the tails of the distribution. Children at the higher lead levels were almost four times as likely to have a verbal IQ below 80. Similar effects have been found by Winneke and Yule, but these studies have been challenged by Erdman, who has criticized their methodology, and found no I.Q. effects in her studies. Recently studies of hair lead by Thatcher showed significant correlation between lead and I.Q. with no threshold. EPA studies have shown that EEG changes occur as a function of blood lead over a range of 6-57 ug/dl.

Blood Lead Levels and Lead Poisoning

Billick Analysis

The United States has been screening children for lead poisoning for more than a decade. EPA has reviewed the original data for New York, Chicago and Louisville. This data contains blood lead levels, age, and race. The Ethyl Corporation has survey data showing the amount of leaded and unleaded gasoline sold in each of these SMSA's monthly. The Bureau of Mines and the Motor Vehicle Manufacturers Association have survey data showing the average lead content of leaded gasoline in each SMSA. Dr. Billick has performed statistical studies for us upon this data:

- evaluating the strength of association between gasoline lead and the geometric mean blood level of black and white pre-school children in each city;

- regressing the number of children with blood lead concentrations above 30 micrograms/dl (the HHS safety level with total lead emitted from gasoline; and

- predicting the number of black and white children with lead poisoning under various lead phasedown scenarios.

These studies show that:

o In all three cities gasoline lead emissions are the highest correlant with blood lead. Charges in gasoline lead levels explain about half of the variation in blood lead levels.

o Increasing gasoline lead levels from the current 0.5 grams per gallon to 1.0 grams per gallon would increase lead poisoning among pre-school children.

  - For black 2-3 year olds, the regression predicts a 14% increase in children above the safety level cases in Chicago, a 19% increase in black 2-3 year

olds above the safety level in New York, and a 47% increase in black 2-3 year olds above the safety level in Louisville. This corresponds to an additional 2-4% of all black 2-3 year olds suffering lead poisoning in these cities.

- o For the other proposed levels, the increase in cases of 2-3 year old black children above the safety level in 1983 will be:

  - at 0.8 grams per gallon

    a 12% increase in cases above the safety level in Chicago; corresponding to an additional 1.7% of all black 2-3 year olds

    a 16% increase in cases above the safety level in New York; corresponding to an additional 1.5% of all black 2-3 year olds, a 30% increase in cases above the safety level in Louisville, corresponding to an additional 2.3% of all black 2-3 year olds

  - at 0.65 grams per gallon

    a 7% increase in cases above the safety level in Chicago

    a 9% increase in cases above the safety level in New York

    a 2% increase in cases above the safety level in Louisville

The incidence of cases above the safety level diminishes as children get older, but it is still significant in black children aged six to seven years. For these older children:

- o increasing gasoline lead levels from the current 0.5 grams per gallon to 1.0 grams per gallon would cause the same an increase in cases above the safety level. The predicted increase is:

  - an additional 2% of all children in Chicago, 1.8% in New York, and 2.8% in Louisville having lead levels above the safety level.

Tables I, II, and III display the regression projections of children with lead poisoning at various gasoline lead levels for 1983, 1984 and 1985, and for all of the proposed phasedown schedules. Tables IV, V and VI show the results for white children. While the

absolute percentage above 30 mg is lower for white children, the increase in lead poisoning cases from 1.0 gpg is still between about 2% of all white two year olds in New York and Chicago and about 4% in Louisville.

These are based on Dupont's projection of future gasoline sales nationwide, and on Mobile Source Enforcement's projections of the change in the leaded/unleaded mix, starting with the known 1980 splits in each city. The regression was a multiple regression which included variables for age, race and season as well as gasoline lead. This was to minimize the assignment of variation in blood lead to gaoline lead, when it might be accounted for by other factors. The projections of lead use in these cities is probably low, since we assumed that leaded gasoline would contain the national average lead content in them. Since New York and Chicago have higher than the national average fraction of unleaded, refiners that market a significant share of their gasoline in those SMSA's will in fact be able to put more lead in their leaded gasoline and still meet the same average lead content per gallon, (since the will make more than the national average amount of unleaded). By assuming otherwise, we have underestimated exposure to lead, and therefore underestimated the number of lead poisoning cases.

OPRM Analysis

In October 1980 major refiners significantly reduced their gasoline lead content, leading to a 16.9% reduction in the average lead content of gasoline between the second quarter of 1980 and the second quarter of 1981. It would be useful to examine data that extends into 1981, so as to capture that effect. Unfortunately Ethyl Corporation stopped gathering SMSA sales data in September 1980, so it is difficult to obtain data for regression purposes. The above regressions went up to mid 1980. I have looked at two approaches. First, on a qualitative level, the National % of screened children who had lead poisoning, as reported to CDC in the second quarter of 1981 was 3.55% compared to the second quarter of 1980, when it was 4.3%. This is a 17% drop, which compares well to the reduction in average gasoline lead content from 0.71 gpg in II80 to 0.59 gpg in II 81 (16.9%).

The CDC data base isn't optimal for analysis, since it is not broken down by age or race, and since we do not know what screening techniques were used. This is why we went back to the original data sources in three cities. However, even recognizing these flaws, the above data is suggestive. We know of no other environmental variable that could have changed that significantly over a single year.

While regressing the data into 1981 is made difficult by the absence of SMSA data, Ethyl did collect state-wide data in 1981. Since the Providence SMSA is such a large fraction of Rhode Island, I have regressed the percent of screened children with lead poisoning versus total lead emissions in the Providence SMSA. For IV 80, I81, and II 81, I allocated to that SMSA the same ratio of state-wide leaded gasoline sales as the Ethyl SMSA and state data showed for the same quarters a year earlier. The Motor Vehicle Manufacturers Association October 15, 1980 survey of gasoline was used for IV 80, and the January 15, 1981 data was applied to I 81 and II 81. The regression showed, for 14 quarters I78 to II 81.

% screened with lead poisoning = -0.7356 + .1328 gas lead

gas lead is in 10^6 grams

Correlation Coefficient = 0.8641
Coefficient of Determination = 0.7467

To get an idea of what changing our standard would do, without putting too much reliance on the details due to the weakness of the data base, I looked at a 40% increase in lead emissions from 1981 and a 10% decrease in lead emissions as proxy's for the approximate effects of complete recission and no change. The 40% increase would imply 3.45% of all children (both races) screened having lead poisoning, while a 10% decrease would imply 1.95% of all children screened would have lead poisoning. Thus the range of alternatives EPA is studying could change the percentage of all children with lead poisoning by 1.5%. This is consistent with the results of the more detailed analyses for New York, Chicago and Louisville, and the correlation coefficient is consistent with both those cities and the CDC analysis of the NHANES II survey. It suggests that 1.0 gpg would lead to over a 40% increase in lead poisoning cases in Providence. In addition, as shown in Figure 3, there were sharp drops in the % of children with lead toxicity in the quarters after the two mandated EPA reductions in gas lead. (It takes about a quarter for the effect to show up because the lower lead numbers must work their way through the gasoline inventory). Figure 4 shows the same pattern in the Nationwide lead screening data.

