Approved: 2-10-98
Date

MINUTES OF THE SENATE COMMITTEE ON PUBLIC HEALTH AND WELFARE.

The meeting was called to order by Chair Sandy Praeger at 10:00 a.m. on February 4, 1998 in Room 526-S of the Capitol.

All members were present except:

Committee staff present: Emalene Correll, Legislative Research Department
Robin Kempf, Legislative Research Department
Norman Furse, Revisor of Statutes
Jo Ann Bunten, Committee Secretary

Conferees appearing before the committee:

Gary Mitchell, Secretary, Kansas Department of Health and Environment
Sam Umscheid, Wyandotte County Health Department
Debbie Nickels, Kansas Association of Local Health Departments
Patrick DeLapp, Shawnee County Landlords of Kansas

Others attending: See attached list

Hearing on SB 437 - Prevention of child lead poisoning

Gary Mitchell, Secretary, Kansas Department of Health and Environment, testified before the Committee and offered a Substitute bill for **SB 437** which would address childhood lead poisoning prevention, accreditation of training programs related to lead-based paint activities and licensure and/or certification of individuals, business entities and public agencies who provide, engage in or conduct lead-based paint activities. Secretary Mitchell noted that the bill would create a program subject to appropriations, and that a lead-based paint hazard fee fund account would be established. He also pointed out that if the state does not have statutory authorities by August 31, 1998, EPA will establish the program in Kansas. (Attachment 1) During Committee discussion on provisions in the bill, Secretary Mitchell noted that a fiscal note would be provided to the Committee on the substitute bill.

Sam Umscheid, Wyandotte County Health Department, testified in support of the bill and provided the Committee with a breakdown of the population of low income families in Wyandotte County that would be benefitted by passage of this bill. Mr. Umscheid expressed his concern that if the bill does not pass, HUD dollars would not be available to the county. (Attachment 2)

Debbie Nickels, Kansas Association of Local Health Departments, testified before the Committee in support of **SB 437** and stressed the need that funding be made available to local health departments for blood lead screening, public and professional education, case management, nutritional intervention and environmental lead assessments as noted in her written testimony. (Attachment 3)

Patrick DeLapp, Shawnee County Landlords of Kansas, spoke in opposition to the bill because he felt that the bill would set up another state bureaucracy. He noted that the federal government will soon have their own regulations addressing lead poisoning and provided information to the Committee on the decline in blood lead levels in the United States as well as other written material on the subject. (See Attachment 4)

Written testimony was also provided to the Committee from Karen France, Kansas Association of Realtors, in opposition to **SB 437.** (Attachment 5)

Because of lack of time, the Chair announced that the hearing on **SB 437** will be continued at a later date in order to hear other conferees testify on the bill.

Adjournment

The meeting was adjourned at 11:00 a.m.

The next meeting is scheduled for February 5, 1998.

SENATE PUBLIC HEALTH AND WELFARE COMMITTEE
GUEST LIST

DATE: 2-4-98

NAME	REPRESENTING
Carolyn Middendorf	KSNA
[illegible]	KALHD
Sam Umscheid	Wyandotte County Health
Susan Anderson	Hein & Weir
Bill Allen	Kickapoo Tribe in Kansas
Sally Finney	Kansas Public Health Association
Pat Dehoff	S.C.L.A. Shawnee County Landlord Assoc
Jo Funk	Salina-Saline Co. Health Dept./KAS/Ks. Lead Council
[illegible]	KDHE
Mike Meacham	-
Amy Lignitz	"
Dawn Reid	KDNA
Rich Guthrie	Health Midwest
~~ED Sanstiro~~	~~T.A.K.~~

KANSAS
DEPARTMENT OF HEALTH & ENVIRONMENT
BILL GRAVES, GOVERNOR
Gary R. Mitchell, Secretary

TESTIMONY PRESENTED TO

THE SENATE PUBLIC HEALTH AND WELFARE COMMITTEE

Wednesday, February 4, 1998

by

Gary R. Mitchell
Secretary of Health and Environment

SENATE BILL 437

Madam Chair and members of the committee, thank you for the opportunity to appear before you today to discuss Senate Bill 437. I apologize for delaying the hearing scheduled last week. My staff notified me that the original bill draft I provided to the committee needed some corrections; rather than try to do so in committee I felt it would be more time-efficient and less confusing to provide you with a "clean" version this week. I appreciate your patience and understanding.

SB 437 Addresses:
- Childhood lead poisoning prevention,
- Accreditation of training programs related to lead-based paint activities,
- Licensure and/or certification of individuals, business entities and public agencies who provide, engage in, or conduct lead-based paint activities.

PREVENTION

Centers for Disease Control and Prevention (CDC) Estimates
- 4.4% of children 6 months - 6 years have elevated levels of blood lead
- CDC estimate for Kansas = 10,600 children

Children ages 1-3 highest risk; major exposure due to dust in home contaminated by deteriorating lead based paint.

900 SW Jackson, Suite 620
(785) 296-0461

Consequences of Lead Poisoning in Children

- Adverse effects on learning, behavior and growth
- High levels can cause seizures, coma and death.

Benefits of Prevention

- Avoided medical and special education costs
- Increased lifetime earnings
- Reduced infant mortality

ACCREDITATION and LICENSURE

Directed at meeting requirements of Section 402 of the Toxic Substances Control Act, as required by the Residential Lead-Based Paint Hazard Reduction Act of 1992 (administered by EPA).

X **Federal Law requires programs in place by August 31, 1998.**
If Kansas does not have statutory authorities by that date, EPA will establish the program in Kansas.

Section 1018 of the federal law: Joint EPA/HUD rules

- Requires disclosure of lead-based paint hazard by the seller or landlord of housing built prior to 1978, and the seller/landlord must provide pamphlet on possible hazards of lead-based paint.
- Provides a buyer the opportunity to have home inspected by qualified individual.
- Inspectors must be certified to make the lead-free determination.

Results

- Since Kansas does not currently have program to certify inspectors - buyers must hire inspector from another state, or one who has become certified in another state. Kansas is relying on certification programs in Missouri, Iowa, Nebraska, Oklahoma.
- Kansas has approximately 800,000 dwellings painted with lead-based paint.

EPA has federal grant funds available for states to establish licensing/accreditation programs.

- Kansas is in the first year of funding from EPA for lead poisoning prevention
- Eligibility will be lost if a licensure/certification/accreditation program is not authorized by August 31, 1998.

Kansas Lead Council

- Established by Secretary Mitchell June, 1997
- Formed to provide communication between KDHE, industry, EPA, Local Health Departments, and grass roots organizations representing the public.
- Council has met 4 times to discuss lead poisoning prevention in Kansas, and provided input to SB 437.

Role of Local Health Departments in Childhood Lead Poisoning

- Parent education,
- Blood screening,
- Medical Management
- Environmental Assessments

KDHE has had no funds available to support local activities for medical and environmental follow-up.

SUMMARY

SB 437 would provide:

- Framework for more comprehensive statewide childhood lead poisoning prevention program,
- Licensing/Certification program directed at assuring qualified firms/individuals are available to perform lead abatement work.
- Kansas determined program, rather than one established by EPA.

Childhood lead poisoning will some day be a thing of the past. Until then, many children will benefit from a coordinated effort to reduce exposure to environmental lead. SB 437 contains the tools to support that coordinated effort. This bill does contain a "Sunset" provision of 2003.

I appreciate your time today and your consideration of this bill. I would be pleased to answer any questions from the committee.

Wyandotte County Population/Births

Year	Population	Births
1996	--------	2,570 (preliminary)
1995	153,826 (estimate)	2,620
1994	155,075 (estimate)	2,722
1993	--------	2,820
1992	158,704	2,878
1991	--------	2,907
1990	162,026	3,103
1980	172,335	3,522
1970	186,845	3,874
1960	185,495	5,184

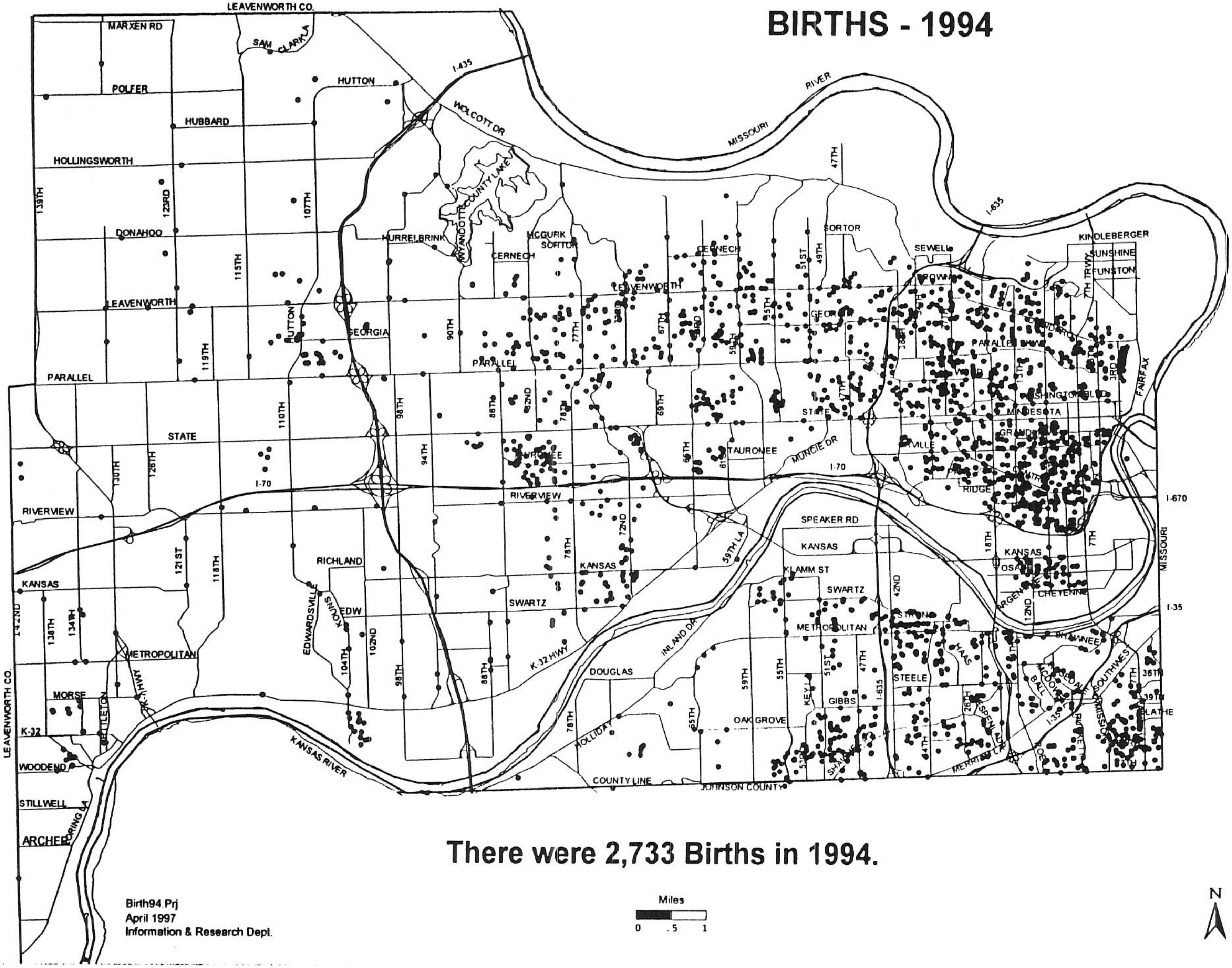
1980: 11.0% of families below poverty level.
13.9% of persons below poverty level.

