ROADS AND HIGHWAYS COMMITTEE MEETING - FEBRUARY 10, 1972

The Roads and Highways Committee met in room 510 S at 2:45 P. M. on February 10, 1972. Chairman Dierdorff called the meeting to order and all members were present except Allison, Gray and Ossmann.

Conferees were: Delegation from Southeast Kansas - William R. Hagman, Pittsburg; R. H. Hartley, Baxter Springs; George Nettels, Pittsburg; Paul Armstrong, Columbus; D. J. Saia, Frontenac; Ray Shepherd, Fort Scott; on House Bill 1666 - Representative John Peterson; Dr. Albert Burgstahler, Professor of Organic Chemistry at Kansas University; Jim Heidebrecht, Kansas Tuberculosis Association' Robert C. Butler, Manager, Environmental Affairs for the Petroleum Chemicals Division of the Du Pont Company; Amos Kramer, Executive Director of the Kansas Petroleum Industry; Don Bell of KOMA.

A roster of others who registered their attendance is attached.

Dr. Hughes introduced the group from Southeast Kansas and each of them complemented the committee on the passage of Senate Bill 137, which would permit the building of roads in Kansas. They stressed that their concern for roads is bi-partisan - that their big interest is the 69 corridor and how it would help the economic growth of Kansas. They would prefer to be on the freeway system but would rather have roads sooner than years in the future - they would like to have them to enjoy in their lifetime and would be willing to go the toll route in order to get the roads.

HOUSE BILL 1666 - AN ACT relating to taxation of motor-vehicle fuels, special fuels and LP gas fuel used in motor vehicles; providing rates of tax; authorizing allowances for certain losses; providing for refunds in certain cases;...

Mr. Peterson explained the bill. The purpose of the bill is to give users of non-leaded gasoline an incentive in the form of a tax break and to fight pollution. The tax would be increased on motor vehicle fuel from 7¢ to 8¢ per gallon, and in the case of fuel containing less than three-tenths gram of lead per gallon reduces the tax from 7¢ to 5¢ per gallon. On special fuels, the tax would be increased from 8¢ to 9¢ per gallon and in the case of special fuels containing less than three-tenths gram of lead per gallon, the tax rate would be reduced from 8¢ to 6¢ per gallon.

A copy of Albert Burgstahler's remarks is attached - Exhibit I.

Amos Kramer said the number of gallons of non-lead gasoline purchased by the motoring public in Kansas is small compared to the number of gallons that are consumed. To the best of his knowledge there is only one company selling on a limited basis. Some others might be but it would be in the .5 category. Until the automobile manufacturers and the government come to an agreement on the requirements for conversion, there won't be much demand. In his opinion, the tax increase would be unnecessary.

A copy of Robert C. Butler's remarks is attached - Exhibit II.

Don Bell just appeared in opposition to the bill.

Mr. Ratner explained the bill the sub-committee worked out on installation of safety devices at certain railroad grade crossings designated by the state corporation commission; and providing for the apportionment and payment of the costs thereof.

Mr. Ratner made a motion, second by Dr. Hughes, that the bill be introduced as a committee bill and referred back. Motion carried.

The Chairman announced to the committee that on Monday, February 10th, we could consider House Bill 2082 on Liquefied Petroleum.

The meeting was adjourned.

Fran Stafford, Recording Secretary

APPROVED:

ARDEN DIERDORFF, CHAIRMAN
February 10, 1972

2-10-72

ROADS AND HIGHWAYS COMMITTEE

GUESTS

NAME	ADDRESS	FIRM OR CORPORATION REPRESENTED
Robert C. Butler	Wilmington, Del.	DuPont Co
Willard K. Jantz	9408 Roe Prairie Village, Ks	DuPont Co
Amos Kramer	Kansas Petroleum Council	Wichita.
John REALS	Wichita, Ks.	Self.
Wm. R. Hagman	Pittsburg	Self
R. V. Hadley	Baxter Springs	self.
George Nettels	Pittsburg	Self.
Earl Armstrong	Columbus, Ks.	self.
D J Saia	Frontenac Kans	Self
Ray Shepherd	Fort Scott, Ks	Kansas Oil Marketers Assn
Neil Hutton	Wichita Kansas	Kansas Oil Marketers Assn
Donald A. Bell	Wichita, Kansas	"
Walter McNutt	Toheka, Kans	
Walter W. Kicker	Topeka	Ks. Motor Carriers Ass'n.
David Dillon	Hutchinson	
Ray Ellis	Topeka	Kansas Association of Commerce and Industry
James Heidebrecht	Topeka	Kan TB & Health Assn
Mary Ann Bradford	Topeka	League of Women Voters

566 | 1666
Fiscal Note | Bill No.
1972 Session
January 25, 1972

The Honorable Arden Dierdorff, Chairman
Committee on Roads and Highways
House of Representatives
Third Floor, Statehouse

Dear Mr. Dierdorff:

SUBJECT: Fiscal Note for House Bill No. 1666 by Mr. Peterson

In accordance with K.S.A. 75-3715a, the following fiscal note concerning House Bill No. 1666 is respectfully submitted to your committee.

Effective July 1, 1972, House Bill No. 1666 increases the tax on motor vehicle fuel from 7¢ to 8¢ per gallon, and in the case of fuel containing less than three-tenths gram of lead per gallon reduces the tax from 7¢ to 5¢ per gallon. On special fuels, the tax would be increased from 8¢ to 9¢ per gallon and in the case of special fuels containing less than three-tenths gram of lead per gallon, the tax rate would be reduced from 8¢ to 6¢ per gallon. The bill also provides for an inventory tax on all motor vehicle and special fuels owned at 12:01 a.m., July 1, 1972, at the rate of 1¢ per gallon except that motor vehicle and special fuels containing less than three-tenths gram of lead per gallon are to be subject to a refund of 2¢ per gallon.

With regard to the revenue impact of the provisions of this bill, it would depend on the extent to which consumers desire to purchase "lead-free" products. The Department of Revenue notes that based on current estimates, increasing the tax on motor vehicle fuel from 7¢ to 8¢ per gallon would provide for increased revenues totaling $12,276,000; however, if one-third of the gasoline sold was "lead-free", the 5¢ tax per gallon would offset the increase on leaded gas. With regard to the tax rates on special fuels, the Department of Revenue notes that all special fuels meet the "lead-free" requirement and that based on current estimated receipts, the reduced tax rates would provide for a direct reduction of approximately $2,582,000 in FY 1973.

In the case of inventory tax, receipts are estimated to total approximately $300,000. The inventory of "lead-free" gasoline is thought to be negligible presently and would offset only a small portion of that amount of inventory receipts.

Distribution of motor vehicle fuel and special fuel receipts are 2% to the State General Fund; after refunds, $2,500,000 to the County Equalization and Adjustment Fund; and of the balance, 51% to the State Highway Fund, 14% to the State Freeway Fund, and 35% to the Special City and County Highway Fund. The net decrease in revenues from the special fuels tax decrease would reduce FY 1973 State General Fund receipts by approximately $52,000. The State General Fund share of the inventory tax would total approximately $6,000.

James W. Bibb

James W. Bibb
Budget Director

JWB:AKS:mad

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(Thursday, Feb. 10, 1972)

Air Pollution from Lead in Gasoline

(Summary of testimony prepared for hearing on Kansas H.B. No. 1666)

by

Albert W. Burgstahler, Ph.D.
Professor of Chemistry
The University of Kansas
Lawrence, Kansas 66044

TODAY, in the United States, about 300,000 tons of lead or 25 per cent of the total annual consumption are used each year in the production of anti-knock agents (mainly lead tetraethyl) for motor fuel additives. The amount of lead present per gallon of gasoline treated ranges from about 2.3 grams for regular fuel (92-94 octane) to 2.7 grams for premium fuel (97-100 octane).

Lead is present in the air from various sources, including coal burning, volcanic emissions, and manufacturing processes. Generally, however, these sources now constitute less than 10 per cent of the 180,000 tons of lead released each year in the U.S. from auto and truck exhausts. There exists a close correlation between air-lead levels in major cities and the total gasoline consumption in them.