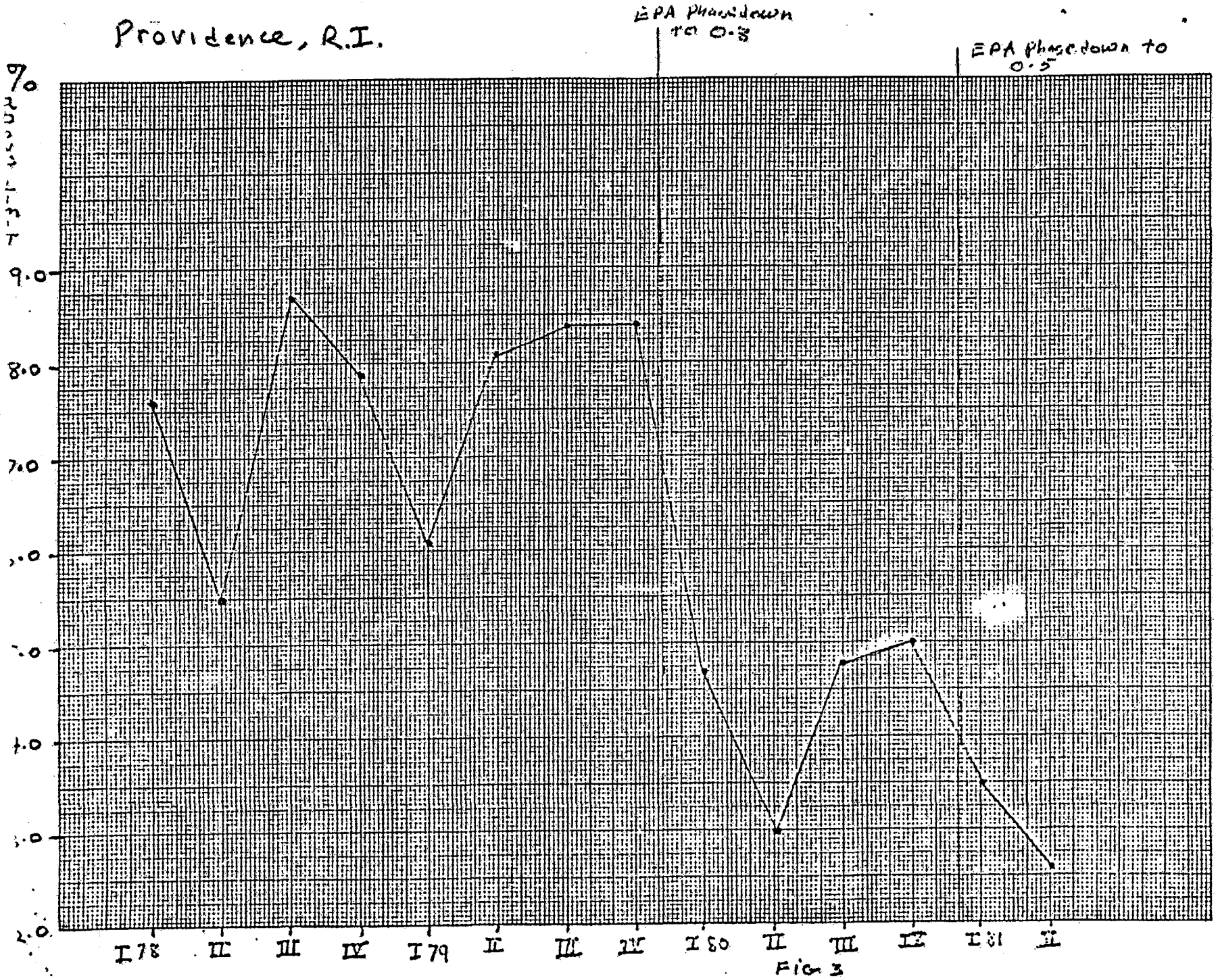

Fig. 3

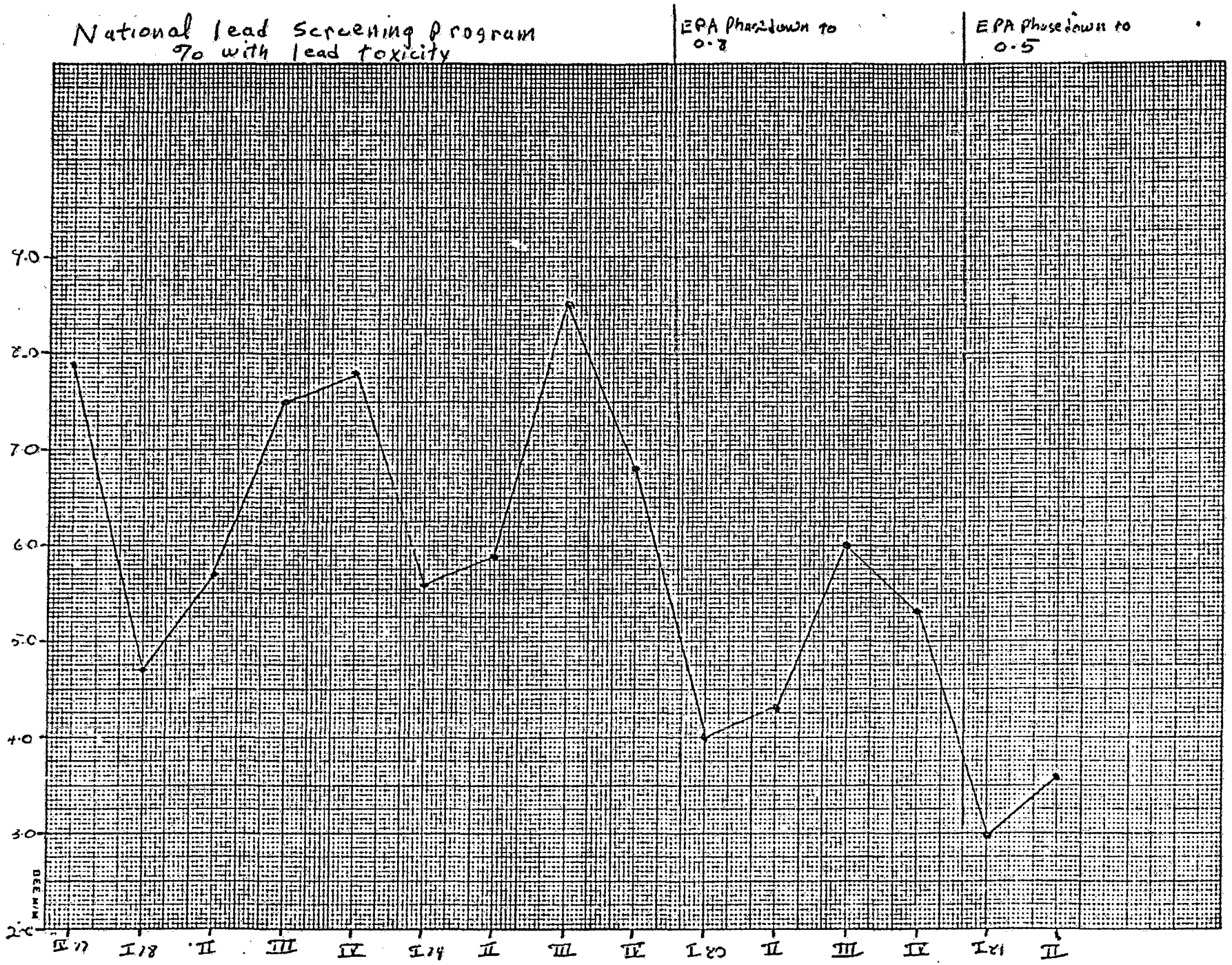

Fig 4

How Meaningful is the Regression

The NHANES II data and the regressions for Louisville, Chicago, New York and Providence show strong correlations between gas lead and blood lead in children, and if interpreted causally imply that relaxing EPA's lead standard could push an additional 2-4% of black and white pre-school children over the lead poisoning threshold in 1983. Since there are 16 million pre-school children in the United States, this could mean an additional several hundred thousand children above the danger level, even assuming much lower changes outside of urban areas. Eliminating the regulation would save $100 million in that year. This suggests (after allowing for uncertainties in the statistics) that it is likely that EPA's regulations cost between $500 and $2000 per child kept under 30 ug/dl. It is extremely likely that the cost lies between $250 and $4000 per child. However, this assumes that the regression is predictive. The central question is do we believe that and how much.

Regressions only show coincidence. To believe that they have predictive value, we need a qualitative causal model suggesting a connection between the two variables, and a reason to believe that much of the correlation between the two variables is not accidental.

There is good reason to believe there are causal links between gasoline lead and body lead in children. First gasoline lead is the major contributor of lead to air and it is recognized that inhalation of air lead leads to uptake of lead in humans. It is also recognized that gasoline lead is the major contributor to lead in dust and dirt in urban areas. For instance soil lead concentrations in the front of Cincinnati homes are three times the concentration of soil lead in the rear. Also, studies have shown high levels lead in household dust. A finger wiped across a table left undusted for three days will pick up several times the safe daily dose of lead for a child. Children often lick their hands or put them in their mouths. None of this quantitatively estimates the effects of air or dust lead on children's blood lead levels, but it suggests a theoretical link that justifies using the regression model.

A more serious question is whether other variables, not included in our data base, changed over the same time period, leading to an inaccurately high attribution of the variance in blood lead to gasoline. Some errors are obviously to be expected, which is why I use a range of $500-$2000 per case avoided. The question is whether there is a major error that puts us out of this range. There are a number of possibilities.

We did not have any variable in our data to cover lead paint exposure. As long as lead paint exposure did not vary over the time period, this would cause little problem. But what if it did? We can put this hypothesis in perspective by looking at some numbers. HUD has estimated that there are 30 million housing units in the United States with lead paint. It costs $5000 per unit to strip the lead paint from the walls, or a total national cost of $150 billion. There has been no program of anything like that order to remove lead based paint. Indeed, the CDC annual reports on the lead paint program show only several thousand to 10,000 units per year reported as having lead paint removed. Other local efforts may exist which are not reported to CDC, but the total must be small compared to 30 million housing units, and seems unlikely to be a significant contributor to the 37% drop in mean blood lead levels from 1976 to 1980. To further evaluate this issue, we asked MITRE Corporation to examine the Louisville data base including census tract in the regression.

