LEAD Action NEWS

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Stuttering and other evidence of lead poisoning in families

Contents

A guide to researching a family tree for evidence of previously unrecognized lead poisoning in family members

This guide is intended as an aid for the reader trying to determine if they or a family member (perhaps now deceased) unknowingly experienced lead poisoning from lead supplied by the Bell System. Although the guide may help a reader make sense of seemingly disparate family events from long ago (and in the process perhaps arouse suspicion that poisoning occurred), the connect-the-dots approach espoused here should not be confused with proof of poisoning.

All the reader may have at his or her disposal are family stories and letters that vaguely allude to past problems but leave the reader without any real understanding of what the problems were.

Perhaps a search for the truth can begin by recalling stories, if any, that suggest strife and conflict in a family.[1][i] Memories and stories of domestic turmoil are more likely to have survived the years then events that evoked fewer emotions. Lead poisoning can lead to adverse behaviour in both adults and children. A parent's irritability and anger in the face of a child's failure to meet expectations at home or school can look like a normal reaction to a child misbehaving, and perhaps that's the accurate assessment. But an alternative explanation is that the behaviors of the child, parent, or both were actually symptoms of lead poisoning masquerading as domestic strife.

Having concluded that strife and conflict occurred a closer look may be informative. An effort should be made to determine if the child was hyperactive, easily distracted, or a slow learner. For school-age children official reports or family letters may have survived documenting the child's academic performance. If they indicate that the child struggled with poor grades and/or disruptive behavior in the classroom it can add support to the idea that lead poisoning occurred in the family. Even in the absence of any other information about a child's performance, knowledge that a child was held back in school and forced to repeat one or more grade levels is supportive information.

Attention is turned to the family's adults. Lead can result in irritability and anger in an adult resulting in emotional outbursts that may seem out of proportion to instigating events (it can affect children in the same way). In the case of occupational exposure to lead any such symptoms can be expected to have occurred in the family wage earner along perhaps with other adults in the family. However in the absence of a history of chronic irritability and anger in the wage earner, serious doubt is thrown on occupational lead exposure as the cause even if there is evidence of it in another adult in the family.

Since the irritability is pathologic and therefore largely not under voluntary control, evidence may have survived that the irritability worked to the detriment of the individual expressing it. In the workplace this may have surfaced as: being fired from a job, demoted, failing to be promoted, frequent changing of jobs, or friction with co-workers. Old family photographs may be of help as well. Individuals will generally try to look their best for formal portraits even if they've had a bad day. In addition to dress this means showing a pleasant face. A formal photograph that shows evidence of irritability and anger in the facial features, particularly if that is a change from photographs taken earlier and/or later, adds support to the hypothesis that lead poisoning occurred. Finally, the irritability and anger may have engendered fear in others

[i] Family violence on the other hand has not been linked to lead poisoning.

(particularly in young children), fear that the anger might be directed toward them. Memories of that fear may have survived in stories and letters.

Although unprotected exposure to lead is dangerous whenever and wherever it occurs, Bell System construction and maintenance personnel, and in particular Western Electric employees working in installation, were subjected to perhaps three peaks of intense lead exposure during the last century; once in the late '30s and early '40s when the first lead-sheathed transcontinental telephone cable was being laid (the pre-war years), once during the war years (approximately 1942-'45) when the lead content of all types of solder skyrocketed due to a shortage of tin, and once again in the early '50s when workers were exposed to lead-contaminated dust and debris associated with the widespread demolition and swapping out of manual for automated telephone switching equipment across the Bell System. If the reader is able to temporally place the events of interest into any or all of these time periods that is supportive information.

