidence, while limited, does not support any conclusions regarding correlations between levels of Mn in the brain and either exposure or disease.

Studies in experimental animals provide no support for the hypothesis that exposure to Mn by inhalation leads to higher levels of Mn in the brain than does exposure by other routes. Mouri (1973, as cited in Cooper 1984)) compared the absorption and distribution of MnO_2 dust after oral and inhalation exposure in mice. Mice were exposed to air concentrations of 8.91 and 5.55 mg/m^3 Mn for 2 hours per day for 8 and 15 days respectively. Levels of Mn in various tissues were compared to those in tissues of mice receiving comparable oral intakes of Mn. While Mn levels in the lung, trachea, and G.I. tract were much higher in the mice exposed by inhalation than in mice exposed orally, Mn levels in other tissues were only slightly higher in mice exposed by inhalation. Thus, the ratio of Mn concentrations in the brain for inhalation and orally exposed mice was 1.3. It appears from these results that mice are able to regulate body burdens of Mn regardless of the route of exposure. It should be noted that the air concentrations to which these mice were exposed were in excess of the current ACGIH TLV for Mn dust and compounds and

exceed the upper limit estimate of airborne Mn concentrations that might be associated with the use of MMT in gasoline by a factor of more than 50,000.

Morganti et al. (1985) exposed young adult male mice to MnO_2 dust for 7 hours per day, 5 days per week. The exposed animals were observed for signs of overt toxicity and tested for alterations in behavioral and learning performance. Animals were sacrificed at 4-week intervals from 16 to 32 weeks of exposure and 4 weeks after exposure ended and tissue levels of Mn were measured. The concentrations of airborne manganese to which the mice were exposed were measured at 49.1 mg Mn/m^3 for the first 12 weeks and at 85.3 mg Mn/m^3 for weeks 13 through 32. The mass median diameter of the MnO_2 particles was 1.5 um. These concentrations exceed the current ACGIH TLV for manganese dust and compounds by a factor of from 10 to 17. They are also more than 500,000 times higher than the upper limit estimate of ambient airborne manganese concentrations that might possibly result from the use of MMT in unleaded fuel.

Tissue level measurements indicated that manganese levels were significantly higher in all tissues, including the brain, in the exposed animals after 16 weeks of exposure, compared to the sham-exposed control animals. However, from weeks 16 to 32 the manganese levels of all tissues except the liver decreased in the exposed animals and after 32 weeks were not different from tissue levels in the control animals. No gross toxicological effects were observed in the exposed animals but there were subtle differences between exposed and control animals in some, but not all, of the behavioral assessments.

The authors concluded that after an initial increase in tissue manganese levels during the early weeks of exposure, the liver controls the body burden of manganese by concentrating manganese for biliary excretion, and this mechanism serves to regulate tissue levels of manganese even at the excessive airborne exposure levels in this study.

Drown et al. (1986) studied the uptake and elimination of radiolabelled $MnCl_2$ and Mn_3O_4 after intratracheal administration to adult male Sprague-Dawley rats. Their results indicated that the soluble form ($MnCl_2$) was taken up and excreted more rapidly than the insoluble form (Mn_3O_4). Concentrations of Mn in the

brain peaked one day after administration of $MnCl_2$ and 3 days after administration of Mn_3O_4. Levels of Mn in the brain decreased after 2 weeks and fell off sharply after 60 days. These results indicate that Mn absorbed through the lungs is not sequestered irreversibly in the brain.