Census tract can serve as a proxy variable for lead paint exposure. This is because some tracts will have fewer lead painted homes, because some tracts will have more peeling paint, increasing exposure, and because any paint removal programs probably varied widely by census tract. The attached report from MITRE showed that including census tract information caused little change in the regression coefficient for gasoline lead, and accounted for a small part of the variance in blood lead. While not a perfect proxy for lead paint exposure, this suggests that lack of such a variable has not significantly overestimated the effect of gasoline lead.

Another method of guaging the effect of not having such a variable in our data base is to look at age as a proxy for lead paint exposure. If the fall in mean blood lead and % of children over 30 ug/dl were due to fall in lead paint exposure, rather than gasoline lead exposure one would expect a much larger drop in the age categories that are more prone to eating paint, and a much smaller drop in other age groups. In fact, the 37% drop in mean blood lead levels found by NHANES II occurs in adults as well as children. It is difficult to believe adults eat much lead paint. Again, looking at the regression coefficients, we see the following:

Does This make sense?

	Ln % greater than 30 ug/dl		
	N.Y.	Chicago	Louisville
Coefficient			
gas lead	2.04	1.46	29.6
1-2 year	.28	.46	.34
2-3 year	.45	.71	.50
6-7 year	.16	.44	not statistically significantly

The difference between the age coefficients is small compared to the gasoline value. If changes in lead paint exposure were the major cause of the drop in blood lead, we would expect to see larger differences between 1-3 year olds and 6-7 year olds. The raw data does not bear this out.

Between I Q 75 and I Q 79 in Chicago cases above 30 ug/dl went from

13.8% to 5.0% for blacks aged 1-2

17.4% to 0% for blacks aged 6-7

While these results have a lot of noise because of the small age sample, an examination of the raw data over the years shows six year old blood lead dropping about as fast as one year olds, which does not support the hypothesis that changes in lead paint exposure caused a significant fraction of the general decline in blood lead that the regression attributes to gasoline lead. Table 5 in Billick's report shows the age variation of blood lead to be small. Also the following figures, from the study by Billick, Curan, and Shier, show that blood lead in young children (1-12 months, 13-24 months) show the same seasonal peaks as gas lead. The raw data is in Appendix A.

A second hypothesis is that the decline in the lead content of canned food caused a significant fracton of the general decline in blood lead that the regression attributes to gasoline. The FDA has in the last five years been working with the canning industry to bring this about. A number of items suggest that this too should not lead us to discount the regression results.

First of all, figures 9, 10 and 11 in the Billick study show the regression line for percent of children over the safety limit plotted through the actual data points. The data points to the right (e.g. high lead levels) correspond to years that predate the canning industry's major lead removal efforts. In the case of Chicago (Figure 9) this includes data back to 1967. Eyeballing the graphs shows that the lines agree well with the early data, where there was no canned food program to attribute the decline in blood lead to.

Secondly, the age data again suggests that we are not misattributing the decline in blood lead. Looking at the data for children 0-11 months (who eat little canned food), we see similar drops in % greater than 30 ug and in mean blood levels as we see in older children. The correlation coefficients are also similar. Table 5 of the Billick report again supports this so do the enclosed figures.