Having made it this far with suspicions intact, the reader may want to examine the table on the following page for further help. Although each of the findings and symptoms listed in the table could conceivably have had any one of a number of causes, as each finding or symptom is added to a list of events occurring in one family living under one roof, the odds increase that some or all were due to lead poisoning. i (see last page for endnote)

Some physical finding and symptoms said to be associated with lead poisoning		**Can occur in;**		
Symptom/finding	**Comment (if any)**	**Adults**	**Children**	**Animals**
Hydrocephalus	Hydrocephalus occurs not only as a congenital abnormality in children but as an acquired condition in adults and children as well. Whether chronic lead poisoning can cause hydrocephalus in adults has never been studied although it has been documented in young children and in animals.	?	Yes	Yes
Headache	Little has been written about the headache of lead poisoning in detail, other than it is likely the result of cerebral edema and usually bitemporal in location. In general headaches due to cerebral edema are quite persistent, may be severe, and require more pain relief then can be provided by aspirin or Tylenol®. Because occupational lead exposure among Bell System workers was intermittent the headaches, if any, would have been intermittent as well. The headaches could conceivably have been confused with migraines. Unlike migraines, however, there would not have been sensitivity to light, or the prodrome (mainly visual effects) that is often seen in migraine sufferers just before onset of the headache.	Yes	?	?
Emotional or behavioural problems manifesting as anger, hyperirritability, or nervousness		Yes	Yes	?
Cataracts	Lead has been found to interfere with the	Yes	?	?

	movement of essential elements into and out of both animal and human lenses. Unimpeded exchange of elements such as zinc is needed in order to maintain lens clarity			
Hearing loss	Often a high-frequency loss around 4000 Hz	Yes	Yes	?
Attention deficit/hyperactivity/difficulty learning (ADHD)	Researchers have concluded that the occurrence of the condition known as ADHD is largely genetic in origin although environmental contaminants also play a role. However there are cases of ADHD that have no known genetic component (that is, there is no history of ADHD among either first degree relatives or offspring). In those cases ADHD may be the result solely of environmental contaminants such as lead. Maternal cigarette smoking is thought by many to be a major risk factor as well although the evidence so far is inconclusive. In families where lead contamination and smoking are both present it may be difficult to separate out the effect that each has on the occurrence of ADHD. However there may be a clue and that is that if there are additional indicators of lead poisoning in a family aside from the presence of ADHD these would be supportive of lead as the cause. Fergusson (see ref) found that even taking into account the effects of smoking lead poisoning remained an independent predictor of ADHD.	Yes	Yes	Yes
Lower intellectual functioning	Strong evidence that lead poisoning can reduce IQ in children, and evidence that it can reduce occupational achievement in adults who had lead poisoning as children.	Yes	Yes	Yes
Sexual impotence/decreased libido/decreased fertility/difficulty attaining an erection	A brief anecdotal comment about this can be found at www.lead.org.au/bellsystemleadpoisoning/images/Lead poisoning news-Australia.pdf.	Yes	-	Yes
Stomach pain/constipation ("lead colic")	One author (see Janin) pointed out that the term "colic" is somewhat of a misnomer and that the pain is more of a dull ache. Furthermore it is not clear if constipation from lead poisoning is irrevocably linked to the occurrence of pain or whether constipation can occur with little or no pain. For Bell System workers exposed outside the factory the question arises as to whether cyclic exposure to lead for many years may have resulted in permanent damage to the normal function of the bowel due to recurrent constipation-related straining at the stool. One medical review found that the constipation and accompanying enlargement of the colon	Yes	Yes	?