Newland et al. (1987) studied the clearance of Mn from various body regions in monkeys who were given acute doses of radiolabelled $MnCl_2$ by intratracheal installation. Estimated doses of 0.01 - 0.02 ug of radiolabelled $MnCl_2$ were administered to two female monkeys using an endotracheal tube connected to a respirator for approximately 30 minutes. They then monitored radioactivity in the chest area, the head and in the feces for over a year. They found that radioactivity in the head area decreased at a slower rate than did radioactivity in the chest area, leading them to conclude that "long-term exposure to even low levels of manganese will cause significant accumulation in the brain." This conclusion must be considered to be purely speculative, however, in light of the limited nature of the data that they presented. First, the method used to determine the levels of radioactivity in the various regions of the body could not determine in which organs or tissues the radioactivity was localized within those general areas. Thus, the total radioactivity in the head region may include radiolabelled Mn in the blood, hair, and skin as well as in the brain. Second, the authors failed to differentiate between the kinetics of Mn turnover in the head region and mass balance. Slower turnover of Mn in the head compartment is not sufficient, by itself, to indicate accumulation which can only be determined by measuring the concentrations of total Mn in the brain. Finally, the authors noted that the slower rate of decline of radioactivity in the head area probably reflects replenishment of Mn in that compartment from radiolabelled Mn deposited in other organs. Radioactivity in the abdominal region (liver, kidneys, spleen, G.I. tract) and in the whole body were not determined in this study. The apparent retention of radioactivity in the head may very well have been secondary to the release of radiolabelled Mn from some other storage depot. In view of these limitations and the fact that the study involved the acute administration of a soluble Mn compound to only two animals, it cannot be considered to be evidence for the authors' speculative conclusion that inhalation of Mn may lead to brain accumulation.

Finally, it should be emphasized that in the most relevant animal study conducted to date, there were few signs of toxicity and no histopathological evidence of tissue damage in rats and monkeys exposed by inhalation to up to 1.15 mg/m^3 of Mn_3O_4 prepared by the combustion of MMT 24 hours per day for 9 months (Ulrich et al. 1979). Unfortunately, these authors did not measure Mn concentrations in the brains of exposed animals. However, they did measure Mn concentrations in the blood, lung, liver, kidneys and spleen. The study found that while lung levels were significantly elevated at all exposure levels, Mn levels in the blood and spleen were elevated only at the highest dose, and liver levels were not increased at any dose level. This indicates that Mn levels in the body are homeostatically controlled regardless of the route of exposure.

In conclusion, the data available from studies in experimental animals exposed to concentrations of airborne manganese far higher than any likely to result from the use of MMT as a gasoline additive do not show significantly increased uptake, accumulation (including in the brain), or toxicity when compared to animals exposed to similar doses by the oral route. Only a small fraction of inhaled Mn is absorbed through the lungs with the remainder passing to the G.I. tract or being expelled in the expired air. Body burdens of Mn after exposure by inhalation seem to be controlled by the same mechanism (primarily increased biliary excretion in the liver) that serves to control Mn body burden after exposure by any other route.

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SYSTEMS APPLICATIONS INC.

ATTACHMENT 2

MEMORANDUM

TO: Ethyl Corporation

FROM: Lyle R. Chinkin
Ralph L. Roberson

DATE: October 17, 1990

SUBJECT: Estimate of Maximum, Short-Term Ambient Air Manganese Concentrations

The purpose of this memorandum is to describe our analysis for estimating potential short-term, maximum ambient air concentrations of manganese (Mn) in a typical New York City urban setting. The assumed source of Mn for this analysis is that exhausted from mobile sources that burn unleaded gasoline with the Mn-containing additive HiTEC 3000 at a concentration of 1/32 gram Mn per gallon of fuel.

TECHNICAL APPROACH

The proposed approach is straightforward, but relies heavily on several key assumptions, which are listed below.

- Ambient air concentrations of CO (ppm) are due only to mobile sources.
- Ambient CO concentrations are directly proportional to MOBILE4 emission estimates (gm/mi).
- Ambient Mn concentrations are similarly proportional to Mn emission rates.
- Manganese emission rates are proportional to CO emission rates.

Using these assumptions we can estimate ambient Mn concentrations with the following equation:

$$\text{Ambient [Mn]} = \text{Ambient [CO] [Mn (g/mi)] / [(CO (g/mi)]} \quad [1]$$

assuming emission rates for Mn and CO were obtained under similar test conditions (i.e., similar temperatures, speeds and vehicles).