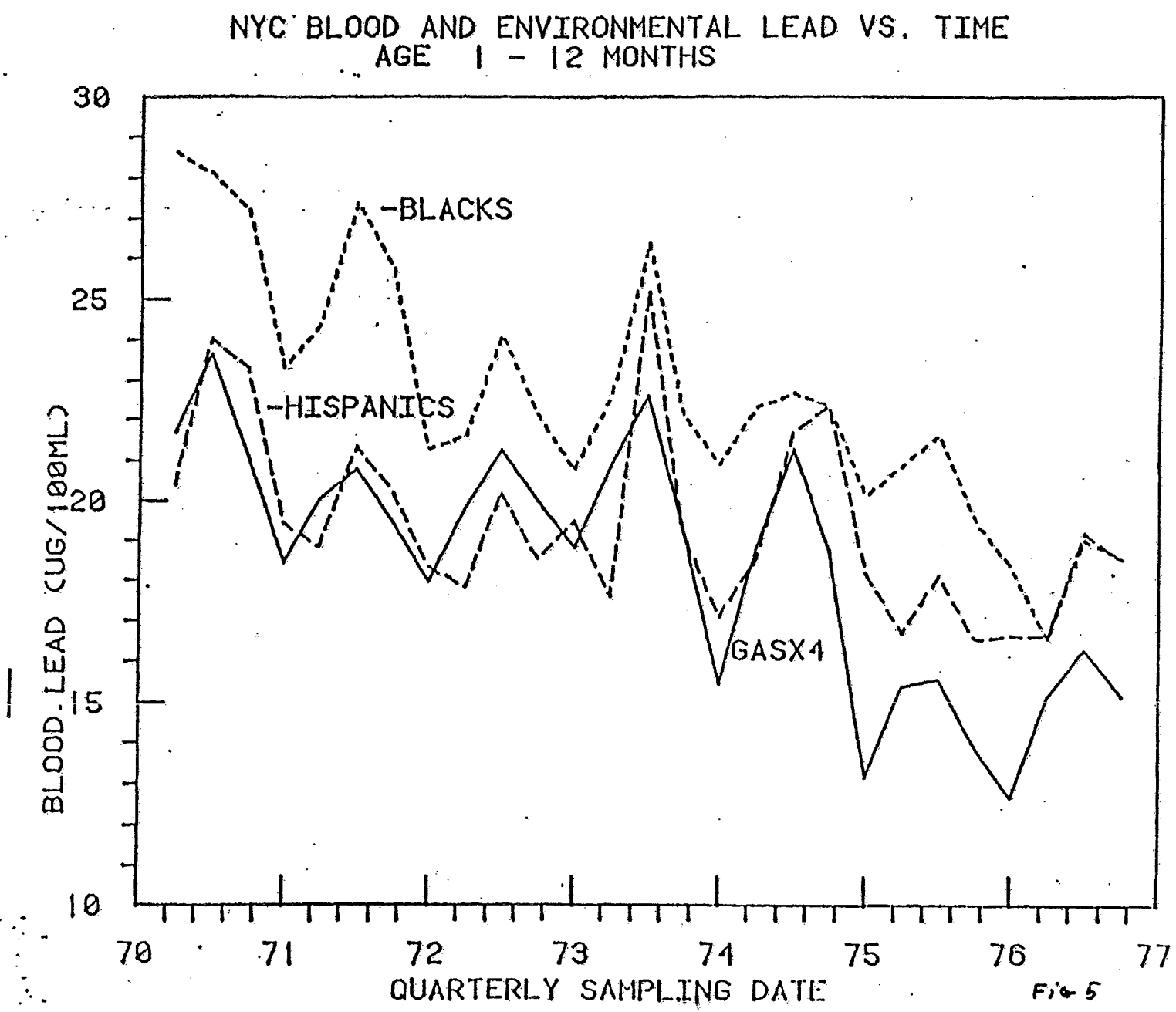

Fig 5

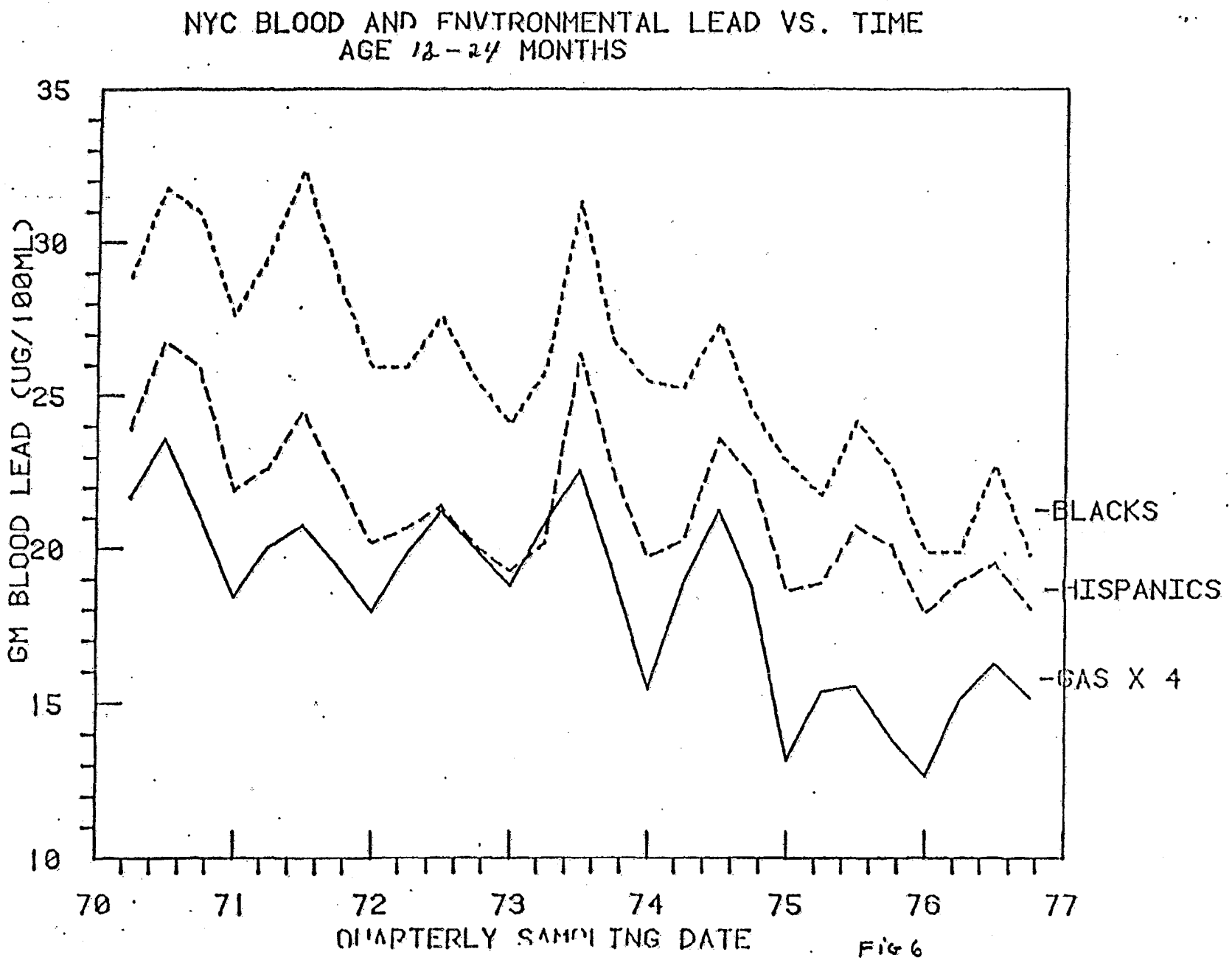

FIG 6

NYC BLOOD AND ENVIRONMENTAL LEAD VS. TIME
AGE 25-36 MONTHS

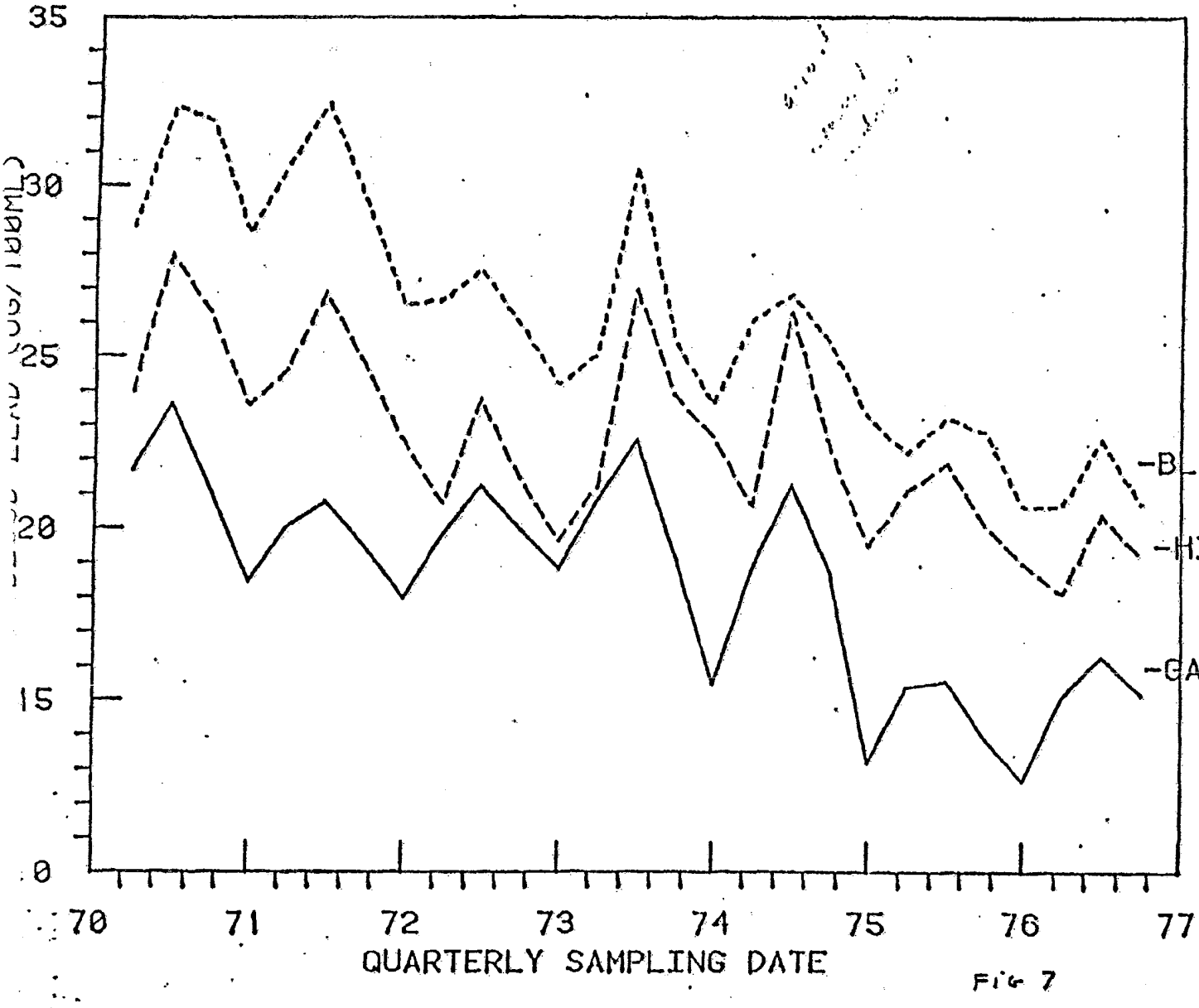

Fig 7

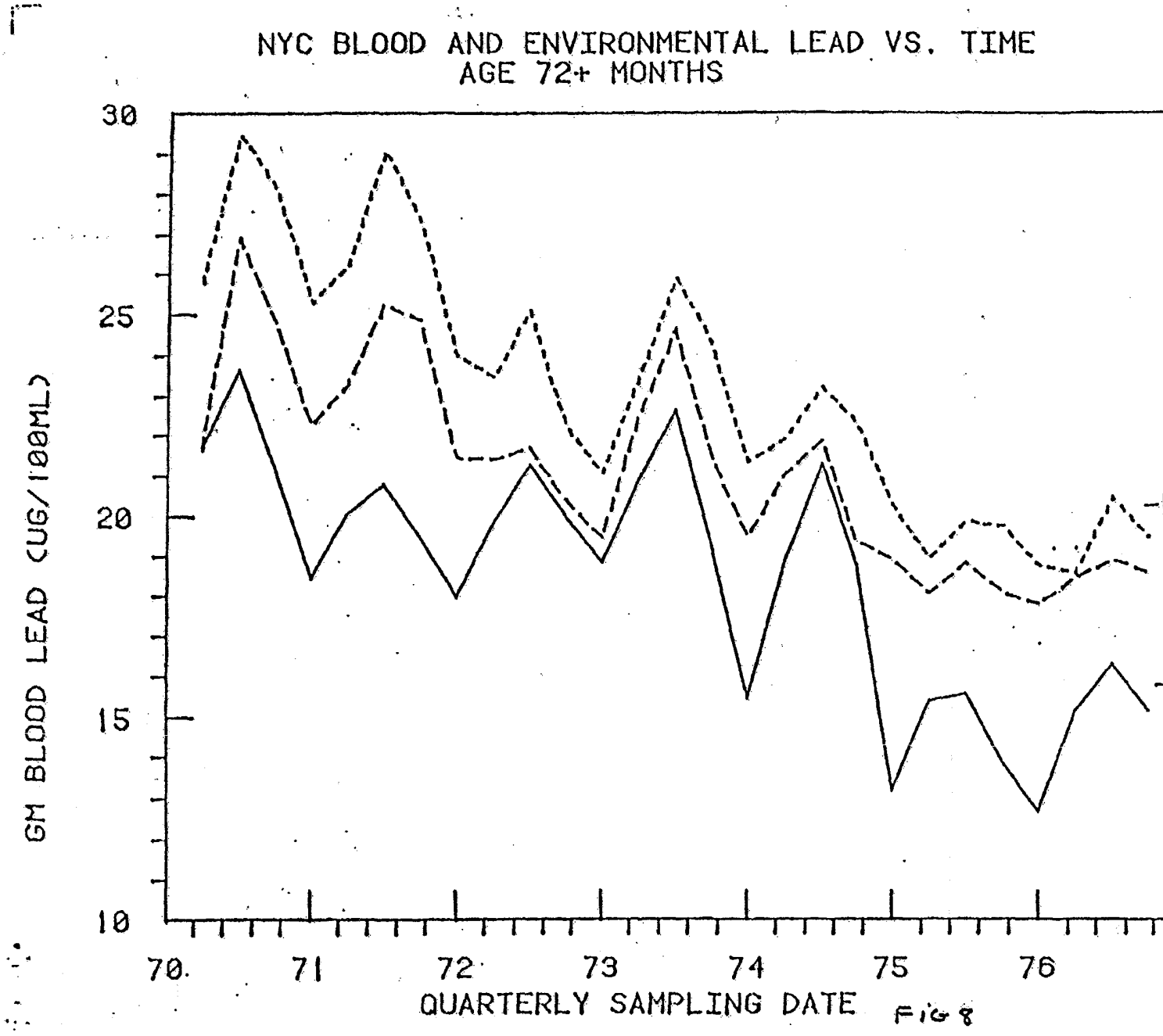
NYC BLOOD AND ENVIRONMENTAL LEAD VS. TIME
AGE 72+ MONTHS
GM BLOOD LEAD (UG/100ML)
30
25
20
15
10
70
71
72
73
74
75
76
QUARTERLY SAMPLING DATE

FIG 8

There is one final factor that argues against specification error causing gross error in the gasoline regression coefficient. This is the seasonal variation in the gas lead and blood lead data. Figure 1, the NHANES II data shows five seasonal excursions of the gas lead data. All five are matched by similar excursions in the blood lead data. (One additional blood lead excursion occurs). This pattern holds true for the quarterly data for New York, Chicago, and Louisville as well. Figures 1, 2 and 3 of the Billick report show gas lead and blood lead over time for all three cities. All show that quarterly peaks in gas lead almost always are associated with quarterly peaks in blood lead. Figure 4 shows that when the blood leads of all three cities are plotted together, the peaks coincide. In particular, they peak in mid year. Summer is the time both of maximum daily gasoline useage and of maximum lead content per gallon. (Because of the high temperatures, less low vapor pressure high octane components can be used in the summer. Extra lead is usually substituted to make up for this octane loss.) Figures 6, 7 and 8 show the same seasonal trend occurs for the percent of children above 30 ug/dl. The fact that the number of children above the safety level has the same variation as the mean blood level, and peaks when gas lead peaks is highly suggestive.

TRUE?

WOW!!

All of this suggests that the regression is not likely to be grossly wrong. Therefore $500-$2000 per child kept under 30 ug/dl seems like a reasonable estimate of what we are paying under this regulation.

Benefits of Gasoline Lead Rules