In 16 percent of U.S. cities lead in the air has been reported to have values ranging from 1.6 to 9.8 micrograms per cubic meter ($\mu g/m^3$). In one percent it ranged from 9.8 to 17.5 $\mu g/m^3$ for varying time periods. In rural air the lead content ordinarily averages only 0.2 to 0.7 $\mu g/m^3$. Standards for maximum average levels in Czechoslovakia and USSR are set at 0.7 $\mu g/m^3$; 2.0 $\mu g/m^3$ in Germany; and 5.0 $\mu g/m^3$ in Pennsylvania (for a 30-day period).

Dusts in city streets often contains as much as 1 per cent of lead, whereas the average content in soils is only 16 parts per million (0.0016%). Foods grown along highways and freeways show abnormally high lead content, usually in the ppm range, whereas "normal" values are of the order of hundredths of a part per million.

The daily intake of lead from food and water is estimated to average 300 μg (0.3 milligram), of which only 5 to 10 percent (15 to 30 μg) is actually absorbed into the body. (The total body content of lead in adults in the U.S. is now estimated to be about 100 to 200 milligrams, a figure which is about 10 times that present in pre-modern times, and about double that present in non-contaminated parts of the world.)

The daily (24-hour) intake of lead by respiration is quite variable. If the figure of 20 cubic meters (m^3) is taken as the quantity of air breathed, and 50 percent of the air-borne lead is absorbed in breathing, then even with only 1.0 $\mu g/m^3$ of lead in the air the absorbable intake is 0.5 X 20 or 10 μg. This is already a significant fraction of the larger intake estimated from food and water.

However, when air-lead levels reach 5 $\mu g/m^3$, as commonly happens in larger cities and along busy highways and freeways, the intake of lead from air easily reaches 50 μg per day, or 2 to 3 times the amount ingested from food and water.

The toxic level (causing definite bodily harm) for lead ingested from food and water has been estimated to be in the range of 2 to 3 milligrams per day for adults (10 times the estimated present level of ingestion) and 0.5 to 1.0 milligram (500 to 1,000 μg) per day for children. Taking the lower last figure and assuming 10 per cent absorption, this means that a total daily absorbed dose of 50 μg would be in the toxic range for children.

If an active child (breathing 20 cubic meters of air per day) lives in an area where the average air-lead level is 5 $\mu g/m^3$ (which is now quite common in busy traffice areas),his average daily ingestion of absorbed lead from air alone would be 50 μg (50% of 5 X 20). This, of course, is in the admittedly toxic range and may well help to account for the fact that an estimated 5 percent of U.S. city children are believed to be suffering from varying degrees of lead poisoning. (The principal source of much urban lead poisoning is from lead in articles consumed, including lead-contaminated dusts, etc., as well as lead-containing paints.)

Evidence for toxic effects on populations exposed to air-borne lead from automobile traffice has been presented and is cited in the references given below (see esp. ref. 5).

Brief Bibliography

1. H. A. Schroeder, "A Sensible Look at Air Pollution by Metals," Arch. Environ. Health, 21:798-806 (Dec. 1970); cf. H. A. Schroeder and I. H. Tipton, "Human Body Burden of Lead," ibid., 17:965-978 (Dec. 1968).

2. R. Gillette, "Lead Poisoning: Zoo Animals May Be the First Victims," Science, 173:130-131 (9 July 1971).

3. Editorial, "Lead in Air," Brit. Med. Jour., 18 Sept., 1971, 653-654.

4. Articles: "The Air of Poverty," "Half Step Forward," and "Timetable for Lead," in Environment, 13, June 1971.

5. D. Bryce-Smith, "Lead Pollution--a growing hazard to public health," Chemistry in Britain, 7(2):54-56 (Feb. 1971); "Lead Pollution from Petrol," ibid., 7(7):284-286 (July 1971) in reply to A. L. Mills, "Lead in the Environment," ibid., 7(4):160-162 (April 1971).

2-10-72

BRITISH MEDICAL JOURNAL

LONDON SATURDAY 18 SEPTEMBER 1971

Lead in Air

There is no disputing the fact that the air many people breathe daily contains measurable quantities of lead. In fact chemists[1] and geophysicists[2] have claimed that man is so polluting his environment with lead that a serious deterioration in general health, with a rising incidence of mental defect and disorder, can be expected. The blame for this pollution is laid mainly on the use of lead additives in petrol. As this practice is likely to increase unless alternative forms of motor fuel are made available, it needs to be looked at carefully.

During the past 15 years many measurements of it have been made, particularly in the U.S.A. One large project[3] embraced hundreds of air samples in three big cities over a period of one year together with many blood lead determinations in the general population. More recent studies[4] in another U.S. community have shown that the concentration of lead in air appears to be increasing by 5% per annum and that this lead comes from petrol. Data on the lead in air in British cities are sparse but consistent with those from U.S. studies. In Fleet Street, London,[5] in 1964 the concentration during the day was 3·2 $\mu g/m^3$, and in High Street, Warwick,[6] in 1965-6 the levels ranged from 2·8 to 4·4 $\mu g/m^3$ in a big survey. In both London and Warwick the concentration in the air a few hundred yards from the main throughfares was much lower. A similar decline in areas increasingly distant from "thruways" has been noted in the U.S.A.[7] There is therefore no basis for concluding that Britain is entirely different in this respect from the U.S.A., though climatic conditions may result in somewhat greater variations.

The figures available suggest that people in urban communities may have to breathe air containing up to 1·5μg of lead per cubic metre. Respiring 20 m^3 of air each day a person would retain 15 μg of lead (50% of the amount inhaled). From food and water about 30 μg is absorbed, which represents 10% of amount ingested. The inference is commonly drawn from the published data that the blood level of lead in the general population is related to the atmospheric concentrations to which the people are exposed. This generalization provides the main basis for the argument that lead should be removed from petrol, because the combustion of petrol is likely to continue to increase. But what is apt to be ignored is the "flatness" of the correlation between lead in the air and lead in the blood.[8] Thus, if the lead in the air increases by 100 times (0·5 to 50 $\mu g/m^3$), the lead in the blood only slightly more than doubles (from 15 to 35 $\mu g/100$ g). R. A. Kehoe's data suggest a reason for this.[9] Volunteers exposed for 8 hours a day for many weeks to 150 μg of lead per cubic metre of air showed a sharp increase in urinary excretion of lead during the period of exposure to it. The blood level did not rise continuously during exposure, and the urinary level fell after exposure ceased. These observations emphasize the fact that lead is not entirely cumulative. But they also underline our ignorance of the exact significance of blood levels of lead as indices of exposure to it.

Nevertheless, it is important to consider what significance should be attached to blood levels of lead in terms of the danger they may represent. Much anxiety has been expressed, for instance, about the danger to young children from exposure to lead, and there is evidence that excessive ingestion can produce serious immediate and late effects on brain function. While American paediatricians[10] consider a blood lead level of 80 $\mu g/100$ g in children to be an indication that lead encephalopathy is imminent and that chelation therapy should be introduced, such a blood level in adults is considered evidence only of an unnecessary and undesirable degree of exposure but no cause for immediate treatment.[11]

The anaemia due to lead poisoning results in part at least from failure to synthesize enough haemoglobin. The lead interferes with several enzymes, including one (ALA dehydrase) involved in the synthesis of haem for both haemoglobin and the cytochrome pigments essential for many steps of cellular metabolism. The ALA dehydrase remains in the circulating red cells, where it no longer has any recognized function. In this respect it resembles the enzyme acetylcholinesterase, which is also present in red cells but has a recognized function only in the nervous system. The activity of the ALA dehydrase in red cells is inhibited by lead, and the degree of inhibition is directly related to the blood lead levels.[12] So sensitive is the enzyme that a level of lead in the blood as low as 1 $\mu g/100$ g will depress its activity. In the blood it does not matter whether the enzyme functions or not; what matters is the level of ALA dehydrase activity in tissues such as the brain, where the level of haem synthesis may be important. It is impossible to study levels of brain enzyme activity in man, though it has been done in animals.[13] At present, therefore, we do not know what blood lead level in chil-

dren depresses enzyme activity in the brain and jeopardizes its development.

Lead poisoning in children is usually due to excessive ingestion, mainly of lead paint, and the most urgent need at present is to improve the environment of poor homes. But the possibility that lead can harm the developing mammal deserves thorough study. The suckling rat is much more sensitive than the adult to ingested lead, and the biochemical basis of the brain damage produced could be investigated.[14] What is needed is more information on the metabolism of lead in young people and adults at low levels of exposure by ingestion and inhalation, with particular reference to the significance of blood levels. At the same time we ought to know more about the morbidity and mortality of men with a working lifetime of exposure to lead. While mortality from cancer was found to be less than expected, deaths from cerebrovascular disasters were more frequent.[15] At what level of exposure does this risk become indistinguishable from normal?