	seen with lead poisoning both resolved with resolution of the poisoning. However this conclusion assumes two things, one is that the sufferer received competent medical attention for the problem, and second that once the problem occurred the victim's unprotected exposure to lead ended. However due to the nature of their exposure (that is, the lead exposure was intermittent, random, of variable intensity, and prolonged), none of these assumption may have been true for at-risk Bell System field workers. In that case conclusions based on these assumptions may need to be revisited.			
High blood pressure	High blood pressure has been observed in individuals with active lead poisoning as well as in individuals whose poisoning resolved decades before. A cause and effect relationship between lead poisoning and high blood pressure has been shown by several researchers.	Yes	?	Yes
Juvenile delinquency	-	-	Yes	-
Speech impediment	For more then 50 years it has been known that a child's ability to speak can be impaired by severe lead poisoning. Furthermore there are striking similarities between the damage that lead can do to the muscles of the voice box and damage observed to the same muscles in some individuals who chronically stutter. This has led researchers to suspect that lead poisoning may be a causative factor in a type of stuttering known as "neurogenic" [2]. Although the association has yet to be proven, it is remarkable that as the frequency of lead poisoning has declined over the years the frequency of stuttering has declined more or less in step. (see also, document footnote # 10 at "Uncovering a sixty year-old story of lead poisoning", as well as the web page "Lead poisoning and stuttering", on this website)	?	Yes	-
Dental problems	Individuals with lead poisoning are prone to dental cavities (caries). Researchers hypothesis that this may be the result of changes in the antimicrobial properties of saliva caused by lead substituting for calcium in the salivary glands. This in turn can lead to higher levels of plaque and cavities. In addition lead can substitute for calcium in the dental enamel of the developing teeth of children resulting in a weak, brittle, enamel.	Yes	Yes	Yes

[2] Although there are various subtypes of neurogenic, or acquired, stuttering, in general this type is said to have the following vocalizing characteristics: "involuntary repetition primarily of correct sounds and syllables anyplace in the word"

(Rosenbeck, p 46); the absence of accessory features such as grimacing; periods of silence associated with a transient inability to initiate any kind of sound; a lack of openly expressed anxiety about the stuttering on the part of the stutterer.

References:

Headache:

Goetz C., Washburn K., Kompolito K "Metal Intoxication: Lead" in Clinical Neurology, Lippincott, Williams and Wilkins, 1998.

Cullen M.R., Robins J, Eskenazi B "Adult inorganic lead intoxication: presentation of 31 new cases and a review of recent advances in the literature", Medicine 62(4); pp 221-247, 1983.

Stewart W, Schwartz B, "Effects of lead on the adult brain: a 15year exploration". American Journal of Industrial Medicine 50: p 729-739, 2007.

Lead colic:

Janin Y, Couinaud C, Stone A, Wise L. "The 'lead-induced colic' syndrome in lead intoxication" Surgery Annual 17; pp 287-307, 1985

Hydrocephalus:

Hirano A, Iwata M, "Neuropathology of lead intoxication" in Handbook of Clinical Neurology 36, pp 35-64, 1979.

Goetz C., Washburn K., Kompolito K "Metal Intoxication: Lead" in Clinical Neurology , Lippincott, Williams and Wilkins, 1998.

Irritability/Anger

Lucchini R, Elisa A, Cortesi I, Placid D, et.al "Assessment of neurobehavioral performance as a function of current and cumulative occupational lead exposure", Neurotoxicology 21(5), pp 805-812, 2000.

Byers R, Lord E, "Late effects of lead poisoning on mental development", American Journal of Diseases of Children 66(5), p 471, 1943.

Sciarillo W, Alexander G, et. al. "Lead exposure and child behavior", American Journal of Public Health 82(10), p 1356, 1992.

Cullen M.R., Robins J, Eskenazi B "Adult inorganic lead intoxication: presentation of 31 new cases and a review of recent advances in the literature", Medicine 62(4); pp 221-247, 1983.

High Blood Pressure

Lead Poisoning : the Report of the Committee on Lead Poisoning; p 23, The American Public Health Association, NY,NY, 1930.

A Guide to Researching Lead Poisoning in a Family Tree 2/20/08 Schwartz J, "The relationship between blood lead and blood pressure in the NHANES II survey", Environmental Health Perspectives 78, pp 15-22, 1988.

Hu H, "A 50-year follow-up of childhood plumbism: hypertension, renal function, and hemoglobin levels among survivors", American Journal of Diseases of Children 145, pp 681- 687, 1991.

Juvenile delinquency

Needleman H, Riess J., et.al, "Bone lead levels and delinquent behavior", JAMA 275(5), p 363-369, Feb 1996.