We can estimate the cost of gasoline lead rules fairly easily. Benefits are more difficult to assess, and we have not commissioned a formal benefit study. Nevertheless, we can make some attempts to get a handle on it. As pointed out in the previous section, the regression seemed to suggest that total relaxation would cause about an additional 2% of pre-school in urban areas to exceed 30 ug/dl. Assuming that lower increases prevail in rural areas, this suggests about 1-1 1/2% of all pre-school children, or about 200,000 children. In addition, children above five are also predicted to exceed the standard. Allowing room for error in the regression 50,000 seems the minimum reasonable estimate nationwide (the regression predicts over 10,000 black children in N.Y. and Chicago alone). 500,000 is probably the maximum. How much is it worth to prevent this?

At a minimum, it must be worth the cost of medical care for these children. This can be estimated.

Suggested Treatment and Cost

In 1978 the Centers for Disease Control published a statement on "Preventing Lead Poisoning in Young Children" (Journal of Pediatrics, Vol. 93, N04 pp. 709-720). In it CDC suggests provocative EDTA testing for children at risk but below 70 ug/dl, stating "it is particularly useful when the screening tests indicate that the child has undue lead absorption and there is some question as to whether chelation therapy is indicated." The ideal suggested method involves 2 doses at 12 hr. intervals with a 24 hr. urine collection. A ratio of >1 mg Pb/mg CaEDTA in the amount of lead excreted per EDTA injected "is indicative of a fivefold increase in the mobile or potentially toxic fraction of the total body lead burden. Correlation studies suggest that such levels are associated with a significantly increased risk of toxicity due to lead." A positive reading (>1) suggests a full EPTA therapy. A negative reading suggests further monitoring and a search for sources.

Dr. Rosen of the Albert Einstein Medical Center testified on the results of his use of this approach in New York. In his testimony and a follow-up phone call, he stated that