1990: 13.9% of families below poverty level.
17.1% of persons below poverty level.

Senate Public Health & Welfare
Date: 2-4-98
Attachment No. 2

WYANDOTTE COUNTY
BIRTHS - 1994

2-1



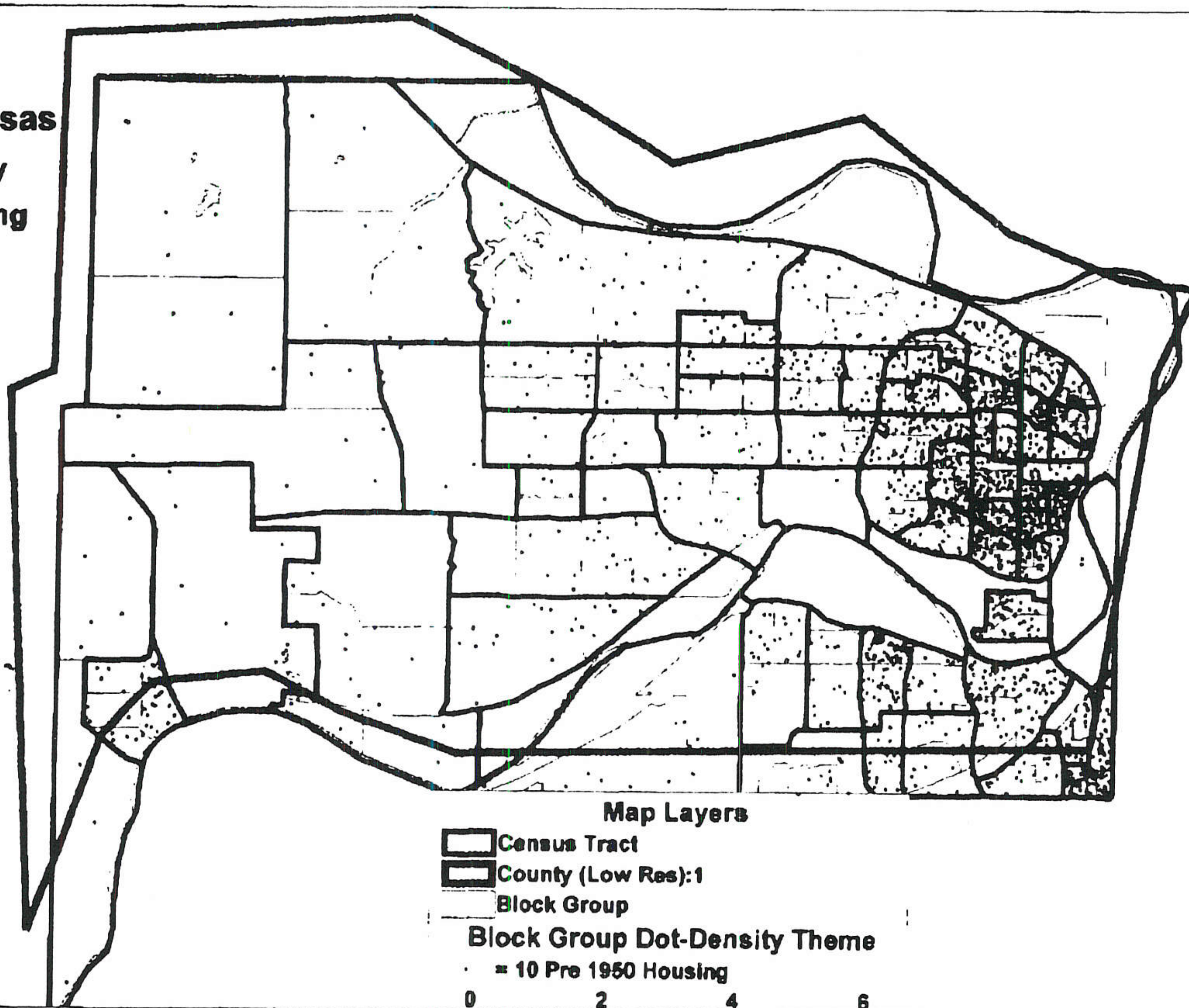
There were 2,733 Births in 1994.

Birth94.Prj
April 1997
Information & Research Dept.

Kansas City, Kansas

Wyandotte County

24,283 Pre 1950 Housing



K·A·L·H·D

TESTIMONY TO THE SENATE PUBLIC HEALTH AND WELFARE COMMITTEE
1-29-98

Dear Madame Chairman, and Committee members,

My name is Debbie Nickels and I am the Administrator/Health Officer for the Jefferson County Health Department. I am here today on behalf of the members of the Kansas Association of Local Health Departments to support a Childhood Lead Poisoning Prevention Program for Kansas through SB 437. Attached is a copy of our issue paper that the Kansas Association of Local Health Department's board voted on in May of 1997.

In our discussions with Secretary Mitchell we assured him of our commitment to assist with this most needed public health program for Kansas children. We understand that there are still many issues that need to be addressed including adequate resources for both the state and local health departments to efficiently and effectively add this comprehensive program to our already fiscally stressed infrastructure. Those few lead environmental assessments local health departments have been involved in are labor intensive, and require extensive case management services to educate and assist families in seeking appropriate medical treatment and follow up, developmental evaluations, home re-mediation, and/or new housing.

The Association did request of the KDHE lead committee that environmental assessment license/certification and training fees be waived for local health departments and that those individuals across the state who have met the training requirements be "grandfathered" into the program. I understand that these requests were inadvertently left out of the legislation. We ask that these issues be addressed and added to the legislation.

I wish to reiterate that the benefits of preventing lead exposure for children and fetuses has been extensively studied by the Centers for Disease Control. The quantified benefits are:

- Reduced Medical Costs
- Reduced Special Education costs
- Increased Future Productivity
- Reduced Infant Mortality

The weight of the evidence clearly supports that decrements in children's cognition are evident at blood lead levels well below 25 micrograms/deciliter. Let me point out that lead exposure can be a by-product of poverty, and a contributor to the cycle that perpetuates and deepens the state of being poor.

By adopting a Childhood Lead Poisoning Prevention Program for Kansans, we can decrease lifetime health and education costs, and have a win-win situation. Thank you for your consideration to this legislation.

KANSAS ASSOCIATION OF LOCAL HEALTH DEPARTMENTS

P.O. BOX 780406
WICHITA, KANSAS

67278-0406

PHONE: 316 • 684 • 0624

Senate Public Health and Welfare
Date: 2-4-98
Attachment No. 3

KANSAS ASSOCIATION of LOCAL HEALTH DEPARTMENTS

FY 1999 Issue Paper
Lead Program

I. Issue Definition

Childhood lead poisoning resulting from environment exposure is estimated by the Centers for Disease Control (CDC) to affect 4.4%[1] of children ages six months to six years. Contrary to the national findings of childhood lead poisoning being a urban inter-city problem, in Kansas childhood lead poisoning is four times higher in rural areas than for urban areas; urban - 126.0 per 100,000, rural 510.3 per 100,000[2]. Preliminary test data shows that as many as 15% (35,000) of Kansas children may have blood lead levels above the action level of 10 mcg/dL[3].

Addressing this issue requires a statewide comprehensive lead program consisting of public and professional education, lead screening, case management, medical management and environmental assessment.

II. Background

Lead poisoning is one of the most common and preventable pediatric health problems today. Lead is a highly toxic metal, producing a range of adverse health effects, particularly in children and fetuses. Effects include nervous and reproductive system disorders, delays in neurological and physical development, cognitive and behavioral changes and hypertension.

Data indicates significant adverse effects of lead exposure in children at blood lead levels previously believed to be safe. Some adverse effects have been documented at blood lead levels at least as low as 10 micrograms per deciliter (mcg/dL) of whole blood.

Most lead poisoned children do not appear to be sick. There are usually no symptoms unless a child is severely poisoned. Therefore, elimination of lead hazards before children are poisoned must receive more emphasis.

Lead poisoning is widespread. No socioeconomic group, geographic area or ethnic population is spared. Children ages six to 72 months are most at risk. Preliminary test data shows that many children are subject to adverse health effects from lead poisoning.

The risk to Kansas children is great. Risk factors include: living near lead-based industrial facilities or having a family member employed in such a facility, hobbies with lead exposure such as stained glass, living in older houses where lead solder was used in the plumbing and exposure to lead-based paint. Lead-based paints were widely used and applied to homes constructed prior to 1978.

The greatest risk factor is deteriorated lead based paint. The U.S. Environmental Protection Agency (EPA) in 1995 estimated approximately 64 million housing units (half the nation's entire housing stock) are affected by lead-based paint. Twenty million of these homes have paint in hazardous condition which could affect 3.8 million children under age six years.

The focus of lead poisoning prevention should be lead-based paint. Since treatment of houses following the appearance of a child who has been lead poisoned fails to realize the benefits of primary prevention, strategies should be developed to educate occupants and reduce exposure risks before the children are affected.

Lead-contaminated soil is also a source of lead exposure. Lead-based paint, gasoline, industrial sources, mining sites and smelters have contributed significantly to the contamination of soil. In 1985, the EPA reduced the amount of lead in gasoline which accounted for a profound reduction of lead emissions to the environment. Next to lead-based paint, soil and dust are believed to be the most important source of lead exposure to children.