However, the ambient CO concentrations to be used in Equation 1 are not likely to have occurred at the temperature of 75°F used for the Federal Test Procedure (FTP) for which the CO and Mn emission rates are available from test fleet data. Nor is it likely that the actual vehicle fleet which lead to measured ambient CO concentrations in New York City traveled at the average speed of the FTP, or is appropriately reflected in test fleet data. As a result, to obtain emission rates representative of the conditions which might have caused the CO concentration, we propose to modify Equation 1 to account for the more typical New York City urban speeds, winter-time temperatures, and vehicle fleet characteristics. To account for these differences Equation 1 is modified as shown below and becomes:

$$\text{Ambient [Mn]} = \text{Ambient } [CO]_{winter} \, (Mn_{test}/CO_{test@FTP}) \, (CO_{test@FTP}) \div (CO_{NY,urban,speed,winter}) \quad [2]$$

Note that the term ($CO_{test@FTP}$) appears in both the numerator and denominator of Equation 2 and thus cancels out. To estimate ambient Mn

concentrations, we need: winter-time ambient CO concentrations, Mn emissions (gm/mi) and CO emissions (gm/mi) for typical New York City urban speeds, winter-time temperatures, and vehicle fleet characteristics.

COMPUTATIONS AND DATA ANALYSIS

First, we use MOBILE4 to estimate average CO emissions (gm/mi) for a New York City fleet of mobile sources. We run MOBILE4 for average January temperatures (i.e., 31°F) and for 7.1 miles per hour (mph) to represent the New York City driving cycle (NYCC). For these conditions, the fleet average CO emissions calculated by MOBILE4 is 77 gm/mi.

We obtain ambient CO concentrations from EPA's Aerometric Information Retrieval System (AIRS). We have CO data from an AIRS Quick Look Report for New York City for four monitoring sites for three years: 1983, 1984, and 1985. We use 1983, 1984, and 1985 data because they are the most current CO data in our files. However, we note that the most recent EPA Trends Report* shows a 16 percent decrease in second highest non-overlapping 8-hour CO concentrations between 1984 and 1988. Moreover, the Trends Report states that CO emissions from transportation sources decreased 19 percent during this 5-year period. Thus, we believe that 1983-1985 data provide a conservative estimate of current maximum hourly CO concentrations.

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* National Air Quality and Emissions Trends Report, 1988, U. S. Environmental Protection Agency, Research Triangle Park, N.C., EPA-450/4-90-002, March 1990.

The Quick Look Report provides the maximum hourly CO concentrations (mg/m^3) for each monitoring site and for each year. Averaging across years, we compute the following maximum hourly CO concentrations for the four New York City monitoring sites: 22, 24, 26, and 31 mg/m^3. For our analysis, we use a maximum hourly ambient CO concentration of 25 mg/m^3 (i.e., the average across the four monitoring sites is 25.7 mg/m^3).

Next, we compute an estimate of Mn emissions (gm/mi). To compute Mn emissions we use the results of emission tests completed by EPA August-October 1990. We have Mn test results for several different vehicles, different mileages, and the NYCC driving cycle. For each Mn determination, we compute Mn emissions as follows. We begin with 1/32 gram per gallon of Mn added to the fuel and divide by the fuel economy (mi/gal) as reported for each test. This computation yields Mn input in the units of grams/mile. Then, we multiply Mn input by the percent of Mn emitted as reported on the EPA data sheets. This computation provides an estimate of Mn emissions in the units of grams/mile.

Lastly, we solve Equation 2 with a maximum average hourly CO concentration (25 mg/m^3), the MOBILE4 CO emission rate (77 gm/mi), and the computed Mn emission rates for the NYCC driving cycle to determine potential maximum hourly ambient Mn concentrations.

RESULTS AND DISCUSSION

Table 1 presents our estimates of maximum hourly ambient Mn concentrations based on eight vehicles exposed only to Mn from fuel containing HiTEC 3000. The results indicate the average maximum hourly Mn ambient concentration to be 0.20 $\mu g/m^3$. In computing the average maximum hourly Mn concentration, each vehicle is weighted equally. That is, if we have three Mn determinations for an individual vehicle (e.g., EPA Vehicle ID 0051), those three determinations are averaged prior to computing average concentrations for all vehicles.