[1] Bryce-Smith, D., *Chemistry in Britain*, 1971, 7, 54.
[2] Patterson, C. C., *Archives of Environmental Health*, 1965, 11, 344.
[3] Ludwig, J. H., Oiggs, D. R., Hessellberg, H. E., and Maga, J. E., *American Industrial Hygiene Association Journal*, 1965, 26, 270.
[4] Chow, T. J., and Earl, J. L., *Science*, 1970, 169, 577.
[5] Waller, R. E., Commins, B. T., and Lawther, P. J., *British Journal of Industrial Medicine*, 1965, 22, 128.
[6] Bullock, J., and Lewis, W. M., *Atmospheric Environment*, 1968, 2, 517.
[7] Thomas, H. V., Milmore, B. K., Heidbreder, G. A., and Kogan, B. A. *Archives of Environmental Health*, 1967, 15, 695.
[8] Goldsmith, J. R., and Hester, A. C., *Science*, 1967, 158, 132.
[9] Kehoe, R. A., *Archives of Environmental Health*, 1964, 8, 235.
[10] American Academy of Pediatrics Subcommittee, *Pediatrics*, 1969, 44, 291.
[11] Lane, R. E., *et al.*, *British Medical Journal*, 1968, 4, 501.
[12] Hernberg, S., and Nikkanen, J., *Lancet*, 1970, 1, 63.
[13] Millar, J. A., Battistini, V., Cumming, R. L. C., Carswell, F., and Goldsberg, A., *Lancet*, 1970, 2, 695.
[14] Pentschew, A., and Garro, F., *Acta Neuropathologica (Berlin)*, 1966, 6, 266.
[15] Dingwall-Fordyce, I., and Lane, R. E., *British Journal of Industrial Medicine*, 1963, 20, 313.

Bacteria in Cream

Whether thick or thin, heat-treated or raw, clotted or sterilized, a vast amount of commercially produced cream is now consumed in Britain. More than 150 million gallons of milk were used for this purpose in 1970. But except when it is sterilized in retail containers the shelf-life of cream is short—as housewives know only too well—mainly because of its suitability as nutritious food for bacteria.

Though it is usually of excellent bacterial quality when processed, millions of organisms may be present when the cream is consumed. They may include some organisms which survive heat-treatment, but more often they indicate subsequent contamination because of faulty or unavoidable handling during production, distribution, or storage. The bacteria affect the keeping quality of the cream and are ultimately responsible for spoilage, though apparently rarely for disease. Nevertheless, food-poisoning from cream or its products is occasionally observed, and a number of investigations[1] have indeed given cause for concern that pathogens may sometimes be introduced into cream.

It is reassuring therefore to learn from a recent report[2] on the hygiene and marketing of fresh cream by a working party of the Public Health Laboratory Service that potentially dangerous bacteria were only occasionally found in a study it carried out. In this survey the hygiene and methods of production employed in 31 dairies, ranging from large to small, were observed, and more than 5,000 samples of cream were tested during a period of ten months. The samples included 4,385 heat-treated and 517 untreated liquid creams and 282 clotted creams. All were examined by conventional methods for their total bacterial content, for the presence of coliforms, *Escherichia coli*, and other organisms, as well as by a methylene-blue dye-reduction test. So far as possible the results were related to the date of production, the method of distribution, the conditions of storage, and the season of year.

The hygienic standards and the methods of processing the cream in these dairies were, with one or two exceptions, found to be reasonably satisfactory, and in general they complied with the recommendations contained in an advisory code of practice[3] for cream producers. Heat-treated and clotted creams, as expected, gave better results in all tests than raw creams made from untreated milk. Indeed raw cream accounted for nearly all the pathogens isolated, including 54 strains of *Staphylococcus aureus*, probably mostly of bovine origin, and one strain each of *Brucella abortus*, *Salmonella typhimurium*, and *Escherichia coli* 0126. The bacterial flora in all types of fresh cream increased more quickly in summer than in winter, and, likewise, inadequate refrigeration yielded creams with higher bacterial counts than those distributed and stored under correct conditions, though organisms able to grow at low temperatures, even in the refrigerator, sometimes caused anomalous results. As might be expected, the larger dairies with greater resources usually, though not always, produced creams which gave better bacteriological results than those of their smaller competitors. The working party, however, confirmed the findings of previous investigations that, despite apparently adequate heat treatment, too many samples of cream contained too many bacteria, especially *Escherichia coli* and coliform organisms, to be regarded as satisfactory. The better the keeping quality of cream, the safer it is likely to be. Most coliform organisms may not actually be harmful themselves—though antibiotic resistance and its possible transfer to pathogenic bacteria should not be forgotten—it is in the contamination of cream after heat treatment that the report suggests greater care could and should be taken, with resulting benefit to both consumer and producer.

There are no official bacteriological standards for retail cream in Britain, and in considering their possible use the working party looked at some already in force in other countries. Perhaps the working party thought their standards were too stringent, for it considered that improvement in the keeping quality of cream could best be achieved here at present by strengthening the code of hygienic practice for producers. Standards should, of course, be introduced only when they can be consistently attained, and it would have been interesting to know how cream in these countries compared with our own and how much standards are in fact maintained.

Few would disagree with the need for a yardstick of some kind, and the report confirmed that the methylene-blue dye test, though not entirely satisfactory, was the simplest of the tests used and statistically gave the most reliable guide to the hygienic quality of retail dairy cream. The working party therefore recommends it for use as a suitable screening test, provided the age, nature, and history of samples are known, though no suggestions about the frequency with which creams should be examined as a routine are given. Repeatedly unsatisfactory samples from the same source should be followed by joint consultation between the dairy, the local authority, and the public health laboratory in order

Mike Gravel (D–Alaska), who has become a vocal foe of nuclear power, seems to think so. In a speech to the Oregon State Legislature not long ago he urged that every state "stop all construction of nuclear power plants until the safety problems are resolved and until we achieve the safety-first policies to which we are entitled." Last May, voters in Eugene, Oregon, approved a 4-year moratorium on a nuclear power facility planned for their area. Similar movements are afoot in the Oregon legislature, in Minnesota, in New York City, and in California where a citizens' group has succeeded in placing on the June 1972 ballot a proposal to ban power reactor construction for 5 years.

AEC officials understandably find such activities unjustified. And so, it seems, do the most influential conservation organizations, which say they prefer to weigh the merits of atomic power plants on a site-by-site basis rather than putting up blanket opposition to nuclear power.

For their part, AEC officials say that conservatism in plant design and operation should compensate for any uncertainties that remain in the behavior of reactors.

One AEC authority in reactor safety, and a man who is less reserved in his criticism of the agency than most, sums it up this way:

"I believe that nothing in the water reactor safety program is of low priority. It should all be done. And until these tasks are completed we are going to have to use rather more conservative bases for design judgments on plants, and we are going to have to make decisions with a certain lesser degree of cheerfulness, or confidence, than if we had the results of this research.

"We think we can set boundary conditions, so no matter how a reactor goes we are quite sure it's safe. But I find having to work this way intellectually less satisfying. . . . I prefer to *know*, in a quantifiable way, what the limits of safety are.

"However, I think we're in good shape, and that in the long pull, when we look back, we may see we spent money unnecessarily. At least that's what I trust we'll see."

—ROBERT GILLETTE

Lead Poisoning: Zoo Animals May Be the First Victims

New York. Death and illness as a result of simply breathing polluted urban air is a specter of the future that only the more alarmist environmentalists conjure up from time to time. Nevertheless, researchers at New York Medical College (NYMC) have discovered that a large proportion of the animals at Staten Island Zoo suffer from lead poisoning. And while some of the lead in the animals' bodies may have come from paint in their cages, the major source appears to be atmospheric contamination. In the words of Ralph Strebel, the pathologist who directed the study, "The findings have ominous implications for the people who live in that area of the city."

The first indication of trouble at the zoo came last November, when an 11-month-old leopard became weak, started losing its hair, and refused to eat. The cat was taken to New York Medical College, where sick animals from the city's five zoos are treated under the comparative pathology program. Although Strebel and his colleagues could find no evidence of disease, the leopard died 24 hours later.