In December of 1996, EPA and HUD jointly passed regulation requiring disclosure of known lead-based paint and/or lead-based paint hazards by persons selling or leading housing constructed be fore the phase-out of residential lead-based paint use in 1978. However, this only requires disclosure. Reduction of sources of environmental lead exposures is not formally addressed in Kansas.

III. Recommendations

It is recommended that KDHE in collaboration with local health departments develop and implement a statewide Childhood Lead Poisoning Prevention Program. It is recommended that funding be made available to local health departments for blood lead screening, public and professional education, case management, nutritional intervention and environmental lead assessments.

There will need to be adequate resources, staffing and operating funds to perform childhood blood level screenings in addition to performing tests on field samples collected during the environmental assessments.

IV. Legislative Implications

KDHE has the authority, granted by K.S.A. 65-101, to implement a lead exposure reduction program including lead screening, public and professional information, case management and consultation regarding medical and environmental management.

1 *Alliance to End childhood Lead Poisoning,* Special Edition March, 1997

2 *Reportable Diseases in Kansas 1995 Summary,* Kansas Department of Health and Environment, Office of Epidemiologic Services

3 Kansas Department of Health and Environment

Approved by the Board of Directors 05-20-97

We do not support SB437 which will create a state program to prevent lead poising in children for the following reasons:

- -The federal government will soon have their own regulations adressing this problem; True they are offering what I understand is a one time 2-3 milion dollar grant to get this going, but what will be the cost after this money runs out? Over a 10-15 year period it is conservative but will grow to to maybe 20-30 million per year maybe more.

- -Lead blood levels have dropped dramaticly over the entire US population. (JAMA, July 27, 1994; study is enclosed)

  :The mean blood level of persons aged 1 to 74 years dropped 78% (12.8 to 2.8 ug/dL)

  :The mean blood level of children aged 1 to 5 years dropped 77% (13.7 to 3.2 ug/dL)

  :The prevalence of blood lead levels 10ug/dL, or greater, for children aged 1 to 5 declined from 85% to 5.5% for non-hispanic white children and from 97.7% to 20.6% for non-hipanic black children. Siilar declines were found in population subgroups.

- -The study took place during a 15 year period, 1976 to 1991. The reason given for such a dramtic decline in lead blood level has been associated with the elimination of lead in gasoline and use of lead solder in food containers.

  :In 1980, 47% of food and soft drinks cans were soldered with lead solder.

  :By 1985 this figure of using lead solder for food can had dropped to 14%, in 1990 the figure was only 0.85%. In November 1991, Lead-solered food or soft drink cans were no longer manufactured in the United States.

  :The EPA has estimated that the typical daily intake of lead from food to have dropped from 30 ug/d in 1982 to 1.9 ug/d in 1991, 42% of lead in food came from the solder in the cans.

- -The research on the low level lead poining is highly contested. DR. Needleman who's study pushed low level of lead to the forfront methodology was flawed, and it was recommended, by the Federal Office of Research Integritiy, that he publish a correction. Why is this not well Known?

Patrick DeLapp

Senate Public Health & Welfare
Date: 2-4-98
Attachment No. 4

High lead levels in youngster lead officials to old salvage yard

MANHATTAN, Kan. (AP) — An 18-month-old boy runs the risk of stunted growth, hyperactivity and other health problems because the ground around his home is contaminated with lead, possibly from an old salvage yard.

Ricky Simms appears healthy, but a routine physical recently showed he had almost four times the normal amount of lead in his body.

"We couldn't figure out where he had gotten it," said Ricky's father, Chad Simms. "Nothing seemed wrong with him, but we were scared."

Follow-up tests at Kansas State University confirmed the January findings — that Ricky's lead level was twice what's considered hazardous.

A Kansas Department of Health and Environment official and a Riley County environmental health specialist checked the area around the family's mobile and found what may be the remnants of an old salvage yard.

What appeared to be an 8- to 10-foot radius of chips and caps from old car battery casings were found on the surface of the soil near the Simmses' mobile home. In some areas, the chips and caps were buried as deep as 2 feet.

Subsequent tests of the soil showed it had 8,500 parts of lead per million. David Williams, on-scene coordinator for the Environmental Protection Agency, said 50 to 100 lead parts per million is considered normal.

Williams took 15 additional soil samples from around the property. They were found to contain 1,000 to 4,000 lead parts per million from a 90-by-120-foot area surrounding the site where the battery parts were found. High lead levels also were discovered under Ricky's swing set.

The child could have ingested the lead by getting his hands dirty and sticking them in his mouth. Or he could have inhaled lead in the form of dust, said John Fisher, senior toxicologist with the Agency for Toxic Substances and Disease Registry.

Williams said the lead and battery parts could be what's left of a salvage yard believed to have occupied the site sometime in the 1930s or 1940s.

High lead levels in young children can cause hyperactivity, learning disabilities, growth retardation and hearing loss.

Now, health officials are trying to decide whether to move the Simmses and their next-door neighbors from the affected property and clean up the area.

At an informal meeting recently, Williams told area residents a decision hadn't been made on whether to move the Simmses or occupants of a neighboring trailer on the affected property. If the residents are relocated, Williams said they would be placed in apartments until a cleanup is completed.

"We'll have to take several more tests before we know if a cleanup is warranted,' he said, adding that it could take several weeks to finish the tests and obtain results.

Until then, Simms said he and his wife are keeping a close eye on Ricky.

"We take him out to the lake or to the park, but we don't even let him out in the yard," Simms said. "He is my pride and joy, and I'm not gonna let anything happen to him."

6/30/93 Topeka [illegible]

4-2

Haunted Housing

Last year American families sold a record 4.1 million homes, and the number looks to move even higher in 1997. We hope these folks know where to get their all-important lead-paint forms and radon kits. Lead paint, found in most pre-1978 houses, is the latest obsession of the regulatory industry. Forget to hand over the right set of lead forms in the right order to your buyer, and you could find yourself spending the summer with your lawyer.

Decades of heavy regulation have left many homeowners with a list of things to worry about. How long that list has grown is the subject of a recent study by Cassandra Chrones Moore, adjunct scholar with the Washington-based Competitive Enterprise and Cato institutes. Dr. Moore's "Haunted Housing: How Toxic Scare Studies Are Spooking the Public out of House and Home" shows how government attempts to protect families from government-defined health hazards often hurt them instead. Start with radon, which makes most people light up with images of Los Alamos. In reality, few in the science community think indoor exposure to this natural gas poses any kind of significant risk. But that didn't keep the federal government from inventing the Indoor Radon Abatement Act and State Indoor Radon Grants Programs, dedicated to radon education and radon hazard reduction.

Elementary kids thrilled to "Jeff Meets the Intruders," a National Theater for the Environment play about the evils of dust, passive smoke and radon that toured Pennsylvania thanks to the Environmental Protection Agency.

Some of their parents meanwhile got to experience the EPA more directly. Lucky towns like Montclair, N.J., chosen as EPA clean-up models, took a hit in property values. Home buyers today may pay what amounts to a "radon tax" in extra construction precautions taken almost purely to insure against possible litigation or property value loss.

Is this not beginning to remind some people of the last great danger to global health and the human species? Back in the 1960s and 1970s, scientists and gov-

Continued on page 2

Continued from page 1

ernment officials predicted up to 50,000 asbestos-related deaths a year. The number seems to be more like 600, with a good share of those resulting from occupational exposure. As far as Dr. Moore can document, precious few school janitors, let alone a child, have ever come down with asbestos disease as a direct result of sitting in an asbestos-insulated basement.

Dr. Moore estimates that school children have done without $200 billion in funds for textbooks and teaching over the years so that school administrators could rip out asbestos.

So now it's lead's turn. Lead levels that used to be the American norm are now blamed for hyperactivity and reduced intelligence in children. Certainly, the Lead Poisoning Prevention Act passed during the Bush Administration has driven down home prices and cut the supply of low-income housing. Following rules on lead removal mandated by federal and state law can cost families between hundreds and tens of thousands. This "lead tax" also hits landlords. In Maryland, for example, landlords must have rental units cleared for lead hazard before they hand keys to tenants. Realtors can also be fined up to $10,000 a day if they fail to warn of lead obligations.

The lead police of course have their own beneficiaries: the sinecured employees of federal and municipal health departments, consultants, hazard remediation firms, tort lawyers. From lead-lined Chicago, the firm of Much Shelist reports it is investigating some 900 potential lead cases. Then there are the lobbies, working hard to keep the anxiety levels high. When the Centers for Disease Control recently released data showing a national decline in lead poisoning, the executive director of the nonprofit Alliance to End Childhood Lead Poisoning sent out a worried fax: "Ironically, this impressive progress may under-mine our resolve to finish the job of eliminating lead poisoning...I hope you will editorialize in support of a significant national initiative..."

Judge Louis B. York of New York's Supreme Court must be an answer to their dreams: He recently imposed fines of $5,000 a week on the city and threatened to imprison the Housing Commissioner for failing to speed lead removal from apartments housing children under seven years of age.

Any modern environment has the potential to pose dangers, some more serious than others. These home-based threats, however, should serve as case histories of liberalism's apparent incapacity to modulate the too-large financial and bureaucratic claims that its good intentions impose on people. Lead, radon, asbestos, it all sounds so reasonable on Day One.

Years later, it's mostly an exercise in expensive unreason.

Reprinted Courtesy of the Wall Street Journal.

Lead Poisoning: Is There a Thief Somewhere?

Skip Schloming and Alfred Singer

Rampant lead-poisoning of children in their homes – it is a specter that has instigated a quagmire of misinformation and misregulation across the nation.

In some states, like Massachusetts, the current regulatory approach is like burning a forest to catch a thief – costly and hazardous. Many times, the thief isn't in the forest. Worse yet, it's very likely there never was a thief. Burn the forest anyway, say the regulators. Other states, like Maryland, are moving toward caution: they burn the forest edges only.

That's right. Maybe there's no thief at all.

The first fact to grasp is that there is no epidemic of childhood lead poisoning at present. The days of 'real' lead poisoning – convulsions, coma, brain damage and death – are over.

When lead was used in gasoline, it got spewed into the atmosphere from car exhausts in a highly toxic, micro-pulverized form. At the peak of leaded gas consumption in the 1960s, there were over 150 lead-poisoning clinics across the country. Banning lead from gasoline was, arguably, government regulation at its best: simple, effective, no other alternative. After the ban, blood lead levels plummeted nationally to levels considered safe.