TABLE 1. EMISSION TEST RESULTS AND ESTIMATED MAXIMUM HOURLY AMBIENT Mn CONCENTRATIONS FOR VEHICLES USING HiTEC 3000

Vehicle ID	Test Cycle	Mileage (mpg)	Mn (%)	Mn (g/mi)	CO (g/mi)	Ambient CO (mg/m^3)	Ambient Mn ($\mu g/m^3$)
BUICK 2.8L	NYCC	11.5	34.9	9.48E-04	77	25	0.31
BUICK 2.5L	NYCC	13.0	25.0	6.01E-04	77	25	0.20
FORD CROWN VICTORIA	NYCC	9.5	30.1	9.90E-04	77	25	0.32
SUNBIRD 2.0L	NYCC	13.2	20.9	4.95E-04	77	25	0.16
	NYCC	12.1	8.9	2.30E-04	77	25	0.07
	Average			3.63E-04			0.12
DODGE DYNASTY 3.3L	NYCC	10.7	8.2	2.39E-04	77	25	0.08
	NYCC	10.7	23.1	6.75E-04	77	25	0.22
	NYCC	10.6	19.2	5.66E-04	77	25	0.18
	Average			4.93E-04			0.16
GM LIGHT DUTY TRUCK	NYCC	10.1	24.1	7.46E-04	77	25	0.24
	NYCC	9.8	12.8	4.08E-04	77	25	0.13
	Average			5.77E-04			0.19
CAVALIER 2.2L	NYCC	13.5	8.3	1.92E-04	77	25	0.06
	NYCC	13.3	18.2	4.28E-04	77	25	0.14
	Average			3.10E-04			0.10
TAURUS 3.3L	NYCC	10.2	18.7	5.73E-04	77	25	0.19
						Fleet Average	0.20
						Standard Deviation	0.08

ATTACHMENT 3

E.H. PECHAN & ASSOCIATES, INC.

3537 Hampstead Way
Springfield, VA 22151

642-1120

Facsimile (703) 642-1035

October 10, 1990

Mr. Ben Fort
Ethyl Corporation
451 Florida Blvd.
Baton Rouge, LA 70801

Subject: Parking Garage Modeling

Dear Mr. Fort:

We have examined the ambient concentrations of carbon monoxide (CO) and manganese oxide (MnO) that might occur in a typical parking garage if all of the passenger cars in the garages were using gasoline with the Ethyl Corporation fuel additive. Information provided in a paper by Ingalls and Garbe (1982) was used to estimate ambient CO and MnO concentrations for a typical exposure case. The typical garage is an above ground garage, with open sides for natural ventilation and a capacity of 400 to 500 cars.

As described in the Ingalls and Garbe paper, the typical exposure situation was chosen as the fourth level of the San Antonio, Texas, Convention Center parking garage following an event at the adjacent convention center. This garage is a five level, above ground, open structure with parking for as many as 461 cars. It has natural ventilation only. With a wind speed of seven miles per hour, the ventilation rate for the fourth level is 308,000 cubic feet per minute. The total volume on this level is 356,000 cubic feet. There are 17 cars active on the fourth level at all times during the emptying process. A ventilation factor of 0.4 was used for the vehicle exhaust equation.

This particular situation was modeled because Ethyl Corporation requested that we examine the typical parking garage. As pointed out by Ingalls (1985), 90 percent of garages are naturally ventilated. In the Ingalls report, the same typical garage scenario was used as the mode, or the situation corresponding to the greatest frequency, in an attempt to estimate personal exposures.

The above conditions were used to estimate CO and MnO concentrations for the typical situation as shown in Table 1. Emission estimates were based on the results of manganese particulate testing completed by EPA during August through October 1990. Two different emission rates were used for each scenario because the emissions test data were bimodally distributed. For CO, 0.496 grams per minute is representative of the low end of the emissions measurement range, while 3.46 grams per minute represents the high end of the range. MnO emissions were estimated as 0.0128% of CO for the low end emission estimates and 0.0036% of CO for the high emission estimate, which gives a narrower range of MnO emission estimates than that used for modeling CO.