Three weeks later, zoo keepers found the leopard's fraternal twin, a black leopard (formerly known as a black panther) named Mr. Leo Pard, lying paralyzed in his cage; he too was taken uptown to the medical school. Again there was no evidence of any known disease. But in response to symptomatic treatment, Mr. Leo Pard survived and regained his muscular coordination. At this point, Dennis Craston, a toxicologist from the city's Medical Examiner's Office and an instructor at the medical college, tested Mr. Leo Pard for heavy metal poisoning and found extremely high levels of both lead and zinc in the animal's hair, blood, and feces. A check of the first leopard's preserved organs also revealed high concentrations of the same two metals.

After 6 weeks of intensive treatment at the hospital's animal facility, Mr. Leo Pard was well enough to return home to the Staten Island Zoo. But once there, the level of lead in his body again began to rise. After he went into convulsions, he was taken back to the hospital, where he is still recuperating.

On the basis of their experience with the two leopards, the NYMC researchers decided to check other animals in the zoo for lead poisoning. They found not only that other animals had high concentrations of lead in their bodies, but that the victims ranged from reptiles to primates.

For some time, snakes at the zoo had been dying after having lost sufficient muscular coordination to slither properly. Sure enough, chemical analysis of the preserved carcasses revealed high concentrations of lead. Hair clippings, along with blood and fecal samples, from a variety of cats and primates showed many of the animals to be contaminated with lead—often in amounts far exceeding the level considered toxic in man. Even a great horned owl, brought to NYMC because it had lost its feathers, was found to be a victim of lead poisoning.

Searching for the source of the contamination, the NYMC investigators first tested the zoo's water, food, and bedding and found them all to be free of heavy metals. An analysis of the paints used in some of the cages, however, revealed that 11 out of 16 paints contained lead in concentrations ranging from 0.01 to 3 percent. This finding is significant in itself, according to Craston, because all of the paints are marketed as lead-free interior paints.

But perhaps even more significant were the levels of lead found outside the cages. Grass, leaves, and soil collected on the zoo grounds contained lead in quantities as high as 3900 micrograms per milligram dry weight—an amount equal to or exceeding that found along the sides of major highways, where automobiles continually spew out lead-containing exhausts. "We can only conclude," said Strebel, "that most of the lead taken in by the animals resulted from atmospheric fallout."

Significantly, the animals kept in outdoor cages, including those in cages

without paint, showed the highest levels of lead in their bodies. Even the carcasses of dead mice found inside and outside the zoo buildings were loaded with lead.

Lead poisoning of animals apparently is not confined to the Staten Island Zoo. The same doctors made a preliminary investigation of animals in the Bronx Zoo and turned up the same problem, although fewer animals seem to be affected.

Originally an occupational hazard, lead poisoning in recent years has been identified in slum children who eat chips of paint in old, dilapidated buildings (*Science*, 5 September 1969). Several studies have found an increasing concentration of lead in the air over many cities. And, although the question has been raised, little is known about the effect of this lead on the cities' inhabitants.

It is known, however, that increasing exposure to lead in the air can increase the amount of lead in a person's blood. And a recent position paper drawn up by the Air Pollution Control Office of the Environmental Protection Agency concluded that atmospheric lead pollution does indeed pose a health hazard, particularly for children exposed to lead from other sources.

Since there is little data on the levels of lead in the bodies of adult residents of New York, the findings from the zoo animals cannot be compared with those from the human population. But the wide range of species that were affected in the zoo seems to indicate that man might well be in danger. One of the difficulties in detecting widespread lead poisoning is the lack of specific symptoms. For many years, the headaches and listlessness experienced by slum children who were suffering from subclinical cases of lead poisoning were overlooked by doctors—simply because they were unaware of the problem. And so it could be with some of the city's residents who simply breathe the city air.

Over the past few years, however, New York City has compiled a good deal of data on levels of lead in the blood of children. And according to Vincent Guinee, director of the New York lead poisoning prevention program, there is no apparent correlation between levels of lead in children's blood and those areas of the city in which airborne lead pollution is highest. "I would therefore doubt," said Guinee, "that the animals in the zoo developed clinical symptoms just from breathing the air. But," he added, "I'm prepared for surprises."

The NYMC researchers intend to continue their investigation, in order to correlate their findings in the zoo animals with the surrounding human population. "The zoo animals," said Strebel, "could potentially serve as barometers of the medical effects of the variety of pollutants in the city's air."

—ROBERT J. BAZELL

Public Interest: New Group Seeks Redefinition of Scientists' Role

A small group of scientists has formed a new public interest group to explore the frontiers of social responsibility in science and push the members of their profession into a keener appreciation of the significance of their roles in society. The latest addition to what might be called the non-Establishment scientific establishment is the Center for Science in the Public Interest, set up last January by four alumni of Ralph Nader's Center for the Study of Responsive Law. The purpose of the group, according to James Sullivan, who has a doctorate in meteorology and oceanography from the Massachusetts Institute of Technology, is to stoke the social consciences of scientists and "establish the legitimacy of advocacy in the public interest." The group holds that scientists must make value judgments about their work at every level of scientific endeavor and that "the myth of objectivity is the worst myth we've got in the scientific profession."

The CSPI aspires to plant itself in territory that is at present only thinly inhabited—"the middle ground between science and law"—where it hopes to supply reliable and extensive technical input into decision-making processes, both in government and in the courts.

It has staked out three primary areas of activity: making available competent witnesses to testify at hearings on science-related legislation before Congress; conducting studies to supply consumers with information on matters about which data are either unavailable or obscured by conflicting sets of "facts"; and instigating its own lawsuits, as well as acting as co-plaintiff in public interest legal actions.

Of the four men comprising the organization, three are scientists and one is a lawyer. For at least two of them, experience working for Nader seems to have been the catalyst in turning them from scientists into scientist-advocates. Sullivan says that after he got his degree last June he planned to spend a month working for Nader and then return to M.I.T., where he had been offered a job doing research on how to cope with oil spills. He ended up spending 6 months with Nader, upon which, "after much agonizing," he decided he couldn't go back to a job that was "just a cosmetic approach" to more fundamental problems.

Another man on the team is Albert J. Fritsch, a Jesuit priest with a Ph.D. in organic chemistry from M.I.T. One day, he says, he saw Nader make a mistake during a television appearance about the difference between two gases and decided that "he needed scientific input desperately." He joined the Nader group for a year and remains one of their scientific consultants. The other two CSPI men are Michael Jacobson, a microbiologist from M.I.T., and Kenneth Lasson, a Maryland lawyer.

The group feels that most scientists, in the effort to preserve the purity of their work, deliberately avoid making any but practical, technical judgments about what they are doing. They thus fall, inadvertently, into the role of advocate of the particular interests of their employers. The country's number one example of this phenomenon, says Sullivan, is presidential science adviser Edward E. David, who said in a recent speech that he believed the function of a science adviser should be to present options and not to pass judgments. "Advocacy and the traditional scientific approach are not comfortably compatible," he said. But David, says Sullivan, is clearly pushing his boss's policies—notably in his recent efforts to sell the SST.

The CSPI thinks a different kind of advocacy is called for from scientists, one based on thorough consideration of the implications of their work, with priority given to the interests of the public rather than the interests of their employers. Each individual has to assess

concentrate on building on clear sites which universities already own. In addition the new buildings which we put up on those clear sites cannot have a great deal of room for still further expansion of numbers. That is to say, shortage of money forces us to add rather small additions to university libraries or laboratories. We cannot afford to put down extensions which will not be fully occupied for a decade.

The next quinquennium

In our planning for the next quinquennium we have agreed on a capacity with all universities which amounts to 300 000 full time equivalent students in October 1974 when the building programmes already authorized are complete. We hope to put in new buildings for 20 000 more students in the next two years which would give a capacity of 320 000 by 1976/77—the end of the next quinquennium—55 per cent science and 45 per cent arts capacity. We expect universities to put in expansion proposals which square with this and we will submit these to the Government together with the likely running costs which such numbers would imply. **The Government will decide on the numbers and the money and the UGC will then divide it out. Until we get that Government decision all must be conjecture.** I would not expect either the resources or the promotion prospects to go back to the halcyon days of the mid-1960s when all the new universities and new university departments were looking for senior staff. But I believe that the position will still compare favourably with nearly every other university system in the world and leave British universities with the autonomy to work out their own changes in curriculum and organization, and the time and money to do first class teaching and research.