- provocative EDTA testing and follow-up required 3-4 days hospitalization for negative results
- positive readings resulted in 10 days hospitalization for full chelation therapy
- in either case, high lead levels required follow-up monitoring for several months of bi-weekly outpatient visits
- children with blood leads between 30 ug/dl and 40 ug/dl had positive results 40% of the time.
- children with blood leads between 40 and 55 ug/dl had positive results 80% of the time
- hospitalization cost $450/day
- outpatient visits cost $45/visit, including testing.

Putting this together, I conclude:

- o every child over 30 ug/dl should be tested by EDTA
- o at least 40% of those tested will require $4,500 in treatment and $270 in outpatient visits for a total cost of about $4,800

- o 60% at those tested will require $1350 in hospital costs and $270 in outpatient visits for a total of about $1600.

The regression predicts about 11,000 additional black children in Chicago and New York would exceed 30 ug in 1983 if we recinded the rule. If they were all detected, tested and treated, this would cost $32 million. Nationwide, we would incur a cost of $140 million to $1.4 billion. This is significantly higher than the cost of the regulation. Clearly, all the additional children moved over 30 ug will not be tested. But if the medical treatment is reasonable, it means we would test them if we could find them. Since the expected medical cost per child is close to $3,000 (averaging between positive and negative EPTA results) even this rough analysis suggests that recision is not cost effective. Even 0.65 gpg would, according to the regression, cause about 4,000 children in Chicago and New York to exceed the safety limit and 20,000-200,000 nationwide. This would cost $12 million in medical expenses in New York and Chicago, and $60-600 million nationwide. This compares to $22 million in cost savings from relaxing the standard.