Dietrich K, Ris M, et.al. “Early exposure to lead and juvenile delinquency” Neurotoxicology and Teratology 23(5), p 511-518, 2001

Sexual impotence/decreased libido/decreased fertility/difficulty attaining an erection

Cullen M.R., Robins J, Eskenazi B “Adult inorganic lead intoxication: presentation of 31 new cases and a review of recent advances in the literature”, Medicine 62(4); pp 221-247, 1983.

Lancranjan I, Popescu H, et.al. “Reproductive ability of workmen occupationally exposed to lead”, Archives of Environmental Health 30, pp 396-401, 1975.

Varma MM, Joshi SR, Adeyemi AO “Mutagenicity and infertility following administration of lead sub-acetate to Swiss male mice”, Experientia 30 (5), pp486-487, 1974.

Speech impediment

Mayfield S., “Language and speech behaviors of children with undue lead absorption: a review of the literature”, Journal of Speech and Hearing Research 26, pp 362-368, 1983.

Byers R, Lord E, “Late effects of lead poisoning on mental development”, American Journal of Diseases of Children 66(5), p 471-494, 1943.

Schwartz J, Otto D, “Blood lead, hearing thresholds, and neurobehavioral development in children and youth”, Archives of Environmental Health 42(2), p 153-160, 1987.

Rosenbek J “Stuttering secondary to nervous system damage” in Nature and Treatment of Stuttering: New Directions; R. Curlee and W. Perkins (Ed.): Chap 3; College Hill Press, San Diego, 1984.

Wendell Johnson and Associates, The Onset of Stuttering: research findings and implications, University of Minnesota Press, Minneapolis, 1959.

Cataracts

Schaumberg D, Mendes F, “Accumulated lead exposure and risk of age-related cataract in men”, JAMA 292(22), pp.2750-2754, 2004.

Shukla N, Moitra JK, Trivddi RC, “Determination of lead, zinc, potassium, calcium, copper, and sodium in human cataract lenses”, The Science of the Total Environment 181; 161-165, 1996.

Neal R, Cooper K, et.al. “Effects of some sulfur-containing antioxidants on lead-exposed lenses”, Free Radical Biology and Medicine 26; 239-243, 1999.

Neal R, Cooper K, et.al. “Effects of N-acetylcysteine and 2,3-dimercaptosuccinic acid on lead induced oxidative stress in rat lenses”, Toxicology 130; 167-174, 1998.

Hearing loss

Repko J, Corum C, et.al. The Effects of Inorganic Lead on Behavioral and Neurologic Function: Final Report, pp 39-48, DHEW, Contract No. 210-75-0054, U. Government Printing Office, Washington DC, 1978.

Schwartz J, Otto D, “Blood lead, hearing thresholds, and neurobehavioral development in children and youth”, Archives of Environmental Health 42(2), p 153-160, 1987.

Forst L, Freels, Persky V, “Occupational lead exposure and hearing loss”, Journal of Occupational and Environmental Medicine 39(7), pp 658-660, 1997.

Dental Cavities (Caries)

Moss M, Lanphear B, Auinger P, “Association of dental caries and blood lead levels”, JAMA 281(24), pp 2294-2298, 1999.

Gemmel A, Tavares M, et.al., “Blood lead levels and dental caries in school-age children”, Environmental Health Perspectives 110(10), pp. A625-A630, 2002.

Shapiro I, Needleman H, Tuncay O, “The lead content of human deciduous and permanent teeth” Environmental Research 5, pp. 467-470, 1972.

Lower Intellectual Functioning

Bellinger D, Stiles K, Needleman H, “Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study” Pediatrics 90, pp 855-861, 1992.

Baghurst P, McMichael A, et.al. “Environmental exposure to lead and children’s intelligence at the age of seven years: the Port Pirie Cohort Study” New England Journal of Medicine 327(18), pp 1279-84, 1992.

Pocock S, Smith M, Baghurst P, “Environmental lead and children’s intelligence: a systematic review of the epidemiological evidence”, British Medical Journal 309, pp 1189-97, 1994.