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Lead pollution from petrol

D Bryce-Smith

How far are lead alkyls in petrol responsible for the present lead levels in the general population? And are these levels becoming unacceptably high? Points raised in the recent article 'Lead in the environment' by A. L. Mills, chairman of the Institute of Petroleum's advisory committee on health,[1] are reviewed and some further evidence on the subject is discussed.

Mills' article[1] followed the publication of a survey of lead pollution[2] which drew attention to the high levels of accumulated lead now being found among the population, and the responsibility of the petroleum industry, through their addition of lead to petrol, for a significant part of the daily absorption of lead by people living in cities. An extended version of ref. 2 including an epidemiological survey of recent mental health data, has now appeared.[2a]

I was disappointed to find so few indications in Mills' article that he and his committee, who presumably advise the petroleum industry on health matters, are at present prepared to do more than adopt a watchful but defensive posture. If the petroleum industry's policy on the retention of lead additives is really based on the considerations so ably presented by Mills, some further analyses of these considerations may prove useful.

Mills' general case is that lead may for long have been with us at contemporary levels, and that although further study is needed in several areas, the present levels which have accumulated in man normally fall within acceptable limits. Only one point in ref. 2 was explicitly questioned by Mills, namely the significance of N. Greenland ice data; but his alternative explanation will need to be withdrawn. Before these points are dealt with, a key question which does not seem to be in dispute will be referred to, namely the approximate proportion of inhaled lead which can be absorbed by man.

Lead in city air

The figure of 50 per cent absorption of airborne lead used in both articles implies that inhalation can contribute up to about half the total daily absorption of lead from all sources by people in cities; but a note of caution is required over the use of specific figures and the specific conclusions which can follow.[1,2] Many people are undoubtedly absorbing much less than half of their daily lead by breathing, and some may well absorb more than half, for, quite apart from variations in location, the ability to retain both inhaled and ingested lead varies between healthy individuals by a factor of three or more—found by studies[3-5] with the radioactive ^{212}Pb isotope. The amounts inhaled also depend on the volumes of air respired which, in turn, may vary by a factor of five or so among men and women at rest, and may be increased up to 10-fold in an individual by exercise. Thus any average figure conceals the large range of individual susceptibilities, and thereby leads one to underestimate the danger to public health. In such a complex situation, where every person is likely to have his own individual pattern of inhalation, ingestion, absorption and excretion, one can only urge the utmost prudence in setting standards for what is acceptable to the general population.

However, if for the sake of this analysis we take it as common ground that inhalation of airborne lead can contribute up to about half the daily dose of lead for people in cities, the degree of responsibility of the petroleum industry then hangs on the contribution which the use of leaded petrol is making to airborne lead levels. Evidence that this contribution is normally the major one will be found in ref. 2, and especially refs 1, 4, 5 and 6 therein, and Goldsmith and Hexter's study[6] of the relationship

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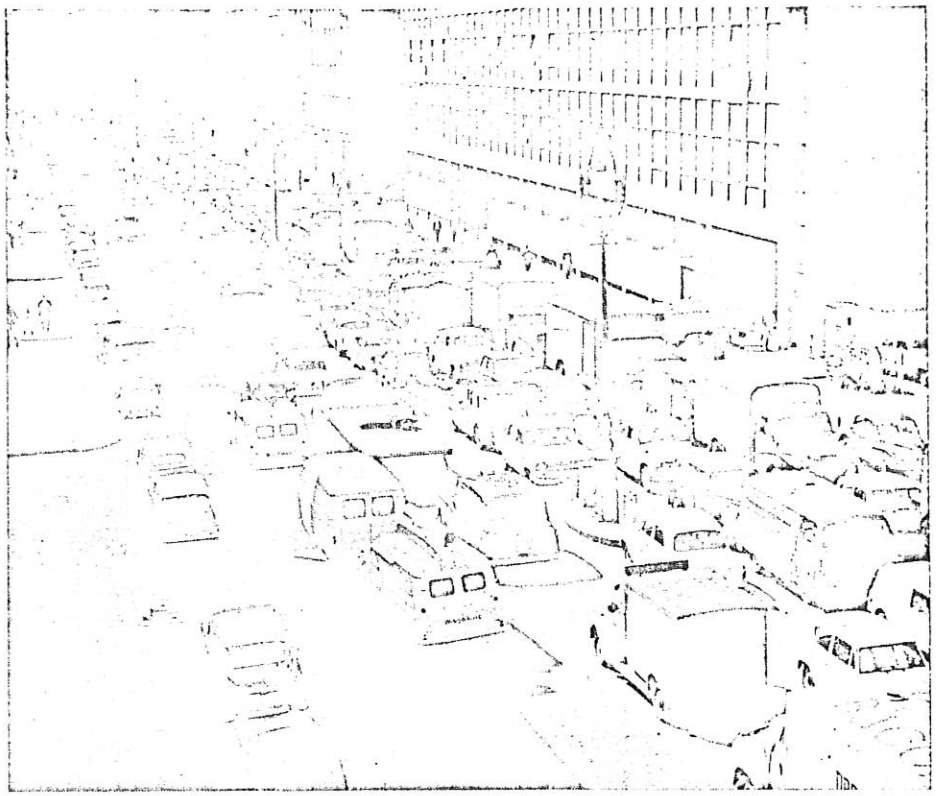
between blood lead and exposure levels should also be read in conjunction with results of the Bochum experiment described below. Recently Chow has used the variability of the natural lead isotope ratios to 'fingerprint' lead in petrol and airborne lead over a world-wide range of cities. In almost every case, the two sets of isotope ratios were in close agreement.[7]

Mills, and others, have referred to coal burning as a source of airborne lead. According to an authoritative estimate from the National Coal Board, supported by analyses, the maximum amount of lead which could have entered the air from coal burning in Britain during 1970 is *ca* 120 tons: the true figure is probably much lower.[8] In comparison, a quantity approaching 10 000 tons of lead (possibly a much higher figure[8a]) came from leaded petrol, well over 50 times more. Moreover, upwards of 200 tons of this petrol lead would have been in the specially dangerous organolead forms, according to the rather scanty data available. So although Mills is correct, his allusion does little to displace the main burden of responsibility from the petroleum industry. Doubtless other more local contributions to airborne lead arise from lead smelting, and the burning of painted wood, lead-filled plastics and other waste material.

The lead content of city dust tends to be about 1 per cent, and is rising: figures approaching 5 per cent have been reported from Germany.[9] A study in Zürich showed that the rise closely paralleled the increase in consumption of leaded petrol over an eight-year period.[10,11] Purves has reported that urban top-soil in Scottish cities generally contains about four times more lead than is found in top-soil from rural areas.[12]

If any doubts remain about the main source of airborne lead in cities, they may well be resolved by work from the University of Münster in which groups of male students, hospital employees, traffic policemen and male smokers were exposed for 3 h at a central road intersection in Bochum (2400–2800 cars per hour).[13] All groups showed elevations of blood lead after exposure, of the order 50–100 per cent. The results led to the important inference that exposure to traffic fumes can also mobilize existing body lead from depots in bone and tissues into the blood: thus some other component of traffic fumes may also be involved.

In another study, blood lead levels in Frankfurt street cleaners were found to be significantly higher than those in the general city population. Although no clinical lead poisoning was apparent, urinary δ-aminolaevulinic acid—a sensitive indication of interference with haem synthesis by lead—was at levels considered dangerous in about 15 per cent of the cases.[14] These findings emphasize the point made in ref. 2 that lead in exhaust fumes is absorbed both directly by inhalation and indirectly through exposure to food, water, dust *etc.* which have been contaminated by fall-out.