We understand that all of the emissions data are from tests performed using the New York City cycle, which has a low average speed and a substantial idle emissions component, but may contain more accelerations than would occur in a garage. Because particulate emissions are generally highest during accelerations, it may be that MnO emission rates would be much lower if idle emission rates alone were measured. This would result in lower peak manganese concentrations. It is impossible to determine the extent of this bias from the available data, however.

Sincerely,

James H. Wilson

jth

Table 1

Parking Garage Modeling Analysis

Estimated Ambient Concentrations

Carbon Monoxide (ppm)		Manganese oxide ($\mu g/m^3$)	
0.496 g/min Emis. Rate	3.46 g/min Emis. Rate	Low Emis. Rate	High Emis. Rate
1.6	11.6	0.25	0.49

Concentration estimates assume a five minute exposure. Units reporting follows the convention of using parts per million by volume to express the concentration of gaseous contaminants, and micrograms per cubic meter to express the concentration of particulate contaminants.

References

Ingalls, 1985: Melvin N. Ingalls, "Improved Mobile Source Pollutant Estimation," EPA-450/3-85-002, Southwest Research Institute, San Antonio, TX, March 1985.

Ingalls and Garbe, 1982: Melvin N. Ingalls and Robert J. Garbe, "Ambient Pollutant Concentrations from Mobile Sources in Microscale Situations", SAE Technical Paper Series (820787), Passenger Car Meeting, Troy, MI, June 7-10, 1982.

ATTACHMENT 4

Further Estimations of Ambient Levels of Manganese From Use of HiTEC 3000

[illegible] of estimating the maximum ambient concentration of Manganese from use of HiTEC 3000 is to utilize the vast amount of information on ambient levels of lead from combustion of gasoline. The use of a gaseous emission, such as carbon monoxide, as a tracer for manganese, a particulate, is inappropriate because of differences in removal mechanisms for gases and particulates. Particulates may be removed from the atmosphere by (1) dry deposition, which includes the mechanisms of diffusion, interception, impaction, and sedimentation and (2) wet deposition, which includes washout or rainout. Lynam (1972) summarized lead - carbon monoxide relationships from data reported in the scientific literature. The summary is reproduced in Table I and shows large variations in CO - Pb ratios based on type of area, mode of vehicle operation, distance from traffic, etc. In a well controlled field investigation, Lynam (1972) measured CO and Pb at seven sampling sites positioned at a right angle to and at intervals of 20, 40, 80, 160, 320, and 640 feet from the edge of a major interstate highway in Cincinnati, Ohio. The maximum atmospheric concentration of lead measured in this study was 15.08 ug/m^3, for a short sampling period of 30 minutes, at the 20-foot downwind station with a traffic volume of 4844 vehicles/0.5 hour. The maximum CO level was 10.5 ppm at the same station with a traffic volume of 4682 vehicles/0.5 hour.

The relation between CO and Pb was highly dependent on sampling location with respect to the highway. Lead and carbon monoxide were highly correlated, especially at distances close to the expressway, but the regression of Pb on CO varied with the distance of the sampling site from the expressway. The carbon monoxide lead ratios are attached as Table I-5. The author concluded that atmospheric lead levels cannot be accurately predicted from atmospheric CO levels or conversely. The use of CO to predict Mn particulate levels will result in overestimating the Mn levels. However, lead data can be used to estimate Mn concentrations.

One of the best studies for estimating the maximum potential exposure [illegible] is the study of AZAR etal, who measured by the personal [illegible] 24 hour average exposure levels of lead [illegible] population [illegible] The group with the highest exposure levels [illegible] taxi cab drivers in Los Angeles who averaged 6.10 ug Pb/m^3. It is assumed that gasoline - related manganese emissions would behave in a similar fashion to lead emissions from combustion of fuel. Lead in gasoline averaged approximately 2.5 g/gal at the time of the AZAR study. Thus, the ratio of manganese to lead should be 0.03125/2.5 or 1/80. Therefore, the 24-hour average exposure to manganese would be 6.10 ug/m^3 ÷ 80 = 0.076 ug/m^3. We believe this would represent the most extreme potential exposure because it assumes manganese is emitted at the same rate as lead, and the taxi cab driver spends most of his day in automobile traffic.