"Safe," that is, until the official definition of "poisoning" was changed. Despite a huge public health victory over lead poisoning, government officials acted to keep a huge public health apparatus in force: doctors, nurses, bureaucrats, the 'deleading' industry, oh, yes, and lawyers. The result has been government regulation at its worst.

By simple fiat of the Center for Disease Control, the official definition of 'poisoning' was changed in 1985 from 60 micrograms of lead per deciliter of blood (or higher) to only 25 micrograms (or higher). The ban on gasoline lead was so effective, however, that even at this new level, 'poisoning' occurs in less than one-tenth of one percent of all children under six. Minuscule. Moreover, there are no clear, visible symptoms of poisoning as before. Vanished.

Boarded-up row houses in downtown Baltimore are testimony of the lead paint controversy.

What, no thief in the forest?

New symptoms were found. Officials tried to link low levels of lead to buzzword symptoms like "hyperactivity" and "attention deficit disorder."

One official 'expert' claimed that low levels of lead stunted children's intelligence. This expert has been "lauded for his crusade by policy makers, journalists, and child advocates," says a recent Atlantic Monthly article, but "has come under heavy fire from scientists." Colleagues checked his research, found his methods faulty, his results exaggerated (the alleged effect was at most four to six IQ points) and publicly criticized him for "deliberate misrepresentation" so that his results "would appear to be a more adequate basis for public policy."

Other scholars reviewed 26 studies and found that factors like parental education or reading bedtime stories or socio-economic status affect IQ many times more powerfully than low-level lead. In the cautious words of the Atlantic Monthly, there is "no scientific consensus on the issue."

So burn the forest anyway?

Nevertheless, government officials have pursued a hard line. In 1991, a new threshold was defined, a so-called "level of concern" at 10 micrograms of lead in the blood. Suddenly one in every 10 children has "a lead problem." An epidemic. Uninformed parents are naturally terrified.

With almost no scientific basis but relying heavily on the emotionally-charged theatrics of "brain-damaged children," government officials have persuaded legislatures to attack the "epidemic" with strict, mandatory protocols. They include universal screening (even though the 'poisoning' occurs in pockets only). And they include mandatory lead paint removal or "hazard control" in all housing units with children (even though in over 99% of the homes there won't be any 'poisoning' at all).

It's very costly. Universal screening of 5,000 children in one poor neighborhood cost $115,000 and uncovered only six cases of low-level 'poisoning,' two of which were caused by Mexican folk remedies. Aggressive deleading, such as Massachusetts requires, would cost property owners $30 billion nationwide, or at least $1 million for each 'poisoned' child. Almost all this expense is completely wasted, since 299 out of 300 deleaded units wouldn't ever have had 'poisoned' children in them anyway. Even greatly scaled-back "lead hazard control" procedures, as in Maryland's new law, still have this same waste factor.

So let's say the cost is worth it. Aren't we stopping the poisoning of a child, after all? No, we are not. All the laws and procedures are targeted at one point: lead paint in the dwelling unit, because that's the only source for which an identifiable group – property owners – can be bludgeoned into action by threat of huge financial damages. Other potentially more dangerous sources of lead are ignored, especially lead in the soil.

Maybe the thief isn't in the forest.

Low-level 'poisoning' occurs mostly in pockets, among minority households in inner cities where traffic patterns are heavy. When gasoline had lead in it, these sites got four to five tons of lead dust dumped annually on each intersection from car exhausts. As one toxicologist said: "That's roughly equal to having a lead smelter at every major intersection."

The result is a very, very large reservoir of 'bio-available lead' in the soil around inner-city housing in poorer neighborhoods. Kids play in it; they track it into the house; it blows in through open windows. Studies show: lead levels go up in the summer when kids contact dirt, in homes with pets tracking in dirt – even in homes with no lead paint.

Burn the forest anyway.

Nevertheless, the laws continue to focus on lead paint, not soil. Only a bureaucrat could think up the contorted, impractical approach used in Massachusetts. Under its law, the family is moved out; double plastic is taped all over floors and furniture; workers come in suited like spacemen; lead-painted windows are replaced; lead-painted moldings and doors are replaced or dry-scraped down to bare wood,

over 4-4

creating buckets of highly toxic lead dust. All debris is carefully wrapped and sealed; all surfaces are vacuumed and washed twice; wipe tests for leftover dust are performed – and the family finally moves back in.

This costly, aggressive 'cure' is riddled with defects often worse than the 'cure.'

Defect 1: After deleading, the 'poisoned' child is still 'poisoned' from the soil or other non-paint source.

Defect 2: After deleading, the 'poisoned' child's lead level goes up. The highly risky, dust-generating procedure has contaminated the home. One study found that 37% of children in the study had blood levels so high from deleading that they needed chelation – painful chemical flushing of lead from the blood. If regulators succeed in their comprehensive agenda, more poisoning will result from deleading than existed in the first place.

Defect 3: Before deleading, the 'poisoned' child's lead level drops. The elevated lead level came from a onetime event – homeowner remodeling, especially sanding of lead-painted surfaces – which puts toxic dust into the air. Simple cleaning solved the problem, not deleading, which the law insists must still be done.

Defect 4: Before deleading, doctors and nurses urge targeted, practical solutions (like washing hands, covering bare dirt, washing window wells, cleaning up flaking paint) and the child's lead level drops. Simple cleanliness solved the problem, not deleading, which must still be done.

Defect 5: Faced with costly deleading for tenants, the owner ignores other hazardous conditions far more serious than lead paint or abandons the building entirely. Mandated procedures do nothing except exacerbate housing conditions precisely in those neighborhoods most likely to have low-level 'poisoning.'

Defect 6: Faced with costly deleading and strict legal liability, owners quietly refuse to rent to families with children. A Massachusetts court recently awarded damages to a family whose child had a blood lead level of zero simply because lead paint was present in the home.

Recognizing these gargantuan defects, Maryland has replaced its strict law with a less strict one. The family usually needn't move out; deteriorating paint is made intact; friction surfaces on doors and windows are improved; dust-collecting window wells and sills (and sometimes floors) are covered or made smooth and cleanable; sometimes the home is specially cleaned. To induce compliance with the more moderate law, owners get a liability cap; they can't be sued for more than $17,000. (Otherwise, the sky is the limit on liability suits.)

But even Maryland property owners must treat hundreds of poison-free apartments to get the one apartment where a child may be 'poisoned.' And they must treat that one apartment and pay $17,000 even though the alleged 'poisoning' doesn't come from paint.

It's time to look for another way. The Japanese live safely in a "lead-toxic" environment just by following strict cleanliness habits: shoes at the front door, wash hands before eating, etc. In Japan, McDonald's restaurants have sinks at the front door! ■

Skip Schloming is a sociologist and editor of a newsletter for small property owners in Cambridge, MA. (Tel: 617/354-2358. E-mail: skore@cybercom.net.)
Alfred L. Singer is vice president of the Property Owners Association of Greater Baltimore, Inc. (Tel: 410/727-1324; FAX 410/528-9147.)

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The Decline in Blood Lead Levels in the United States

The National Health and Nutrition Examination Surveys (NHANES)

James L. Pirkle, MD, PhD; Debra J. Brody, MPH; Elaine W. Gunter; Rachel A. Kramer, ScD; Daniel C. Paschal, PhD; Katherine M. Flegal, PhD, MPH; Thomas D. Matte, MD, MPH

Objective.—To describe trends in blood lead levels for the US population and selected population subgroups during the time period between 1976 and 1991.

Design.—Two nationally representative cross-sectional surveys and one cross-sectional survey representing Mexican Americans in the southwestern United States.

Setting/Participants.—Participants in two national surveys that included blood lead measurements: the second National Health and Nutrition Examination Survey, 1976 to 1980 (n=9832), and phase 1 of the third National Health and Nutrition Examination Survey, 1988 to 1991 (n=12 119). Also, Mexican Americans participating in the Hispanic Health and Nutrition Examination Survey, 1982 to 1984 (n=5682).

Results.—The mean blood lead level of persons aged 1 to 74 years dropped 78%, from 0.62 to 0.14 µmol/L (12.8 to 2.8 µg/dL). Mean blood lead levels of children aged 1 to 5 years declined 77% (0.66 to 0.15 µmol/L [13.7 to 3.2 µg/dL]) for non-Hispanic white children and 72% (0.97 to 0.27 µmol/L [20.2 to 5.6 µg/dL]) for non-Hispanic black children. The prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater for children aged 1 to 5 years declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children. Similar declines were found in population subgroups defined by age, sex, race/ethnicity, income level, and urban status. Mexican Americans also showed similar declines in blood lead levels of a slightly smaller magnitude over a shorter time.

Conclusions.—The results demonstrate a substantial decline in blood lead levels of the entire US population and within selected subgroups of the population. The major cause of the observed decline in blood lead levels is most likely the removal of 99.8% of lead from gasoline and the removal of lead from soldered cans. Although these data indicate major progress in reducing lead exposure, they also show that the same sociodemographic factors continue to be associated with higher blood lead levels, including younger age, male sex, non-Hispanic black race/ethnicity, and low income level. Future efforts to remove other lead sources (eg, paint, dust, and soil) are needed but will be more difficult than removing lead from gasoline and soldered cans.

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From the Division of Environmental Health Laboratory Sciences (Drs Pirkle and Paschal and Ms Gunter) and Lead Poisoning Prevention Branch (Dr Matte), National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Ga; and Division of Health Examination Statistics, National Center for Health Statistics, Centers for Disease Control and Prevention, Hyattsville, Md (Drs Kramer and Flegal and Ms Brody).

Reprint requests to Division of Environmental Health Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, MS-F20, Atlanta, GA 30333 (Dr Pirkle).