Traffic jams can lead to unusually high concentrations of lead in the air. (Camera Press)

Greenland ice studies

Mills' suggestion that the marked rise in lead levels in N. Greenland ice since 1940 could be due to contamination from aircraft using Thule (150 miles distant) and Camp Century (50 miles distant) is untenable for a number of reasons. Firstly, the greatest amounts of lead were found to be deposited in the winter months when precipitation was heaviest and air traffic lightest. Secondly, the lead levels from sites between the Virgin Trench and the bases showed no elevation attributable to significant contamination from the bases. Thirdly, the Virgin Trench site was predominantly upwind from the bases. It is also worth noting that the major increase in ice-lead levels began about 20 years before the closer base was established.[15]

The N. Greenland ice studies have received strong support from an independent study of lead levels in Scandinavian mosses which, through their ability to absorb airborne nutrients, can act as sensitive indicators of air pollution.[16] The analyses show an abnormally high accumulation of lead since about 1950, the amounts increasing with precipitation and closeness to large centres of population. It is interesting that the concentration of lead in ocean water decreases markedly with depth, whereas the concentration of barium, a heavy metal having little industrial use, shows the converse relationship.[17] Of course, lead in surface waters arises both from the air and from rivers, so the levels will tend to reflect the more general aspects of pollution by the metal, together with any tendency for concentration by natural processes in the epipelagic zone.

Mills then cited Cholak's report[18] to suggest that airborne lead levels may actually have been decreasing over the last 30 years, except 'where populations are increasing'—in which cities are they not? The reported findings run contrary to virtually all other evidence, but if the work had been performed with more rigid scientific control of the experimental parameters it might indeed have gone some way towards exonerating petrol as the main source of atmospheric lead. As it is, the various changes of sampling devices, analytical procedures, sampling location and sampling duration which were made during the period of measurements do not inspire confidence in any conclusions based exclusively on the work.

'Acceptable' levels

Mills concluded that 'the mean levels (of lead) in the general population fall well within the acceptable limits'. Two words in this quotation require scrutiny —'mean' and 'acceptable'. First, it should be apparent from the distribution curves given in ref. 2 that mean levels

conceal wide individual variations and provide no indication of the risk to which many people are actually exposed. Mass screening of city children in the US is revealing that 5 per cent and more ed referring to lead poisoning clinics.

a 'control' sample of Manchester children,[19] 4 per cent had blood lead levels above the 0.8 ppm level which is associated by the American Academy of Pediatrics with a great risk of brain damage,[20] and 17 per cent had levels ove 0.5 ppm. Neither the authors of ref. 19 nor Mills who cited the work saw fit to comment directly on these dangerously high levels found for some individual children. The authors concentrated attention on the similarity of the mean values found for control and mentally backward children and cautiously concluded that the latter had not been particularly at risk from lead poisoning. Actually, it is evident from the more extensive American clinical experience that some of these Manchester children were definitely at serious risk of suffering brain damage from lead at the levels found.[20]

The second word 'acceptable' was used by Mills in the context of a statement by 'an international group of medical specialists in lead problems'.[21] Professor A. Goldberg (Glasgow), who was one of this group, informs me[22] that the published statement was intended apply to occupationally exposed lts only, and that he disapproves of its extension by Mills to the quite different problem of an exposed whole population.

Mills' allusion to the undoubted rarity of fatal poisoning among industrial workers handling lead alkyls could be misleading. Lead alkyls such as tetraethyl- and tetramethyl-lead differ from most inorganic lead compounds in being volatile, fat-soluble, and readily able to cross the blood-brain barrier, possibly as R_3Pb^+. Tetraethyl-lead is a psychotropic poison. The symptoms of poisoning in adults can closely mimic those of a conventional psychotic disorder, and differ significantly from those of inorganic lead poisoning, *e.g.* there is little or no elevation of blood lead, so correct diagnosis is very difficult in the absence of a known history of exposure. These facts have long been known in the lead and petroleum industries. Elaborate health precautions are taken to protect industrial workers handling lead alkyls, for example by the provision otally enclosed moonman-type suits and frequent health checks. Yet enormous volumes of these dangerous compounds are put on sale to the general public as a *ca* 0.1 per cent ocarbon solution, *i.e.* leaded petrol, without warning to the users, even to garage mechanics, of the mental derangement which can result from excessive exposure, *e.g.* through the use of petrol as a de-greasing or cleaning solvent. Further, nothing is apparently known of the effects on children. Most of the reports which are usually claimed to have established the safety of lead alkyls under normal conditions of use are reticent on the specific psychic symptoms of poisoning which might have been expected. The implication that lead alkyl poisoning is largely a rare type of specialized industrial disease appears to require serious reassessment by Mills and his Health Committee, and others. Any hazard from organic lead must be regarded as additional to that from inorganic lead in exhaust fumes.

I strongly endorse some of Mills' comments and allusions, for example the need to pay more attention to potentially dangerous sources of lead other than petrol, a list of which would include plumbing, paint (especially primer), lead toys, lead glazed earthenware, and plastics containing lead fillers and stabilizers; but others appear unfortunate, notably his attempt to imply that the safety of present levels of lead pollution might be assessed by reference to the public health standards of New Guinea natives or 3rd century Poles.

Conclusion

The three main toxicological considera-ions may be summarized as follows: (*i*) lead is a cumulative poison in man; (*ii*) it can harm the central nervous system, *inter alia*, and is particularly liable to cause brain damage in children; (*iii*) no other toxic chemical pollutant appears to have accumulated in man to average levels so close to the threshold for potential clinical poisoning. To these may be added a fourth on which the evidence is more limited, and suggestive rather than conclusive: that some illness among the general population may be attributable to the present levels of accumulated lead.

A preliminary analysis of recent trends in UK mental illness statistics has suggested the presence of a recently introduced or augmented causative factor to which the young are most sensitive.[23] Evidence from a group in the Swiss Society of Preventive Medicine indicates that exhaust fumes from motor vehicles may already be causing cases of toxic lead encephalosis (inflammation of the brain) among the urban population: the symptoms include depression, headaches and undue fatigue. Treatment for lead poisoning with calcium-EDTA rapidly led to a cure or improvement in 85 per cent of the cases.[23] Adverse effects on mental efficiency from unidentified components of traffic fumes have been reported[24] (cf. ref. 13). Treatment of some mentally retarded children for lead poisoning has been found to improve their behaviour.

Much more detailed work of this kind is needed, together with mass screening of children for incipient lead poisoning along the lines now proving so successful in the US.

Much of the lead now in our environment is a legacy from past misuse for which it would certainly be unfair to blame petrol alone. Indeed, one can appreciate the expensive efforts which Mills and his colleagues in the petroleum industry are now making to control emissions of sulphur dioxide, carbon monoxide, oxides of nitrogen, and other non-cumulative pollutants. But lead is more dangerous than these.

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2485. [Significance of the heat factor in poisonings induced by industrial poisons from the heavy metal group] Savitskif IV. Vrach Delo 3:138-42, Mar 71 (Rus)
ANIMAL EXPERIMENTS/ CADMIUM, poisoning/ COBALT, poisoning/ COPPER, poisoning/ ENGLISH ABSTRACT/ ENVIRONMENTAL EXPOSURE/ GUINEA PIGS/ HEAT/ INDUSTRIAL MEDICINE/ LEAD POISONING/ MERCURY POISONING/ *METALS, poisoning/ MICE/ *OCCUPATIONAL DISEASES, chemically induced/ RABBITS/ RATS/ TIN, poisoning/ ZINC, poisoning

2486. [First aid in poisoning] Dönhardt A. Wien Med Wochenschr 120:585-91, 5 Sep 70 (Ger)
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BLOOD

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2489. [Clinical value of a new hematological index in the diagnosis of saturnism: average erythrocyte concentration of lead] Grisler R, et al. Med Lav 60:360-5, May 69 (Ita)
ENGLISH ABSTRACT/ *ERYTHROCYTES, metabolism/ HUMAN/ *LEAD, metabolism/ *LEAD POISONING, blood/ LEAD POISONING, diagnosis

2490. [Diagnosis of occupational diseases, a persistent problem. Differential diagnosis: lead poisoning--thalassemia] Symanski H. Z Gesamte Hyg 16:533-7, Jul 70 (Ger)
DIAGNOSIS, DIFFERENTIAL/ HUMAN/ LEAD, blood/ LEAD POISONING, blood/ *LEAD POISONING, diagnosis/ MALE/ *OCCUPATIONAL DISEASES, diagnosis/ *THALASSEMIA, diagnosis