Other data can be used to calculate the potential 24-Hour, Time-Weighted Average exposure of the general population to manganese if HiTEC 3000 is used.

Another case assumes that gasoline-related manganese emissions would behave in a similar fashion to lead emissions from combusted fuel. The highest exposure would be to persons in automobiles during heavy traffic situations. Lead levels of 40 ug/m^3 in cars during peak traffic periods have been reported at about the time gasoline lead usage was at a maximum (Konopinski and Upham, 1967). Lead averaged approximately 2.5 g/gal at that time; therefore, the level of manganese inside cars should be 0.03125/2.5 or 1/80th as much. Therefore, the levels in cars could reach 40 ug/m^3 ÷ 80 or 0.5 ug/m^3. We can further assume that general urban airborne levels of manganese would be about the same as those found in Toronto (.04 - .05 ug/m^3) where HiTEC 3000 is used at twice the concentration proposed for use in the U.S. To be conservative, we will calculate based on .05 ug/m^3. We can also assume that levels of manganese indoors would be about 60% of those outdoors based on a lead model (Davies et al, 1987).

A typical adult may spend about 10 hours/day inside at home, 8 hours at work (assume exposure same as general outdoors), 4 hours in outdoor recreation and 2 hours/day commuting in heavy traffic. The [illegible] rates would give a "worst-case" estimate. The 24-Hour Time-Weighted-Average exposure would be:

$$\frac{\underset{\text{(At Home)}}{10 \times 0.03} + \underset{\text{(At Work)}}{8 \times 0.05} + \underset{\text{(Recreation)}}{4 \times 0.05} + \underset{\text{(Commuting)}}{2 \times 0.5}}{24}$$

or 0.079 ug/m^3. This is less than 8% of the World Health Organization guideline of 1.0 ug/m^3 which is thought to be protective of even the most sensitive population group.

A third case is based on ambient carbon monoxide concentrations versus concentrations inside the vehicle. Brice and Roesler (1966) found the 1-hour CO concentration inside vehicles to be about 4 times the ambient levels. Using this as the basis for the calculations gives the following results:

$$\frac{\underset{\text{(At Home)}}{10 \times 0.03} + \underset{\text{(At Work)}}{8 \times 0.05} + \underset{\text{(Recreation)}}{4 \times 0.05} + \underset{\text{(Commuting)}}{2 \times (0.05 \times 4)}}{24}$$

or 0.054 ug/m^3 as a 24-hour Time Weighted Average. This is slightly over 5% of the WHO guideline.

A fourth case incorporates levels of manganese in parking garages predicted from calculations by Pechan and Associates based upon carbon monoxide emissions. Let us assume that 20 minutes of the commuting time is spent in a parking garage with the automobiles in the garage evenly divided between those with low emission rates and high emission rates. Then the 24-Hour Average Exposure for the typical case would be as follows:

Typical Case

(at home) (at work) (recreation) (commuting)

$$\frac{10 \times [illegible] + 8 \times 0.05 + 4 \times 0.05 + (1.67 \times 0.2 + 0.33 \times [illegible]}{24}$$

$= 0.057\ mg/M^3$

In the "Typical" case, the 24-Hour average exposure is not significantly different from the model which excludes parking garage exposures. Because slow moving, stop and go traffic generates unusually high CO emissions with low manganese emissions, and since the Pechan calculations are based on a CO:Mn emissions ratio, their predicted values are at the extreme limit and more than likely would never be reached.

There is no realistic scenario that could be developed which could cause the very conservative WHO guideline to be exceeded on a 24-Hour Time Weighted Average. All cases show that the levels of Mn exposure are very low (less than 0.08 ug/m^3) even for taxi cab drivers in Los Angeles.