White R, Diamond R, et.al. “Residual cognitive deficits 50 years after lead poisoning in childhood”, British Journal of Industrial Medicine 50: pp 613-622, 1993.

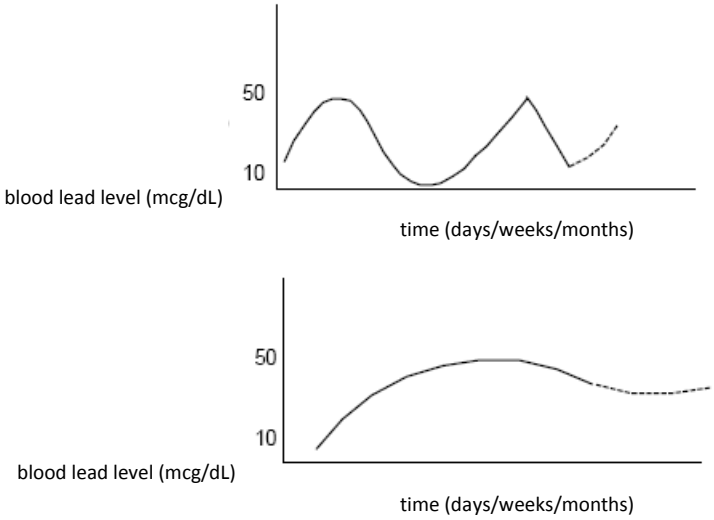
ADHD

Biederman J, “Attention-deficit/hyperactivity disorder : a selective overview” Biological Psychiatry 57: pp 1215-1220, 2005.

Goldman L, Genel M, et.al. “Diagnosis and treatment of attention-deficit/hyperactivity disorder in children and adolescents” JAMA 279 (14): pp 1100-1107, 1998.

Fergusson D, Fergusson J, et.al., “A longitudinal study of dentine lead levels, intelligence, school performance and behaviour”, Journal of Child Psychology and Psychiatry 29 (6): pp811-824, 1988.

i Because chronic lead poisoning has been poorly characterized there is not a standard definition. As a result groups said to have chronic poisoning (that is, long-term low level poisoning) cannot be assumed to have levels of disease and disability that are equivalent. For example blood lead levels drawn over time in two hypothetical individuals, both of whom are said to have chronic lead poisoning, are seen in the graphs below. The top example could represent hypothetical blood lead levels in an exposed Bell System employee working in the field (that is, working outside the factory), while the bottom example might represent an auto industry worker in a battery plant. Based on differences in the variability of their blood lead levels the two individuals could have very different medical experiences. For the Bell System worker symptoms could come and go in such random fashion that he might seldom if ever come to the attention of a physician. The battery plant worker on the other hand would stand a better chance of receiving medical care because his symptoms are more likely to become sustained. Once the battery plant worker is identified as having lead poisoning he is removed from exposure. But because the Bell System employee is never identified as having poisoning his exposure continues unabated, probably for years. Nobody knows what that might mean for the health of the affected worker because research that might have provided an answer was never done. But for some affected individuals the news couldn’t have been good.



Lead Poisoning News [ii]

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Posted April 10, 2008

Visit www. Occupationalhazards.com!

Readers of this posting are encouraged to visit http://www.occupationalhazards.com. This is the web site of the magazine 'Occupational Hazards' published by Penton Media, Inc. The publication provides much online content and is fully searchable.

Posted December, 2009

[ii] *Note: This document contains hyperlinks to other web sites. At least one of these sites (www.cwalocal7810.org) have recently placed restrictions on access to content. These restrictions were put in place shortly after a link to the site appeared in 'Lead Poisoning News'.*

NIOSH report on occupational hazards at the central telephone office of the New York Telephone Company, White Plains.