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2494. [Porphyrin metabolism and hematologic diseases in lead poisoning] Cotrone D, et al. Minerva Med 62:1041-8, 10 Mar 71 (Ita)
*ANEMIA, etiology/ ANEMIA, HEMOLYTIC, etiology/ *BONE MARROW DISEASES, etiology/ HUMAN/ IRON, metabolism/ LEAD, blood/ *LEAD POISONING, complications/ LEAD POISONING, metabolism/ *PORPHYRINS, metabolism

2495. [Gout and hyperuricemia associated with lead poisoning] Lejeune E, et al. Rev Rhum Mal Osteoartic 38:161-73, Apr 69 (Fre)
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2496. Plumbism exists today. Reddick LP.
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2497. [Bone changes in chronic lead poisoning] Grinberg AV, et al.
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2498. The measurement of urinary delta-aminolevulinic acid in detection of childhood lead poisoning. Vincent WF, et al. Am J Clin Pathol 53:963-4, Jun 70
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2504. Plumbism exists today. Reddick LP.
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ADULT/ ANEMIA, etiology/ BLOOD CHEMICAL ANALYSIS/ BRAIN DISEASES, etiology/ CHILD, PRESCHOOL/ EDTA, therapeutic use/ ENVIRONMENTAL EXPOSURE/ GOUT, etiology/ HUMAN/ INFANT/ LEAD, metabolism/ *LEAD POISONING/ LEAD POISONING, complications/ LEAD POISONING, diagnosis/ LEAD POISONING, drug therapy/ NEPHRITIS, etiology/ NEUROMUSCULAR DISEASES, etiology/ PICA, complications/ REVIEW/ SKIN ABSORPTION

2505. [Diagnosis of occupational diseases, a persistent problem. Differential diagnosis: lead poisoning--thalassemia] Symanski H.
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DIAGNOSIS, DIFFERENTIAL/ HUMAN/ LEAD, blood/ LEAD POISONING, blood/ *LEAD POISONING, diagnosis/ MALE/ *OCCUPATIONAL DISEASES, diagnosis/ *THALASSEMIA, diagnosis

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2509. [Erythrocytic acetylcholinesterase and serum cholinesterase activity in rats treated with lead nitrate] Rausa G, et al. Med Lav 61:554-62, Nov 70 (Ita)
*ACETYLCHOLINESTERASE, blood/ ANIMAL EXPERIMENTS/ *CHOLINESTERASE, blood/ ENGLISH ABSTRACT/ *ERYTHROCYTES, enzymology/ *LEAD POISONING, enzymology/ RATS

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*ADRENAL GLANDS, enzymology/ *ALCOHOL OXIDOREDUCTASES, analysis/ ANIMAL EXPERIMENTS/ ENGLISH ABSTRACT/ *LEAD POISONING, enzymology/ RABBITS

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EXPOSURE/ HEMOGLOBIN, analysis/ HUMAN/ LEAD, blood/ *LEAD, metabolism/ LEAD, urine/ *LEAD POISONING, metabolism/ LEAD POISONING, occurrence/ LEVULINIC ACID, urine/ MALE/ MASS SCREENING/ OCCUPATIONAL DISEASES, occurrence/ PORPHYRINS, urine/ PYRROLES, urine

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2517. [Metabolism and determination of lead in lead poisoning] Matsumoto H.
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2526. [Gout and hyperuricemia associated with lead poisoning] Lejeune E, et al.
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2-10-72
Att. II

STATEMENT WITH REFERENCE TO

KANSAS HOUSE BILL 1666

Mr. Chairman, Gentlemen, my name is Robert C. Butler. I am Manager, Environmental Affairs for the Petroleum Chemicals Division of the Du Pont Company. We appreciate this opportunity to comment on House Bill #1666.

I will confine my comments to that portion of this Bill which proposes to differentiate between the State tax imposed on leaded gasoline and that which would be imposed on gasoline containing less than 0.3 grams of lead antiknock per gallon. The Bill would provide an economic incentive of 3¢ per gallon to encourage the use of gasoline containing less lead. We presume that the objective of this provision is to reduce air pollution caused by automobiles. While we agree that this is both necessary and desirable, we respectfully suggest that a lead based gasoline tax differential will not improve the environment and is not the best approach from the view of the public interest.

The proposed legislation prejudges the necessity of removing lead from gasoline and by-passes the considerable scientific investigation now being conducted by both industry and the government to ascertain the facts in this complex and controversial matter.

Du Pont is a major supplier of lead antiknock compound to the petroleum industry. We are vitally interested in automotive emissions and have been conducting an extensive research program for the past nine years to study the factors which affect automotive emissions and to determine how such emissions can be controlled. We are pleased to share with you the results of some of this work in the hope that our information will assist you in weighing the need for the legislation you are considering.

Lead antiknocks are added to gasoline as an economical way to increase its octane quality. The availability of high octane gasoline at reasonable cost has made possible the development of today's efficient high compression engines. The use of lead saves motorists an estimated $3 to $4 billion dollars every year in gasoline costs. It also results in a crude oil saving of about 230 million barrels every year at the present rate of refinery crude throughput. It takes about 6% less crude energy to produce leaded gasoline than to produce unleaded gasoline of comparable octane quality.

The continued use of lead antiknock has now been questioned on two grounds: (1) concern about a possible health hazard, and (2) the effect which lead has in reducing the effective life of catalytic converters which are among the devices being developed to remove gaseous pollutants from automotive exhausts.

With regard to the possible health hazard of airborne lead, Du Pont feels it is the responsiblity of appropriate governmental agencies to set air quality standards for pollutants which are adequate to protect the public health and welfare. The need for such a standard for lead is now being evaluated by the Environmental Protection Agency. At their instigation the National Academy of Sciences recently completed a study of the health effects of airborne lead.

This study concluded that "...lead attributable to emission and dispersion into general ambient air has no known harmful effects." The only other reason for restricting the lead content of gasoline would be the effect which lead has in reducing the effective life of catalytic emission control devices. Although two automobile manufacturers have announced their present intention to use such devices in the future, there will be no catalytically equipped cars on the road before 1975, and they will not represent a significant portion of the automobile population for several years after that. No manufacturer has yet demonstrated an emission control system which will reduce emissions to the levels required by the 1975 Federal standards and have sufficient durability and reliability to last for the required 50,000 miles. If restrictions on the use of lead in gasoline are considered to be necessary, we believe they should be tied to the availability of cars which require unleaded gasoline for the effective operation of catalytic systems. Efforts to accelerate the availability of unleaded or low-lead gasoline of today's octane quality either through restrictive legislation or economic incentives could cause other environmental problems more serious and more immediate than lead.

For example, when the lead content of gasoline is reduced the octane quality goes down. If all of the lead is removed the octane drops to about 88.5. Only a small percentage of the pre-1971 cars now on the road will operate on gasoline of this octane quality without knock and possible engine damage. It is reasonable to expect that if the use of lead is restricted, refiners will increase the octane of their gasoline in some other way to satisfy their customers. The most probable way in the light of present day refinery technology is to add additional amounts of compounds called aromatics. These have high octane without

lead. They are also quite reactive. Increasing the aromatic content of gasoline increases the smog forming tendency of the exhaust gases. This affect has been well documented in work done by both the Bureau of Mines and General Motors.

Increasing the aromatic content of gasoline also increases the emission of compounds called polynuclear aromatics. Some of these compounds have shown carcinogenic effects when tested in laboratory animals. Dr. Herbert C. McKee of Southwest Research Institute, who is also chairman of the Texas Air Pollution Control Board, put this problem in proper perspective when he commented in a talk to the American Chemical Society in April, 1969, that "While there is no reason to suspect that this hazard exists at this time, any control measures which would cause a considerable increase in the polynuclear aromatic content of the atmosphere should be viewed with suspicion until more information is available. Therefore, cutting down on the lead content of motor fuel to reduce a suspected but unknown health hazard, might increase another health hazard that is suspected but equally unknown and unproven. Trading one unknown hazard for another hardly seems appropriate, especially since lead additives have been used in motor fuel for over forty years." Our laboratory data confirm that the emission of polynuclear compounds from auto exhaust increases as the aromatic content of the gasoline goes up.