Special Education Costs

Medical costs are not the only costs incurred in children with unsafe lead levels. Lead can cause temporary or permanent learning disabilities. While permanent significant mental retardation is usually associated with extremely high blood lead levels, lower levels have been associated with mild learning disabilities. Arguments still remain over whether these effects are permanent.

Conservatively, it is prudent to assume that children who, under provocative testing excrete amounts of lead similar to children with frank lead poisoning will probably require some special education. Assuming this is of short duration, it may be assigned a low cost, perhaps $2000.

If 40% of additional cases over 30ug require this additional education, then between 20,000 and 200,000 children in 1983 will incur this cost under complete recission. This is an additional $20-200 million. A relaxation to 0.65 gpg is likely to effect 8000-80,000 children at a cost of $8 million to 80 million. The totals are summarized below.

Additional Costs of Increased Lead Use in 1983 (in millions)

	Medical	Educational	Total
0.65 gpg	$60-600	$8-80	$68-680
1.0 gpg	140-1,400	20-200	160-1,600

IQ Effects

The question of whether IQ and other higher order cognitive processes are affected by lead is still quite controversial. Needleman and Yule have found that there are such effects and that they occur at levels below 30 ug/dl. Ernhart has not found such effects in her studies. Recently, Thatcher published hair lead studies of rural children showing a continuous relationship between lead and IQ. Finally, Otto has performed studies of EEG patterns and lead that also show changes at lead levels well below 30 ug/dl, although the significance of the EEG changes is not clear.

Given this uncertainty, we might wish to make decisions on an expected value basis. That is, we could multiply the probability of effect by the cost, and use that as our expected cost. In the absence of any delphi estimate of what the probability distribution is, we can invert the process for EPA decision makers. That is we can frame the question as:

"The cost of effect A, if it occurs, is $100 million, and the cost of avoidance is $10 million. Therefore, you must be at least 10% certain it will occur before it is worth the avoidance costs."

How can we quantify the costs however? Well, if the Needleman study is correct, it suggests that recission of the rule might reduce the mean IQ of pre-school urban children by about one point. Several regressions have been run between IQ and expected lifetime income. They generally predict, after adjustment for socio-economic factors, an IQ point is worth between 1/2% and 2% of lifetime income.* Assuming the average child will, in 20 years, begin earning $20,000/yr in 1982 dollars, that the social discount rate is 6% real, and that real earnings will increase at 2% per year, the present value of lifetime earnings of a child born today is $250,000. Taking the low estimate to be conservative, One half a percent of this is about $1100. If 3.7 million births occur in 1983, the cost of relaxation to the children born that year will be about $4 billion. There are great uncertainties in this estimate, which is not a prediction. They include questions about the correlation between income and IQ and questions about whether lead in

* Inequality, Jencks, et al, Basic Books 1974. ch. 4.

fact effects IQ. The number is primarily valuable in the following sense.

We estimate the cost of keeping the current lead rule at about $100 million. Therefore, if the likelihood of the above scenario occuring is 2.5% or greater then independent of the medical and educational costs cited above, EPA should maintain its current standard.

Controversy Over New York Data

Some of this information has been previously published in scientific journals by HUD scientists (See Figure 9).[2] The rest is an extension of those studies by Dr. Billick done at EPA's request.[3] When the original studies on New York came out, Dupont criticized the gasoline lead numbers that were used, claiming that New York had had a local phasedown rule in effect during the period, and that the actual lead content was lower. They also claimed that the Motor Vehicle Manufacturers data for New York was incorrect.

New York did enact a local lead ordinance in 1971, but there is no evidence that it was enforced, and indeed was dismissed in court. The regulation was stayed on 3/26/73 pending suit in U.S. District Court. In 1974, Exxon lost in District Court. They appeal to the second circuit and hearings were postponed until a decision was reached on EPA's lead rules. No action was taken by New York to enforce the rule between 4/74 and 1976 when it was overturned. This suggests that the MVMA data is more accurate, since the data cited by Dupont shows no effect of the stay. In any case, much of the gasoline burned in the city was purchased in the suburbs, so Dupont's estimate of the average lead content burned in New York would be too low even if their survey data were correct. That is why we used SMSA data, which includes the commuter suburbs. Nevertheless this argument complicated using the New York results.

To resolve the issue, we decided on a two-fold approach: We would rerun the New York study using only data from 1974 on (post stay) to minimize the controversy, and we would study other cities. As can be seen above, the other cities show the same general pattern of correlations between blood lead and lead emissions from gasoline as New York. In addition to the regression results, the coefficient of correlation between blood lead and gasoline lead was 0.835 for blacks in New York, 0.636 for blacks in Louisville, 0.660 for

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2 Billick, Curran, and Shier. Environmental Health Prospectives Vol 34, 214, 1980