References:

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Brice, R.M. and Roesler, J.F., The Exposure to Carbon Monoxide of Occupants of Vehicles Moving in Heavy Traffic, Air Pollution Control Assoc. Journal, 16, 597-600 (1966).

Davies, D.J.A., Thornton, I, Watt, J.M., Culbard, E.B., Harvey, P.G., Delves, H.T., Sherlock, J.C., Smart, G.A., Thomas, J.F.A. and Quinn, M.J., Relationship between Blood Lead and Lead Intake in Two Year Old Urban Children in the UK, Presented at Heavy Metals in the Environment, New Orleans, 1987.

Konopinski, V.J. and Upham, J.B. Commuter Exposure to Atmospheric Lead, Arch. Environmental Health, 14 (1967) 589-593.

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TABLE 1

SUMMARY OF LEAD-CARBON MONOXIDE RELATIONSHIP DATA

LOCATION	AREA TYPE	DISTANCE FROM NEAREST TRAFFIC	NO. OF SAMPLES	SAMPLING TIMES	CORRELATION COEFFICIENT	REGRESSION EQUATION	AMOUNT OF LEAD (µg/m³) FOR 5, 10, & 20 PPM CO, FROM REGRESSION EQUATION			REFERENCE
							5 PPM	10 PPM	20 PPM	
P.H.S. Three-City Study:										26
Downtown Los Angeles	Commercial - 80' above ground	Site #1	3 month period	48 & 72 hours	0.79	Pb = -9.84 + 1.12 CO	Neg.	1.36	12.56	
West Los Angeles	Commercial - 5' above ground	Site #2	3 month period	48 & 72 hours	0.81	Pb = -5.97 + 0.82 CO	Neg.	2.23	10.43	
Pasadena	Residential - 10' above ground	Site #4	3 month period	48 & 72 hours	0.81	Pb = -6.99 + 0.82 CO	Neg.	1.21	9.41	
General Motors Study of Detroit, Los Angeles, & New York:										25
All Sites - All Data	Freeway, Commercial, & Residential	As close as 2 meters - usually less than 20 meters	111	Minimum of 12 hrs. Some 24 hrs. Others longer	0.80	Pb = 0.95 + 0.81 CO	5	9.05	17.15	
All Sites - Day	Freeway, Commercial, & Residential	As close as 2 meters - usually less than 20 meters	40	Minimum of 12 hrs. Some 24 hrs. Others longer	0.72	Pb = 1.65 + 0.71 CO	5.2	8.75	15.85	
All Sites - Night	Freeway, Commercial, & Residential	As close as 2 meters - usually less than 20 meters	40	Minimum of 12 hrs. Some 24 hrs. Others longer	0.83	Pb = 0.46 + 0.91 CO	5.0	9.56	18.66	
Freeway Sites	Freeway	2, 5, 75 meters	31	Minimum of 12 hrs. Some 24 hrs. Others longer	0.94	Pb = -0.42 + 1.42 CO	6.6	13.78	27.98	
Commercial Sites	Commercial	2,3,4, & 5 meters	49	Minimum of 12 hrs. Some 24 hrs. Others longer	0.88	Pb = 1.49 + 0.66 CO	4.79	8.1	14.69	
Residential Sites	Residential	2,6,20,150 meters	31	Minimum of 12 hrs. Some 24 hrs. Others longer	0.83	Pb = -0.85 + 0.98 CO	4.05	8.95	18.75	
All Detroit Data	Freeway, Commercial, & Residential	5,5,150 meters	13	*	0.87	Pb = -0.93 + 1.47 CO	6.42	13.77	28.47	
Lodge Ford Freeway Int	Freeway	5 meters	8	*	0.82	Pb = 2.40 + 1.03 CO	7.55	12.70	23.0	
All New York Data	Freeway-Commercial	2,3, and 75 meters	47	*	0.90	Pb = 1.26 + 0.63 CO	4.41	7.56	13.86	
Herald Square	Commercial	2 meters	15	*	0.84	Pb = 2.90 + 0.53 CO	5.55	8.20	13.50	
Columbus Circle	Commercial	3 meters	12	*	0.81	Pb = 2.50 + 0.39 CO	4.45	6.40	10.3	
Queens Expy. Int.	Freeway	75 meters from freeway	12	*	0.92	Pb = 0.38 + 0.90 CO	4.88	9.38	18.38	
All Los Angeles Data	Commercial, Freeway, & Residential	4,5,6,20 meters	51	*	0.84	Pb = -0.80 + 1.13 CO	4.85	10.5	21.8	
Pico Boulevard	Commercial	4 meters	18	*	0.96	Pb = 0.06 + 0.90 CO	4.56	9.06	18.06	
Harbor-Santa Monica Freeway Interchange	Freeway	5 meters	11	*	0.96	Pb = 1.55 + 1.28 CO	7.95	14.35	27.15	
Santa Monica	Residential	6 meters	11	*	0.88	Pb = -1.58 + 1.09 CO	3.87	9.32	20.22	
Monrovia	Residential	20 meters	11	*	0.75	Pb = -1.83 + 1.15 CO	3.92	9.67	21.17	
1967 CO data from CAMP Station Sites & Lead data from NASN data of same city: 3.3 to 7.5 CO (PPM) / Pb (µg/m³)							0.67 to 1.51	1.33 to 3.03	2.67 to 6.06	27
Statewide Air Pollution Research Center of Univ. of California, Riverside	Unknown	Unknown	90 (over 15 day period)	4 hrs.	0.76	none given	-	-	-	28
110 East 45th Street New York City	Commercial	6 meters above street level and app. 3 meters in from curb	812 (10 weeks)	2 hrs.	unknown	none given	-	-	-	29
Northeastern U. S. City (only identification)	Unknown		Over 3 week period	Unknown	Unknown	Pb = 0.516 + 0.248 CO	1.84	3.20	5.88	30