In addition to the problems which arise from the increased use of aromatics in gasoline, Du Pont has found in its laboratory work that the number of particles emitted to the atmosphere when cars are operated on unleaded gasoline under typical motorist-type driving conditions involving short trips and

some cold starts is several times greater than when leaded gasoline is used. This finding indicated that the wide-spread use of unleaded gasoline could reduce visibility and cause increased soiling since these effects are dependent upon the number and size of particles rather than upon their weight. To find out if our laboratory findings were representative of what happens in a real-life situation, we arranged to run a test in an idle one-and-one-quarter-mile-long two-lane traffic tunnel on the Pennsylvania Turnpike. This tunnel in essence formed a two-million cubic foot test chamber in which we could compare the particulate emissions from cars run on unleaded and leaded gasoline without interference by particulate matter from other sources. Since these data are comparatively new I would like to take just a moment to describe the test.

Our test fleet consisted of eight cars, four cars operated on unleaded gasoline and four on leaded gasoline. These were 1969, 1970 and 1971 Chevrolets equipped with 350 cubic inch V-8 engines, automatic transmissions and two-barrel carburetors. The leaded and unleaded cars were matched for both mileage and emissions prior to the start of the test.

We blocked both portals of the tunnel with large plywood panels. Each car was run back and forth through the tunnel for a distance of 5.4 miles, using a driving cycle which simulated city driving conditions. Each test involved driving four cars a total of 22 miles. This took about an hour, and the pollutant levels built up in the tunnel as measured by carbon monoxide concentration were equivalent to those in heavy traffic in a major city. After the four unleaded cars had been run and suitable measurements taken, the tunnel was cleaned out and ventilated. The four leaded cars were then run and similar

measurements of visibility and soiling were taken. During the hour that it took to run each set of four cars we operated a high-volume sampler in the tunnel. We then used a standard ASTM test to determine the soiling index of the filter pad and the effect on visibility as measured by light transmission. We found that (1) the unleaded fleet caused approximately twice as much reduction of light transmission in the atmosphere as did the leaded fleet; and (2) the unleaded fleet caused approximately twice as much soiling as did the leaded fleet.

I have attached a picture to your copy of this statement which shows the filter pads taken from the high-volume air sampler following one set of tests.

I think it is also important that you be aware that there are factors already at work in the direction of reducing the lead antiknocks used by refiners. For example, automobile manufacturers have reduced the compression ratios of 1971 and 1972 model cars, permitting them to operate on gasoline of lower octane quality. Lower octane quality usually means lower lead dosages. A number of major oil companies are already marketing low-lead or no-lead gasolines, and others have announced their intention to do so when there is a need for it.

There are a number of other considerations which should be weighed in connection with a lead-based gasoline tax.

(1) The burden of such a tax falls on those who own pre-1971 cars. These individuals, particularly as the years pass, would be those who bought second or third-hand cars which were made, entirely legitimately, to use high octane leaded gasoline.

This is a strata of society least able to afford an additional tax burden. Most people do not buy a used car if they can afford a new one.

(2) A tax based on the lead content of gasoline presumes that the issue of the environmental effect of lead antiknocks has been unequivocally settled. Such a dogmatic conclusion is unwarranted by the facts available at this time.

(3) A tax based on lead content discriminates against small refiners who have limited capability to make gasoline of adequate octane quality for today's cars without lead. Larger refiners, able to produce higher octane quality with reduced amounts of lead, could gain a competitive advantage of 3¢ per gallon under the provisions of HB 1666, by reducing the lead content of present gasolines.

(4) A lead-based gasoline tax could have an especially severe impact on farmers. Little has been written about the continuing need for leaded gasoline for farm tractors and other gasoline-driven farm equipment. This equipment has a useful life many years longer than that of the typical automobile -- years in which gasoline with some lead content must continue to be available if this equipment is to continue to operate.

(5) The Environmental Protection Agency is already moving to place restrictions on the use of lead in gasoline. They are expected to issue a proposed rule-making in this area within the next few weeks.

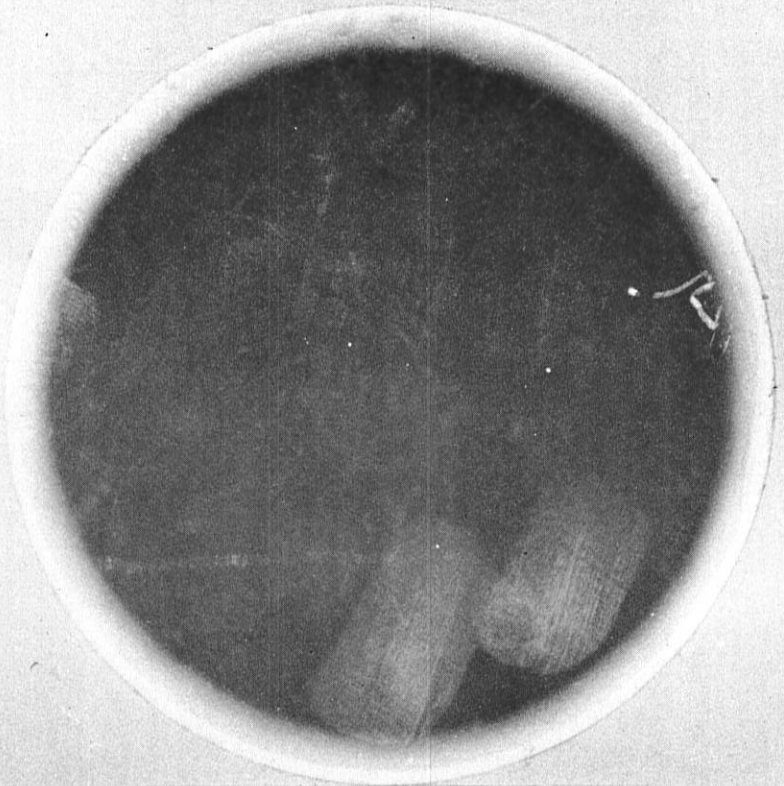
(6) Lead emitted to the atmosphere by automobiles can be greatly reduced through the use of particulate trapping devices. A number of companies have such devices for automobiles under development. One such system, developed by Du Pont, has

demonstrated the ability to reduce exhaust particulate emissions by 90-95% in a 67,000 mile test.

In summary, the objective of the portion of HB 1666 which provides for a tax differential based upon lead content, that is, the reduction of automotive pollution, is already being accomplished more effectively and more quickly by the concerted efforts of industry and government in this area.

I will be happy to answer any questions which this Committee may have.

4 CAR x 6 CYCLE TUNNEL TEST



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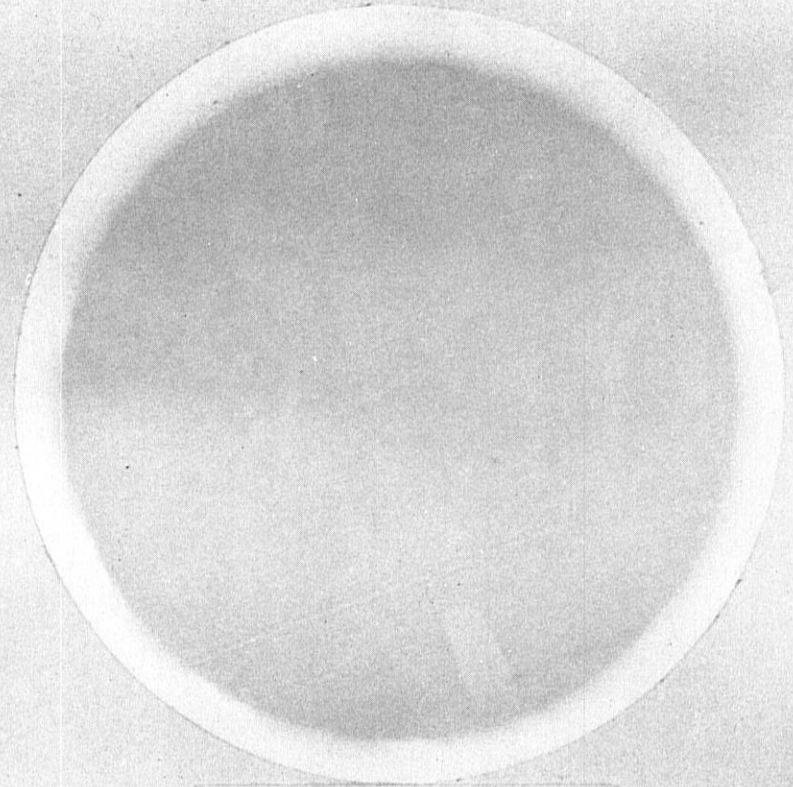

A6
LEADED