LEAD has been dispersed in the environment in substantial quantities over a long period of time. Compelling evidence from the scientific community on a wide range of adverse health outcomes has placed lead in the forefront of environmental health concerns. In the 1970s, federal regulatory and legislative efforts were undertaken to reduce lead hazards, including actions to limit the use of lead in paint and gasoline.[1] The second National Health and Nutrition Examination Survey (NHANES II, 1976 to 1980) established baseline lead measurements for the US population and demonstrated the pervasiveness of lead

See also pp 277 and 315.

exposure across race, urban and rural residence, and income levels.[2] Data from NHANES II showed a decline in blood lead levels from the beginning to the end of the survey period that was closely correlated to declines in the use of leaded gasoline during these years.[3]

Since 1980, intensive federal, state, and local actions directed at primary prevention have been taken to further reduce lead exposure from gasoline, paint, solder, and other sources. Secondary prevention activities, such as screening for early detection and lead education programs, have also been implemented. New data from phase 1 of the third National Health and Nutrition Examination Survey (NHANES III phase 1, 1988 to 1991) permit examination of changes in blood lead levels since 1980 in the US population and evaluation of the impact of these regulatory

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actions. The Hispanic Health and Nutrition Examination Survey (HHANES, 1982 to 1984) provides data on Mexican Americans at an intermediate time point. These analyses of trends in blood lead levels serve both to evaluate the effectiveness of prevention programs and to develop new strategies to further reduce lead exposure in the United States.

METHODS

Design and Data Collection

The National Health and Nutrition Examination Surveys (NHANES) are designed to measure and monitor the health and nutritional status of the US population. The general design of the NHANES is a stratified multistage probability cluster sample of households whose target population is civilian noninstitutionalized persons residing in the United States. Blood lead levels were determined in NHANES II (1976 to 1980), HHANES (1982 to 1984), and NHANES III (1988 to 1994). The estimates from NHANES II and NHANES III are based on a national sample, whereas HHANES sampled three Hispanic subgroups.[4-6]

National trends of blood lead levels presented in this article were based primarily on comparisons of data from NHANES II and NHANES III phase 1 (1988 to 1991). Trends for Mexican Americans were based on a comparison between the estimates from HHANES and NHANES III phase 1. The HHANES also provides an intermediate point in time between NHANES II and NHANES III. The HHANES sample of Mexican Americans included only those residing in the southwestern United States whereas the NHANES III phase 1 sample represented Mexican Americans residing in the entire United States.

Venous blood lead measurements were obtained for persons aged 6 months to 74 years in NHANES II; persons aged 4 to 74 years in HHANES; and persons aged 1 year and older in NHANES III phase 1. Analysis was limited to persons aged 1 to 74 years for national trends and aged 4 to 74 years for trends in the Mexican-American population. The final samples used for analyses included 9832 and 12 119 for the national trends from NHANES II and NHANES III phase 1, respectively, and 5682 and 4067 Mexican Americans from HHANES and NHANES III phase 1, respectively. Data from all of the surveys were collected using a household interview followed by a detailed medical examination in a mobile examination center.

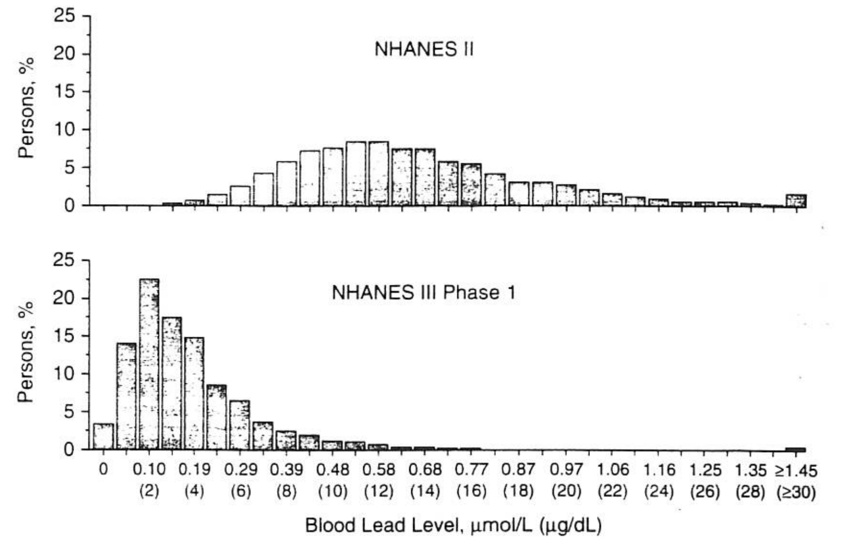

Fig 1.—Blood lead levels for persons aged 1 to 74 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).

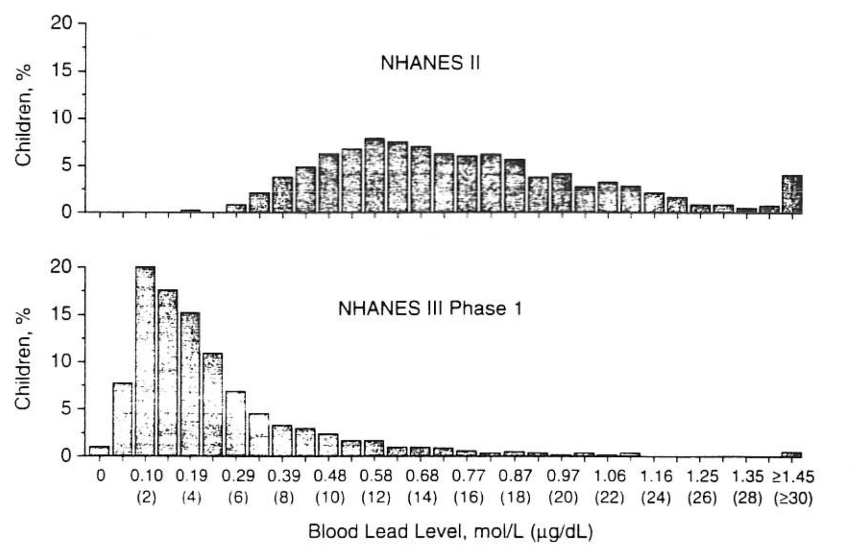

Fig 2.—Blood lead levels for children aged 1 to 5 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).

The response rates for blood lead collection in the three surveys ranged from 61% to 69%. Previous nonresponse bias analyses conducted for NHANES II, HHANES, and NHANES III phase 1 indicated that there was no apparent bias due to nonresponse.[7-9]

Laboratory Methods

All venous blood specimens were collected in the mobile examination centers, frozen, and shipped on dry ice to the NHANES laboratory, Division of Environmental Health Laboratory Sciences, National Centers for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Ga, for analysis. The methods for determining lead in blood, including descriptions of quality control and assurance procedures, have been described for each survey.[9-11] Comparability has been established for the method used in NHANES II and HHANES (modified Delves cup) and that used in NHANES III phase 1 (graphite furnace atomic absorption spectrophotometry), as described by Miller et al.[12] In each of the three sur-

Table 1.—Distribution of Blood Lead Levels for Persons Aged 1 to 74 Years by Age Category, Sex, Race/Ethnicity, Urban Status, and Income Lev[illegible] States, 1976 to 1980 (Second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (Phase 1 of the Third National Health a[illegible]trition Examination Survey [NHANES III Phase 1])

	No.	Geometric Mean, μmol/L (μg/dL)*	95% Confidence Interval, μmol/L (μg/dL)	Percentiles, μmol/L (μg/dL)						
				5th	10th	25th	50th	75th	90th	95th
All persons										
1976-1980	9832	0.62 (12.8)	0.60-0.65 (12.4-13.3)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.01 (21.0)	1.21 (25.0)
1988-1991	12 119	0.14 (2.8)	0.13-0.15 (2.7-3.0)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.23 (4.8)	0.35 (7.3)	0.45 (9.4)
Ages 1-5 y										
1976-1980	2271	0.71 (15.0)	0.67-0.75 (14.2-15.8)	0.39 (8.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.16 (24.0)	1.35 (28.0)
1988-1991	2234	0.17 (3.6)	0.16-0.19 (3.3-4.0)	0.05 (1.1)	0.07 (1.5)	0.11 (2.2)	0.18 (3.7)	0.28 (5.9)	0.46 (9.6)	0.59 (12.2)
Ages 6-19 y										
1976-1980	2024	0.56 (11.7)	0.54-0.60 (11.2-12.4)	0.29 (6.0)	0.34 (7.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.06 (22.0)
1988-1991	2963	0.09 (1.9)	0.08-0.11 (1.7-2.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.06 (1.3)	0.10 (2.1)	0.17 (3.5)	0.26 (5.4)	0.36 (7.4)
Ages 20-74 y										
1976-1980	5537	0.63 (13.1)	0.61-0.66 (12.7-13.7)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.06 (22.0)	1.25 (26.0)
1988-1991	6922	0.14 (3.0)	0.14-0.15 (2.8-3.2)	<0.05 (<1.0)	0.06 (1.2)	0.10 (2.0)	0.15 (3.2)	0.24 (5.0)	0.36 (7.4)	0.46 (9.5)
Males										
1976-1980	4895	0.72 (15.0)	0.70-0.75 (14.5-15.5)	0.39 (8.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.16 (24.0)	1.30 (27.0)
1988-1991	6051	0.18 (3.7)	0.17-0.19 (3.5-3.9)	0.06 (1.2)	0.08 (1.6)	0.12 (2.4)	0.18 (3.8)	0.28 (5.8)	0.42 (8.7)	0.52 (10.9)
Females										
1976-1980	4937	0.54 (11.1)	0.51-0.55 (10.6-11.5)	0.29 (6.0)	0.34 (7.0)	0.43 (9.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	0.97 (20.0)
1988-1991	6068	0.10 (2.1)	0.10-0.11 (2.0-2.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.07 (1.4)	0.11 (2.3)	0.18 (3.8)	0.28 (5.7)	0.36 (7.4)
Non-Hispanic whites										
1976-1980	6816	0.61 (12.6)	0.58-0.63 (12.1-13.1)	0.29 (6.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16 (24.0)
1988-1991	4337	0.13 (2.7)	0.12-0.14 (2.2-2.8)	<0.05 (<1.0)	0.05 (1.0)	0.08 (1.7)	0.14 (2.9)	0.22 (4.5)	0.33 (6.8)	0.43 (8.9)
Non-Hispanic blacks										
1976-1980	1259	0.70 (14.5)	0.66-0.74 (13.7-15.5)	0.39 (8.0)	0.43 (9.0)	0.53 (11.0)	0.72 (15.0)	0.92 (19.0)	1.11 (23.0)	1.30 (27.0)
1988-1991	3274	0.17 (3.5)	0.16-0.19 (3.3-3.9)	<0.05 (<1.0)	0.06 (1.3)	0.11 (2.2)	0.18 (3.7)	0.28 (5.9)	0.45 (9.3)	0.58 (12.1)
Non–central city										
1976-1980	7112	0.60 (12.5)	0.58-0.64 (12.0-13.1)	0.29 (6.0)	0.34 (7.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16 (24.0)
1988-1991	7495	0.13 (2.7)	0.12-0.14 (2.5-2.8)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.22 (4.6)	0.33 (6.9)	0.43 (8.9)
Central city, <1 million										
1976-1980	1612	0.66 (13.6)	0.61-0.70 (12.7-14.5)	0.34 (7.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	1.08 (22.0)	1.25 (26.0)
1988-1991	2909	0.14 (2.9)	0.12-0.16 (2.5-3.4)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.25 (5.2)	0.40 (8.3)	0.50 (10.4)
Central city, ≥1 million										
1976-1980	1108	0.67 (13.9)	0.61-0.73 (12.7-15.1)	0.34 (7.0)	0.43 (9.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	1.08 (22.0)	1.21 (25.0)
1988-1991	1379	0.19 (3.9)	0.17-0.21 (3.6-4.3)	0.06 (1.3)	0.09 (1.8)	0.12 (2.5)	0.19 (4.0)	0.29 (6.1)	0.48 (9.9)	0.64 (13.2)
Income level, low†										
1976-1980	2548	0.63 (13.1)	0.60-0.67 (12.4-13.8)	0.29 (6.0)	0.34 (7.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.11 (23.0)	1.25 (26.0)
1988-1991	4106	0.16 (3.4)	0.15-0.18 (3.1-3.8)	<0.05 (<1.0)	0.06 (1.3)	0.10 (2.1)	0.17 (3.6)	0.28 (5.8)	0.45 (9.4)	0.57 (11.8)
Income level, mid†										
1976-1980	4176	0.61 (12.6)	0.58-0.63 (12.1-13.1)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16 (24.0)
1988-1991	4050	0.13 (2.7)	0.13-0.14 (2.6-2.9)	<0.05 (<1.0)	0.05 (1.0)	0.08 (1.7)	0.14 (2.9)	0.23 (4.7)	0.34 (7.1)	0.44 (9.1)
Income level, high†										
1976-1980	2784	0.63 (13.0)	0.60-0.65 (12.5-13.5)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.01 (21.0)	1.21 (25.0)
1988-1991	2781	0.12 (2.5)	0.12-0.13 (2.4-2.7)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.7)	0.14 (2.8)	0.21 (4.3)	0.30 (6.3)	0.39 (8.0)