3 Billick, I.H. "Prediction of Elevated Pediatric Blood Lead Levels from Gasoline Consumption. Draft April 2, 1982

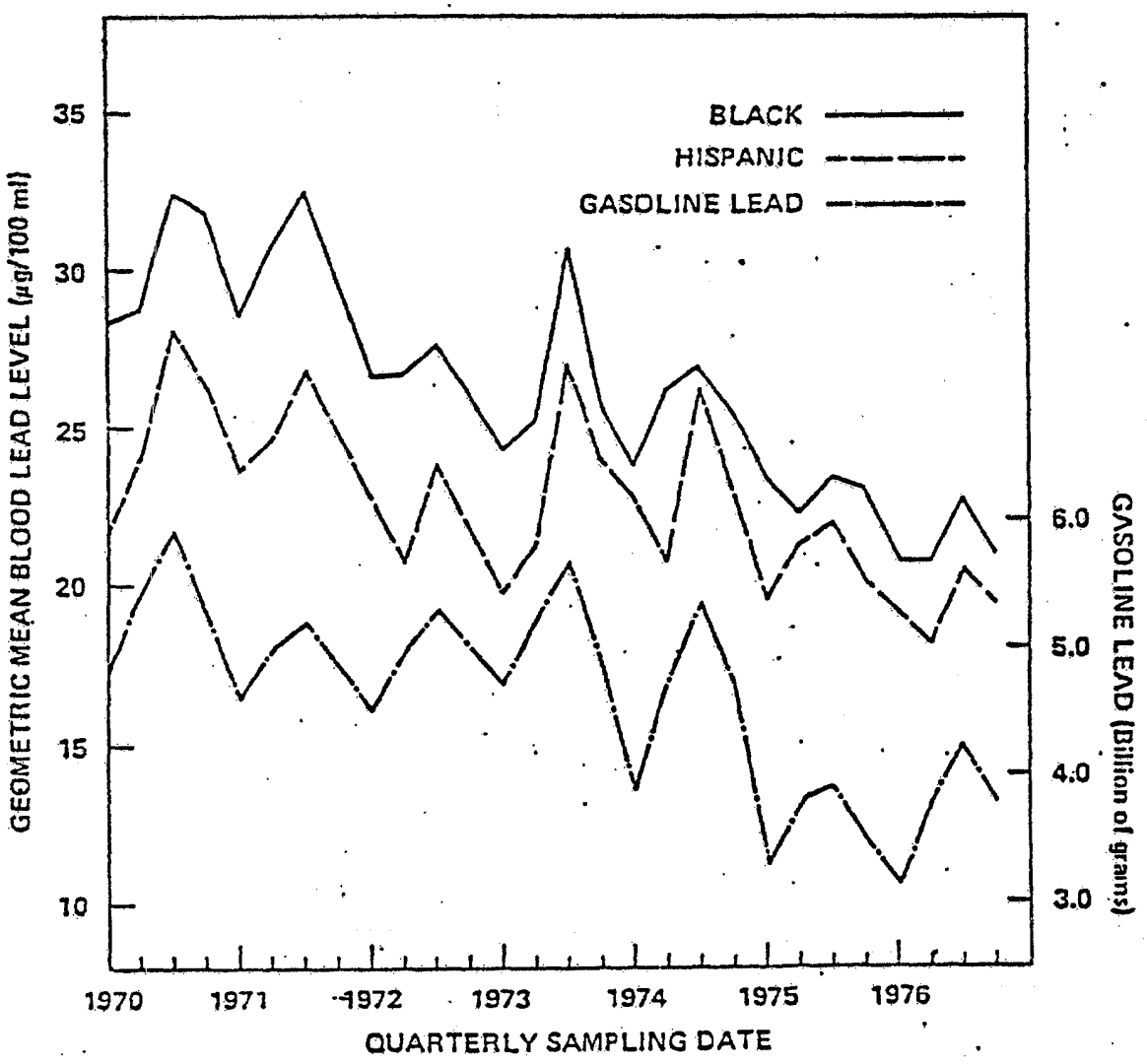

Fig. 9

TEH 0531297

DUP050032508

blacks in Chicago, 0.8641 in Providence, and 0.95 in CDC's NHANES II analysis.

Addressees

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Docket A-81-36

Table I

Chicago - Percent of all children with lead poisoning (>30 ug/dl)

Blacks, July-September Quarter, 24-35 months and 72-84 months

Lead Limit	1983		1984		1985	
	2-3 yr.	6-7 yr.	2-3 yr.	6-7 yr.	2-3 yr.	6-7 yr.
0.5 gpg	18.5%	14.0%	17.9%	13.6%	18.1%	13.7%
0.65 gpg	19.8%	15.0%	18.9%	14.3%	19.0%	14.4%
0.8 gpg	20.7%	15.7%	19.3%	14.6%	19.0%	14.4%
1.0 gpg	21.1%	16.0%	19.3%	14.6%	19.0%	14.4%
no limit	21.1%	16.0%	19.3%	14.6%	19.0%	14.4%

% increase in lead poisoning cases from current law

0.5	-----	-----	-----
0.65	7%	5.6%	4.4%
0.8	11.9%	7.8%	4.4%
1.0	15%	7.8%	4.4%

Chicago city sources estimate that there are 226,000 black pre-school children in the city.

Table II

New York - Percent of all children with lead poisoning (>30 ug/dl)

Blacks, July-September Quarter, 24-35 months and 72-84 months

Lead Limit	1983 2-3 yr.	1983 6-7 yr.	1984 2-3 yr.	1984 6-7 yr.	1985 2-3 yr.	1985 6-7 yr.
0.5 gpg no small	12.9%	9.58%	12.8%	9.5%	12.8%	9.5%
0.65 gpg a small	14.08%	10.46%	14.0%	10.4%	13.7%	10.2%
0.8 gpg a small	14.92%	11.08%	14.6%	10.7%	13.7%	10.2%
1.0 gpg a small	15.37%	11.42%	14.6%	10.7%	13.7%	10.2%
No limit a small	15.37%	11.42%	14.6%	10.7%	13.7%	10.2%

% increase in lead poisoning cases from current law

0.5	———	———	———
0.65	9.1%	9.4%	7.0%
0.8	15.7%	14.1%	7.0%
1.0	19.1%	14.1%	7.0%

New York city sources estimate that there are about 220,000 black pre-school children in the city.

Table III

Louisville - Percent of all children with lead poisoning (>30. ug/dl)

Blacks, July-September Quarter, 24-35 months and 72-84 months age

Lead Limit	1983		1984		1985	
	2-3 yr.	6-7 yr.	2-3 yr.	6-7 yr.	2-3 yr.	6-7 yr.
0.5 gpg	10.1%	6.1%	10.2%	6.2%	10.35%	6.2%
0.65 gpg	12.26%	7.4%	12.4%	7.5%	12.2%	7.4%
0.8 gpg	13.9%	8.4%	13.3%	8.1%	12.2%	7.4%
1.0 gpg	14.8%	8.9%	13.3%	8.1%	12.2%	7.4%
No limit	14.8%	8.9%	13.3%	8.1%	12.2%	7.4%

% increase in lead poisoning cases from current law

0.5	-----	-----	-----
0.65	21%	22%	18%
0.8	38%	30%	
1.0	47%	30%	

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Table IV

Chicago - percent of all children with lead toxicity (>30 ug/dl)

Whites, July-September Quarter, 24-35 months

Lead Limit	1983	1984	1985
0.5 gpg	11.6	11.2	11.3
0.65 gpg	12.4	11.8	11.9
0.8 gpg	12.9	12.0	11.9
1.0 gpg	13.2	12.0	11.9
	% increase in cases from current law		
0.5	--	--	--
0.65	7%	5%	5%
0.80	11%	7%	5%
1.0	14%	7%	5%

Table V

New York - percent of all children with lead toxicity (>30 ug/dl)

Whites, July-September Quarter, 24-35 months

Lead Limit	1983	1984	1985
0.5 gpg	10.0	9.9	9.9
0.65 gpg	10.9	10.8	10.7
0.8 gpg	11.6	11.2	10.7
1.0 gpg	11.9	11.2	10.7
	% increase in cases from present law		
0.5	--	--	--
0.65	9%	9%	8%
0.8	16%	13%	8%
1.0	19%	13%	8%

Table VI

Louisville - percent of all children with lead toxicity (>30 ug/dl)

Whites, July-September Quarter, 24-35 months

Lead Limit	1983	1984	1985
0.5 gpg	8.5	8.6	8.7
0.65 gpg	10.3	10.4	10.2
0.8 gpg	11.6	11.2	10.2
1.0 gpg	12.4	11.2	10.2
	% increase in cases from current law		
0.5	--	--	--
0.65	21%	21%	17%
0.80	36%	30%	17%
1.0	46%	30%	17%