In October 1991 employees working in the central telephone exchange of the New York Telephone Company (NYNEX), White Plains, requested that the National Institute for Occupational Safety and Health (NIOSH) make a study of occupational health hazards at the exchange. NIOSH investigators found lead levels as high as 5% in dust samples collected from under equipment and in the cable vault. This despite the fact that by 1991 there had been no serious use of lead by the telephone industry in central exchanges for almost 40 years. It is sobering to think of the amount of lead that would have been found had these samples been collected in the '40s or early '50s instead, that is, at the height of lead-use by the Bell System. Read the NIOSH report, next.**Posted In March 2008**

Copied verbatim from http://41goodlife.blogspot.com/2007/05/dont-let-small-children-play-with-cell.html on March 20, 2008.

Don't Let Small Children Play With Cell Phones!

"I know this seems a little far from the major subject but this is a serious health issue. There was a report on the news that reminded me of a concern I would like to pass on.

I was employed by a cell phone company for 17 years and involved in electronic communications sense 1960. While working for the cell phone company we received many phones that were water damaged. There was always the excuse that they had not used the unit any where near water. Sometimes the phone would be green with very heavy corrosion on the bottom connector as well as the bottom of the circuit board.

When investigating to try to learn how such damage could happen it was learned that the damage was the result of allowing very young children play with them. When they stick the phone in their mouth saliva enters the unit via the bottom connector which breaks down some of the metals used in the phone which then enters the child's mouth and thus their system.

These pieces of electronic equipment contain some toxic metals. There is solder, for one, which is a lead tin alloy. I don't think I have to tell anyone that lead is toxic.

Cell phones were not designed as child chew toys. Please keep them away from young children.

Just for advice, as long as cell phones and health issues are being discussed, based on my experience and study, it is my personal opinion that the low lever radio radiation power from these units do not offer any health danger. "

Posted in January 2008

A Story About Using Lead from Old Bell System Cable to Cast Bullets

The Shiloh Rifle Manufacturing Company of Big Timber Montana sells replicas of the Sharps rifle. The Nineteenth Century prototype of this weapon fired leaded ammunition, as do many of the replicas. Many owners of Shiloh rifles that use this type of ammunition cast their own bullets. The Company runs an online user's forum. A discussion thread (see link below) was begun on 11/20/06 with the following question;

"I was curious as to how many of you know of a person that has gotten diagnosed lead poisoning from shooting and or casting?

A person responding on 2/2/07 said that he (or she) knew of two people and that

"*One guy worked for the phone company and worked with the lead cable sheathing. They let him keep the old sheathing and he casted it into 5lb ingots (rcbs & lyman) using a plumbers furnace. I used to buy all my lead from him at 25cents a pound. He got lead poisening* [sic] *and couldn't work with lead any more and my supply ended. That was in the late 80's*".

This brief response gives yet another example of Bell System lead coming home, probably to an unsuspecting family.

(http://www.shilohrifle.com/forums/viewtopic.php?t=8284&highlight=phone+company)

Posted in October, 2007

The following was copied from a 2006 posting at http://www.weldingweb.com/archive/index.php/t-4179.html on October 6, 2007.

A Former Telephone Cable Splicer Warns About the Dangers of Lead

"I doubt if there's any more around but if you wanted to see something pretty you needed to see lead work done in telephone company central office vaults.

In the sixties we could see the work they'd done in the fifties and early sixties with the lead sheathed cable and lead closures. Anyone who loves form and function enhanced with metal would go off on it. What was amazing to me was all the work was done with pot lead. That's lead that's heated up outside to a molten state and then applied with pads in the vault.

When I started in the industry we still used lead a lot for closures and in some instances, cables. The first welding I ever did was with an acetylene torch soldering lead spice closures. That was in my early twenties.

Thankfully by the time I had children we weren't working with the lead anymore as the industry was aware of the hazards it presented. It wasn't too tough on adults but it's heck on children, smaller more susceptible to it's poison.

I've decided it's all about individual physical traits when everything is considered on the fume's effect. I worked lead as a young man and it hasn't seemed to make me slow of thought most days. And I can personally tell you one of the old wive's tales about it aren't true. There's been no erectile disfunctions around here that I've been made aware of. (When I took a welding class in college the instructor told us how he'd went impotent for a year because he welded galvanized and got lead poisoning.)