*For each grouping, the geometric means from NHANES II and NHANES III phase 1 are statistically different ($P<.01$).
†Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

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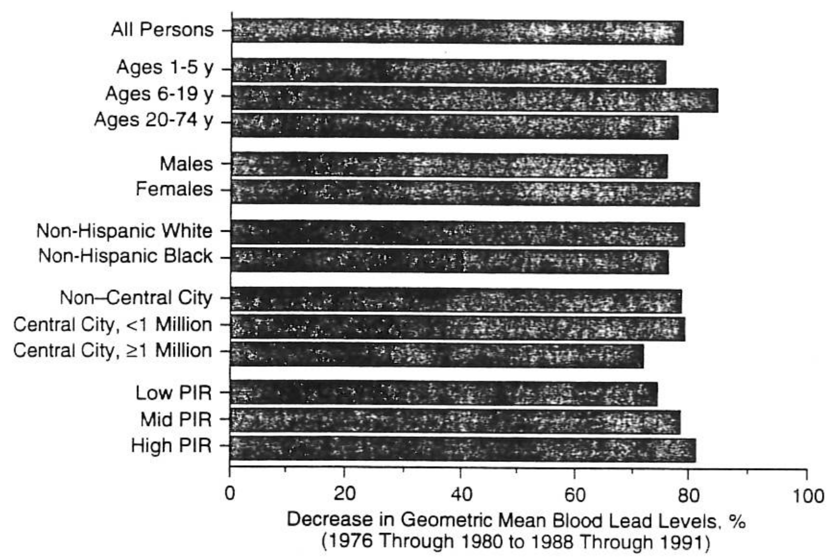

Fig 3.—Percentage decrease in geometric mean blood lead levels for persons aged 1 to 74 years by age category, sex, race/ethnicity, urban status, and income level: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey) to 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey). Income level defined by poverty-income ratio (PIR) as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

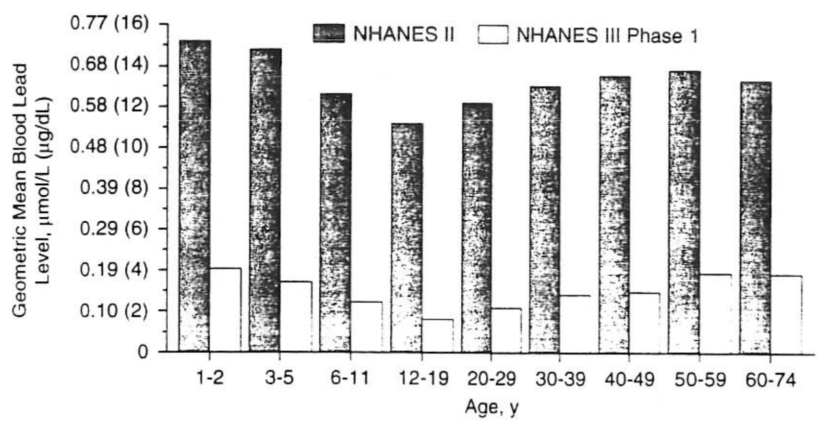

Fig 4.—Geometric mean blood lead levels for persons aged 1 to 74 years by age: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey [NHANES III phase 1]).

veys, the blood lead measurements were calibrated using standards prepared from lead nitrate Standard Reference Material 928 obtained from the National Institute of Standards and Technology, Gaithersburg, Md. The consistent use of Standard Reference Material 928 for calibration assured a common accuracy base across surveys.

Demographic and Socioeconomic Covariates

The trends analysis included stratification by five sociodemographic variables: age, sex, race/ethnicity, urban status, and income level. Age was defined in years and categorized as 1 to 5 years (4 to 5 years for Mexican Americans), 6 to 19 years, and 20 to 74 years for analysis. Race/ethnicity was categorized as non-Hispanic black, non-Hispanic white, and Mexican American. Because of small sample sizes, persons not defined by these three largest US race/ethnicity groups were included only in the overall estimates.

Definitions for income level and urban status were based on those previously determined by the US Bureau of the Census. Income level was defined by the poverty-income ratio (PIR): the total family income divided by a poverty threshold. The PIR was divided into three categories: low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00) Urban status was categorized as non–central city, central city with population less than 1 million, and central city with population 1 million or greater.

Statistical Analysis

Survey-specific sample weights were used in all statistical analyses. Geometric means and percentiles of blood lead were calculated using SAS.[13] Log_{10} transformed blood lead levels were used to normalize the distribution of blood lead levels. Geometric means were calculated by taking the antilog of the mean log_{10} blood lead levels. SUDAAN,[14] a statistical software package that incorporates the sample weights and adjusts for the complex sample design of the survey, was used to estimate the SEs.

RESULTS

The results of the trend analysis in blood lead levels are presented in two parts: first, the change in blood lead levels from NHANES II (1976 to 1980) to NHANES III phase 1 (1988 to 1991), and second, the change in blood lead levels for Mexican Americans from HHANES (1982 to 1984) to NHANES III phase 1 (1988 to 1991).

From NHANES II (1976 To 1980) to NHANES III Phase 1 (1988 To 1991)

The different distributions of blood lead levels for those aged 1 to 74 years from NHANES II and NHANES III phase 1 are presented in Fig 1. A decline of approximately 0.48 μmol/L (10 μg/dL) occurred in the geometric mean blood lead level as well as a clear change in the shape of the distribution. When the sample was limited to children aged 1 to 5 years, similar results were observed (Fig 2).

The geometric means, 95% confidence intervals, and percentiles of the blood lead distribution for the total population and stratified by the five sociodemographic factors are presented by survey in Table 1. For the total population, the geometric mean decreased by 0.48 μmol/L (10 μg/dL). Stratification of the data showed that the size of the decrease was fairly constant across sex, race/ethnicity, age groups, urban status, and income levels.

The decline represents an overall decrease in blood lead levels of 78% for persons aged 1 to 74 years and a decrease of 70% or more for selected subgroups (Fig 3). Children and youths aged 6 to 19 years showed the greatest decline in blood lead levels. However, a decline of 0.48 μmol/L (10 μg/

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Table 2.—Percentage of Persons Aged 1 to 74 Years at or Above Selected Blood Lead Level Cutoffs by Age, Sex, Race/Ethnicity, Urban Status, and Income Level: United States, 1976 to 1980 (Second National Health and Nutrition Examination Survey) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey)

		Blood Lead Levels of Population Group, %					
	No.	≥1.45 μmol/L (≥30 μg/dL)	≥1.21 μmol/L (≥25 μg/dL)	≥0.97 μmol/L (≥20 μg/dL)	≥0.72 μmol/L (≥15 μg/dL)	≥0.48 μmol/L (≥10 μg/dL)	≥0.24 μmol/L (≥5 μg/dL)
All persons							
1976-1980	9832	1.9	5.2	14.9	37.7	77.8	99.2
1988-1991	12 119	0.2	0.4	0.6	1.1	4.3	23.3.
Ages 1-5 y							
1976-1980	2271	4.1	9.3	24.7	52.6	88.2	99.8
1988-1991	2234	0.4	0.5	1.1	2.7	8.9	33.2
Ages 6-19 y							
1976-1980	2024	0.6	2.4	8.2	27.7	71.7	99.1
1988-1991	2963	0.0	0.2	0.4	0.8	2.6	12.2
Ages 20-74 y							
1976-1980	5537	2.3	5.9	16.7	40.3	79.4	99.2
1988-1991	6922	0.3	0.4	0.7	1.1	4.4	25.5
Males							
1976-1980	4895	3.3	9.0	24.1	53.1	89.6	99.8
1988-1991	6051	0.4	0.7	1.1	1.9	6.8	33.5
Females							
1976-1980	4937	0.6	1.6	6.2	23.0	66.7	98.7
1988-1991	6068	0.1	0.1	0.2	0.4	1.8	13.2
Non-Hispanic whites							
1976-1980	6816	1.7	4.8	14.0	36.0	76.9	99.2
1988-1991	4337	0.2	0.4	0.6	0.9	3.6	21.1
Non-Hispanic blacks							
1976-1980	1259	2.8	8.4	22.9	50.9	86.4	99.7
1988-1991	3274	0.2	0.4	1.2	2.6	8.5	33.7
Non–central city							
1976-1980	7112	1.9	4.9	13.9	35.3	75.7	99.0
1988-1991	7495	0.1	0.3	0.6	0.9	3.5	21.7
Central city, <1 million							
1976-1980	1612	1.9	6.1	17.1	43.1	82.1	99.8
1988-1991	2909	0.3	0.3	0.6	1.8	5.9	26.9
Central city, ≥1 million							
1976-1980	1108	1.8	6.0	18.4	44.4	84.8	99.9
1988-1991	1379	1.1	1.4	1.9	2.9	9.8	36.0
Income level, low*							
1976-1980	2548	2.9	6.8	18.0	39.6	78.4	99.2
1988-1991	4106	0.5	0.9	1.6	2.6	8.8	32.6
Income level, mid*							
1976-1980	4176	1.7	4.6	13.8	36.3	76.1	99.3
1988-1991	4050	0.2	0.3	0.5	0.9	3.4	22.9
Income level, high*							
1976-1980	2784	1.5	5.1	14.5	38.3	79.8	99.4
1988-1991	2781	0.1	0.3	0.4	0.6	2.7	18.4

*Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

dL) or greater between NHANES II and NHANES III phase 1 was consistent across the entire age range (Fig 4).