TABLE 1-5

[illegible]

Experiment No.	CO (ppm)/[illegible] Filter						
	20' Station	[illegible] Station	[illegible] Station	160' Station	300' Station	[illegible]	[illegible]
1	0.54	-	0.56	-	1.01	1.30	-
2	0.69	1.0	0.97	-	-	0.83	5.11
3	0.59	-	0.67	0.82	1.03	1.08	2.74
4	1.07	4.06	1.09	1.56	1.35	2.21	5.15
5	1.00	0.80	0.78	1.06	1.40	1.74	3.08
6	0.47	0.68	0.80	1.27	1.21	2.04	7.48
7	0.65	0.95	0.95	2.29	3.80	0.90	-
8	0.68	-	0.80	0.98	1.00	1.51	8.03
9	-	0.66	0.73	1.35	-	2.21	7.63
10	-	0.67	0.66	-	-	2.43	10.30
11	0.64	0.67	0.63	1.09	1.19	1.52	19.40
12	0.58	0.51	0.43	0.88	0.96	1.49	8.16
13	0.82	0.66	0.65	1.12	1.60	1.00	8.02
14	0.60	0.45	0.53	1.02	1.11	1.85	4.53
15	0.62	0.65	0.64	1.03	1.27	-	4.63
16	0.32	0.36	0.31	0.55	0.51	0.63	3.38
17	0.41	0.46	0.46	0.65	0.73	1.10	4.74
18	0.35	0.39	0.40	0.79	0.73	1.01	9.06
19	0.40	0.44	0.47	0.94	0.82	1.37	6.37
20	-	-	-	0.50	0.30	0.64	4.27
21	-	0.58	0.57	0.80	0.81	0.80	2.51
22	0.59	0.85	0.71	0.83	0.60	1.00	2.63
23	0.54	0.57	0.56	0.75	0.70	0.78	2.82
24	0.67	0.57	0.50	0.82	0.83	1.19	-
25	-	0.61	0.61	0.76	0.96	-	-
26	0.89	0.72	-	0.80	0.96	0.93	1.49
27	-	0.66	-	1.04	1.41	-	1.43
Mean:	0.62	0.65	0.65	0.98	1.10	1.32	5.78
S.D.	0.17	0.17	0.19	0.37	0.65	0.53	3.90
N	21	23	24	24	24	24	23