The percentage of the population with blood lead levels at or above selected values is presented in Table 2. These levels were chosen in part because of their prior or potential use in public health policy. For those aged 1 to 74 years, the prevalence of blood lead levels 0.48 μmol/L (10 μg/dL) or greater decreased from 77.8% in NHANES II to 4.3% in NHANES III phase 1. For children aged 1 to 5 years during the same time frame, the prevalence of blood lead levels 0.48 μmol/L (10 μg/dL) or greater decreased from 88.2% to 8.9%. The change in percentage of children at or above selected lead levels from NHANES II to NHANES III phase 1 is presented in Fig 5.

Separate analysis by race/ethnicity revealed that geometric mean blood lead levels declined by 77%, from 0.66 to 0.15 μmol/L (13.7 to 3.2 μg/dL), for non-Hispanic white children and by 72%, from 0.97 to 0.27 μmol/L (20.2 to 5.6 μg/dL), for non-Hispanic black children. The prevalence of blood lead levels 0.48 μmol/L (10 μg/dL) or greater for children in this same age group declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children.

Mean blood lead levels decreased from 0.73 to 0.20 μmol/L (15.2 to 4.1 μg/dL) for children aged 1 to 2 years and from 0.71 to 0.16 μmol/L (14.8 to 3.4 μg/dL) for children aged 3 to 5 years. During the same time period, the prevalence of blood lead levels 0.48 μmol/L (10 μg/dL) or greater also decreased from 88.3% to 11.5% for children aged 1 to 2 years and from 88.1% to 7.3% for children aged 3 to 5 years.

Mean blood lead levels decreased by 60% (1.16 to 0.47 μmol/L [24.0 to 9.7 μg/dL]) for non-Hispanic black children from low-income families living in the central cities with populations of 1 million or more. This compares with an overall decrease in blood lead levels of 75% (0.72 to 0.18 μmol/L [14.9

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to 3.6 µg/dL]) for all children aged 1 to 5 years.

From HHANES (1982 to 1984) to NHANES III Phase 1 (1988 to 1991)

The HHANES was conducted from 1982 to 1984, between the second and third NHANES. Geometric mean blood lead levels were also found to be intermediate between the estimates of the two national surveys. The blood lead levels of Mexican Americans from HHANES were lower than overall levels observed in NHANES II, but not as low as levels of Mexican Americans sampled in NHANES III phase 1.

Geometric means, 95% confidence intervals, and percentiles of the blood lead distribution of Mexican Americans between HHANES and NHANES III phase 1 are presented in Table 3. Mexican Americans showed an overall decrease in geometric mean of 65%, from 0.41 to 0.14 µmol/L (8.5 to 3.0 µg/dL). The geometric mean for children aged 4 to 5 years declined from 0.52 to 0.17 µmol/L (10.9 to 3.5 µg/dL). As demonstrated in the comparison of NHANES II to NHANES III phase 1 estimates, the size of the decrease in blood lead levels was similar in both sexes and across age groups and income levels.

The proportion of the Mexican-American population at or above selected blood lead levels is shown in Table 4. Overall, prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater among Mexican Americans decreased from 41.2% to 5.9%. The percentage of children aged 4 to 5 years with blood lead levels 0.48 µmol/L (10 µg/dL) or greater decreased from 61.5% to 4.9%. These results demonstrate that one in 20 Mexican Americans aged 4 to 5 years continue to have blood lead levels of health concern.

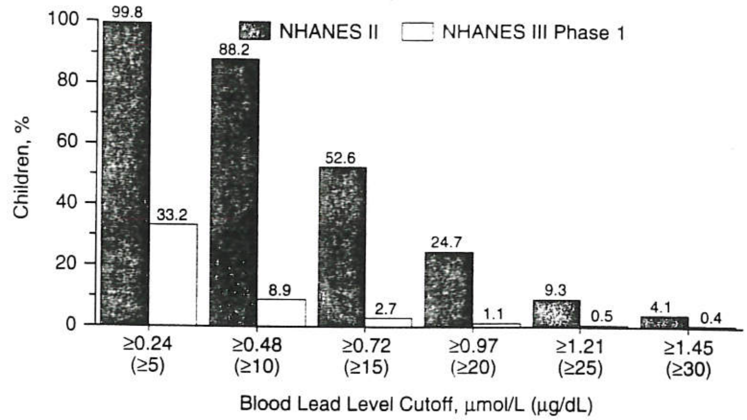

Fig 5.—Percentage of children aged 1 to 5 years at or above selected blood lead levels: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey [NHANES III phase 1]).

COMMENT

The data from two national surveys of the US population, conducted more than a decade apart, demonstrate a substantial decline in blood lead levels. As the consequence of a shift in the overall distribution of lead levels, fewer persons have blood lead levels in the upper ranges. The decrease in mean blood lead levels was observed for the total population and within all race/ethnicity, sex, urban status, and income level subgroups examined in this article. The prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater also decreased sharply from 77.8% to 4.3%.

As discussed herein, exposure to lead from major population-wide lead sources declined between 1976 and 1991. Consistent with this decline, the blood lead levels observed in HHANES (1982 to 1984) were intermediate between levels found in NHANES II (1976 to 1980) and NHANES III phase 1 (1988 to 1991). Consequently, the magnitude of decrease from HHANES to NHANES III phase 1 (65%) was less than from NHANES II to NHANES III phase 1 (78%). The percentage of Mexican Americans with blood lead levels 0.48 µmol/L (10 µg/dL) or greater declined from 41.2% to 5.9%. In NHANES III phase 1, both mean blood lead levels and the prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater of Mexican Americans were closer to those of non-Hispanic whites than to those of non-Hispanic blacks.

The decline in blood lead levels seen in these national surveys is consistent with the results of other studies of environmental lead levels,[1] which indicate that a continued reduction in exposure to lead sources began in the late 1970s and continued throughout the 1980s. Between 1976 and 1991, the three major sources of lead exposure common to the general population were lead in gasoline, soldered cans, and paint. In 1976, a total of 186.47 million kg (205 810 tons) of lead was used in gasoline in the United States.[15] In 1983, this amount had dropped to 51.59 million kg (56 940 tons), and in 1990, lead used in gasoline had been reduced to 0.47 million kg (520 tons).[15] From 1976 to 1990, the amount of lead used in gasoline decreased 99.8%. The reduction of lead in gasoline is most likely the greatest contributor to the observed decline in blood lead levels during the period of the national surveys.[1,3,16,17]

Lead from gasoline and soldered cans contribute to lead in food. Since gasoline lead enters food through multiple pathways,[1,15,16] it is difficult to make a quantitative estimate of the reduction in food lead that resulted from decreasing lead in gasoline. The amount of lead used in soldered cans decreased markedly throughout the 1980s. In 1980, 47% of food and soft drink cans were lead soldered. By 1985, this figure had dropped to 14%, and by 1990, only 0.85% of food and soft drink cans were lead soldered.[18] As of November 1991, lead-soldered food or soft drink cans were no longer manufactured in the United States.[18]

The Food and Drug Administration uses "market-basket" surveys to estimate the average daily intake of lead from food for various population groups in the United States.[19] For 2-year-old children, these surveys estimate the typical daily intake of lead to have dropped from 30 µg/d in 1982 to 1.9 µg/d in 1991.[19,20] The Environmental Protection Agency estimated in 1986 that about 42% of lead in food came from lead-soldered cans.[1] Thus, reducing the amount of lead used in soldered cans has likely been a major factor in reducing food lead levels. Although it is difficult to quantitatively determine the decrease in blood lead levels attributable to reduced amounts of lead in soldered cans, the decline in the amount of lead used in this source probably contributed substantively to the observed decline in blood lead levels.

The manufacture of lead-based paint was limited to less than 0.06% by weight in 1978 by the Consumer Product Safety Commission.[1] Individuals who have left housing with lead-based paint or who reside in lead-abated homes have reduced their lead exposure. Still, lead-based paint remains a problem, predominantly in older, deteriorating housing.[1,15,16] The NHANES do not specifically target persons who live in such

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Table 3.—Distribution of Blood Lead Levels for Mexican Americans Aged 4 to 74 Years by Age Category, Sex, and Income Level: 1982 to 1984 ([illegible] Health and Nutrition Examination Survey [HHANES]) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey [NHANE[illegible] [illegible]ase 1])

	No.	Geometric Mean, µmol/L (µg/dL)*	95% Confidence Interval, µmol/L (µg/dL)	Percentiles, µmol/L (µg/dL) 5th	10th	25th	50th	75th	90th	95th
All persons										
1982-1984	5682	0.41 (8.5)	0.40-0.42 (8.3-8.7)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.87 (18.0)
1988-1991	3611	0.14 (3.0)	0.12-0.17 (2.5-3.5)	<0.05 (<1.0)	0.05 (1.1)	0.09 (1.9)	0.16 (3.3)	0.26 (5.4)	0.40 (8.3)	0.51 (10.6)
Ages 4-5 y										
1982-1984	269	0.53 (10.9)	0.50-0.56 (10.3-11.5)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0.92 (19.0)	1.11 (23.0)
1988-1991	349	0.17 (3.5)	0.14-0.21 (2.8-4.3)	<0.05 (<1.0)	0.07 (1.4)	0.12 (2.5)	0.18 (3.8)	0.28 (5.9)	0.40 (8.3)	0.48 (9.9)
Ages 6-19 y										
1982-1984	2331	0.39 (8.0)	0.38-0.40 (7.8-8.2)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0.82 (17.0)
1988-1991	1188	0.12 (2.5)	0.10-0.15 (2.0-3.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.6)	0.14 (2.8)	0.23 (4.7)	0.36 (7.4)	0.47 (9.8)
Ages 20-74 y										
1982-1984	3082	0.42 (8.7)	0.40-0.43 (8.3-9.0)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.92 (19.0)
1988-1991	2074	0.15 (3.2)	0.13-0.18 (2.7-3.7)	<0.05 (<1.0)	0.05 (1.1)	0.10 (2.0)	0.16 (3.4)	0.28 (5.7)	0.42 (8.6)	0.54 (11.1)
Males										
1982-1984	2638	0.50 (10.4)	0.49-0.51 (10.2-10.5)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.48 (10.0)	0.68 (14.0)	0.87 (18.0)	1.01 (21.0)
1988-1991	1797	0.19 (4.0)	0.16-0.23 (3.3-4.8)	0.06 (1.2)	0.08 (1.6)	0.12 (2.5)	0.20 (4.2)	0.31 (6.5)	0.45 (9.4)	0.57 (11.8)
Females										
1982-1984	3044	0.34 (7.0)	0.32-0.35 (6.7-7.2)	0.14 (3.0)	0.19 (4.0)	0.24 (5.0)	0.34 (7.0)	0.43 (9.0)	0.58 (12.0)	0.68 (14.0)
1988-1991	1814	0.11 (2.2)	0.09-0.13 (1.8-2.7)	<0.05 (<1.0)	<0.05 (<1.0)	0.07 (1.4)	0.12 (2.4)	0.19 (3.9)	0.31 (6.4)	0.41 (8.4)
Income level, low†										
1982-1984	2460	0.42 (8.8)	0.42-0.44 (8.6-9.1)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.92 (19.0)
1988-1991	1664	0.16 (3.3)	0.13-0.19 (2.7-4.0)	<0.05 (<1.0)	0.06 (1.2)	0.10 (2.0)	0.17 (3.6)	0.28 (5.8)	0.43 (9.0)	0.54 (11.1)
Income level, mid†										
1982-1984	2032	0.40 (8.3)	0.38-0.42 (7.9-8.7)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.87 (18.0)
1988-1991	1024	0.13 (2.6)	0.11-0.15 (2.2-3.1)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.6)	0.14 (2.9)	0.23 (4.8)	0.36 (7.5)	0.44 (9.2)
Income level, high†										
1982-1984	674	0.39 (8.1)	0.37-0.39 (7.6-8.6)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.72 (15.0)	0.82 (17.0)
1988-1991	393	0.11 (2.3)	0.09-0.14 (1.8-2.9)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.6)	0.12 (2.5)	0.19 (4.0)	0.28 (5.7)	0.35 (7.3)

*For each grouping, the geometric means from HHANES and NHANES III phase 1 are statistically different ($P<.01$).
†Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

housing. The distribution of blood lead levels in the NHANES reflects exposure in the general population, whereas studies focusing on high-risk populations, such as persons living in older, deteriorating housing, may find a different blood lead distribution. Data from national housing surveys indicate that in 1980 about 24.2 million (30.3%) occupied houses in the United States were built before 1940 when lead-based paint was in common use. By 1989, this number had decreased by 3.4 million to 20.8 million (22.2%), suggesting that population exposure to lead-based paint may have decreased slightly.[21,22] On the other hand, the continuing deterioration of lead-based paint in existing houses could increase the likelihood of exposure for persons in the 20.8 million households who remained in these older houses. On a population scale, it is not clear whether the net effect is an increase or decrease in exposure to lead-based paint.

The consistent decline in blood lead levels across broad population categories of age, sex, race/ethnicity, urban status, and income level most probably reflect changes in exposure to major population-wide lead sources. In addition, selected population groups within the United States are likely to have benefited from other changes in exposure, such as reductions in lead in community water supplies and reduction of lead emissions from local industry.

The public health impact of the observed decline in blood lead levels of the US population is dramatic, especially for children. The change in the proportion of children aged 1 to 5 years with blood lead levels 0.48 µmol/L (10 µg/dL) or greater was at least 70% for non-Hispanic whites, non-Hispanic blacks and Mexican Americans. Although the decline in blood lead levels is encouraging, the number of children with lead levels 0.48 µmol/L (10 µg/dL) or greater remains substantial and disproportionately higher for non-Hispanic black children (one in five children), as discussed in the accompanying article in this issue.[9]

At least 99.8% of lead in gasoline has already been removed, and domestically produced cans are no longer lead soldered. Therefore, to achieve additional

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ble 4.—Percentage of Mexican Americans Aged 4 to 74 Years at or Above Selected Blood Lead Level Cutoffs by Age, Sex, and Income Level: 1982 to 1984 spanic Health and Nutrition Examination Survey) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey)

	No.	Blood Lead Levels of Population Group, % ≥1.45 µmol/L (≥30 µg/dL)	≥1.21 µmol/L (≥25 µg/dL)	≥0.97 µmol/L (≥20 µg/dL)	≥0.72 µmol/L (≥15 µg/dL)	≥0.48 µmol/L (≥10 µg/dL)	≥0.24 µmol/L (≥5 µg/dL)
persons							
1982-1984	5682	0.4	1.3	3.6	12.8	41.2	91.2
1988-1991	3611	0.0	0.2	0.4	1.5	5.9	28.9
es 4-5 y							
1982-1984	269	2.4	4.9	8.8	24.7	61.5	96.4
1988-1991	349	0.0	0.0	0.0	0.1	4.9	32.7
es 6-19 y							
1982-1984	2331	0.3	0.5	2.0	9.0	35.8	90.1
1988-1991	1188	0.0	0.1	0.4	0.9	4.5	23.8
es 20-74 y							
1982-1984	3082	0.3	1.5	4.2	14.1	42.9	91.5
1988-1991	2074	0.0	0.2	0.4	1.9	6.6	31.0
ales							
1982-1984	2638	0.6	2.1	6.2	21.1	58.4	96.8
1988-1991	1797	0.0	0.2	0.5	2.2	8.7	40.5
emales							
1982-1984	3044	0.1	0.4	1.1	4.5	23.8	85.6
1988-1991	1814	0.0	0.1	0.3	0.7	2.8	16.4
come level, low*							
1982-1984	2460	0.5	1.4	4.1	14.6	45.2	92.2
1988-1991	1664	0.0	0.1	0.4	1.7	7.3	33.5
come level, mid*							
1982-1984	2032	0.4	1.3	3.4	11.4	38.0	90.1
1988-1991	1024	0.0	0.0	0.3	1.1	4.2	23.6
come level, high*							
1982-1984	674	0.2	0.8	2.7	10.0	35.8	91.2
1988-1991	393	0.0	0.0	0.0	0.0	1.6	14.7

*Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR≤1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

reductions in blood lead levels in the US population, sources other than lead in gasoline and lead in solder need to be addressed further. The major remaining sources are lead in paint and lead that has already accumulated in dust and soil. Without efforts to reduce these exposures, population blood lead levels are unlikely to continue to decline.

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3644 S. W. Burlingame Road
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Telephone 913/267-3610
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REALTOR®

TO: THE SENATE PUBLIC HEALTH AND WELFARE COMMITTEE

FROM: KAREN FRANCE, DIRECTOR, GOVERNMENTAL AFFAIRS

DATE: JANUARY 29, 1997

SUBJECT: SB 437, CERTIFICATION OF LEAD-BASED PAINT CONTRACTORS

Thank you for the opportunity to testify. The Kansas Association of REALTORS® opposes SB 437. While we recognize the problems caused for children who ingest lead-based paint, we believe the creation of another level of state bureaucracy in return for federal EPA money is not a worthy tradeoff.

Currently, when our members are required make sure a home buyer interested in purchasing a home built prior to 1978 receives two items prior to becoming obligated in a sales contract. They must provide an EPA pamphlet titled "Protect Your Family From Lead in Your Home" and they must also provided a Lead Paint Disclosure, Lead Warning Statement.

The warning statement is divided into three parts. The first part must be filled out by the seller who is required to disclose whether they have knowledge of the presence of lead-based paint hazards in the home and whether there are any written records regarding the presence of lead-based paint in their home. If any written record exists, the seller must provide the records with the disclosure form.

The second part is filled out by the buyer who acknowledges having received copies of the seller's records, a copy of the "Protect Your Family from Lead in Your Home" and they have been given a 10 day, or mutually agreed upon period, to conduct a risk assessment or inspection for the present of lead-based paint or hazards or they can waive the opportunity for an inspection.

The last part is filled out by the real estate licensee, acknowledging they have informed the seller of their obligation to disclose lead-based paint hazards.

An agent who fails to comply with the law can have civil penalties brought against them for up to $10,000 for each violation. The seller, lessor or agent may be liable for three times the damages for injuries sustained by the purchaser or lessee. These damages may include costs of correcting lead-based paint hazards and medical costs related to lead-based paint poisoning. Federal penalties for violating the law can be $10,000 for each violation and imprisonment for up to one year, or both.

This disclosure requirement has been in effect for over a year now. An unscientific survey of many of the real estate companies who sell the highest volume of homes in the state revealed

Senate Public Health and Welfare
Date: 2-4-98
Attachment No. 5

that even with the pamphlet and the 3-part warning statement presented to prospective buyers, very few buyers are requesting lead-based paint assessments prior to purchase. Out of approximately 150 companies asked, only 10 buyers had requested the assessment.

When we asked these same members how they felt about the state setting up a program to license or certify the individuals who do the testing, they responded with questions like: "Why do we need them? Even when told the facts about the risks of lead-based paint, buyers still opt to not have the assessment done". "Why should we set up another state bureaucracy which stands to increase the costs of the assessments if a buyer does choose to have one performed?" and a commonly asked question was, "If the EPA wants this done, why don't they just do it themselves?"

It is our understanding at least 7 other states have declined to institute this EPA mandate. They are not concerned that their children will be damaged any more by lead-based paint hazards if the state doesn't set up this bureaucracy. We ask you to follow the lead of these states.