I smoked three to four packs of Chesterfield Kings for many many years. I quite cold turkey and haven't had any lung issues that we know about. I also used to be exposed to a lot of silica dust in the telephone industry because we used it as a drying agent to remove the moisture in a manhole before we opened a closure.

And I still weld galvanized pipe. I did a bunch of it yesterday as a matter of fact. Inch and a half galvanized pipe handrail on a set of stairs all built in place to attempt to match another set of handrails that had been there probably since the twenties or thirties.

I've tried to figure out how come I've been exposed to all this and am still operating on twelve of eight cylinders while I've seen people drop around me who haven't been exposed to hardly anything. The only answers I can come up with is I've

been blessed with an immunity system that's pretty hardy or more likely I breathe a little different than most folks. By that I mean I seem to breath more through my nose when welding galvanized and I seem to do a lot of holding of my breath.

Now if I was a young man getting into welding I'd take this welding fume stuff real serious. Especially the stuff that we get on our clothes and take home to our family. It's almost like my dad or myself being able to roll in poison oak or ivy without any symptoms of irritation. But my mother could touch the laundry and almost have to go to the hospital for allergic reactions. So if you're working around stuff that COULD pose a hazard to your family you owe being extra careful for their sakes. Lead is a good example of that. Lead isn't bad when it's left alone. But like asbestos it becomes a hazard when it's disturbed and becomes airborne and gets in the lungs.

I know what it's like to be consumed by the project or process and completely oblivious to unimportant stuff like money or health. But just because I've been there doesn't mean I recommend anyone else do it.

Most of this stuff we're dealing with doesn't knock you down immediately. It takes it's time and gets you later. Or as in the case of lead exposure it turns your children into a lot slower thinkers and less healthy individuals than they were meant to be.

And you there's this thing like I mentioned in the beginning of this epistle. We never know where you stand in the scale of susceptibility to these hazards. If you're like me and you never were exposed to it then you'll live longer everything being equal. If you're like some other people I've known your life is going to be considerably shorter and the end isn't anything you'd wish off on anyone.

There's a lot to be said for getting old and being in good health. Not just the obvious like the look of awe in some young guy's face as he tries to duplicate a physical act you just accomplished with little effort. But stuff like walking your daughter down the aisle or having a grand daughter ask for a playhouse like you made her aunt has to go into the mix also. And you'll never know it unless you make a conscious effort to do so."

Posted in June, 2007

(the following text was copied verbatim from www.cwalocal7810.org/menu4item2.html on June 6, 2007)

A Retired Central Office Installer Tells About His Poisoning

HEAVY METAL TOXICITY

DO YOU HAVE IT????

If you were or are a splicer, installer, central office tech, etc… or soldered and handled lead during your phone company career, you may be a victim of metal poisoning.

Lead poisoning is not new. According to the World Health Organization (WHO), 80% of all chronic health concerns are associated with heavy metal toxicity. There are 14 metals considered toxic, aluminum, antimony, arsenic, beryllium, binmuth, cadmium, lead, mercury, nickel, platinum, thallium, tin, tungsten, uranium. One doctor who treats patients with autoimmune conditions such as fibromyalgia, chronic fatigue, rheumatoid arthritis, lupus, sclera derma, etc… could not find even one who tested within safe ranges of heavy metal toxicity.

Medical studies have clearly proven the link between mercury and Alzheimer's disease. Dental fillings used to look like silver, but were really more then 50% mercury in composition and called mercury amalgam fillings. At present data does not exist that indicates a "safe" level of mercury in the human body. The same goes for lead. No amount ingested by skin or by fumes is safe. Lead affects the brain cells and the nervous system. It reduces intelligence, produces memory loss, promotes neuropathy (numbness or tingling in the extremities) and deteriorates thinking equivalent to dementia. Lead paint was banned by the government in the early 1970's. Lead poisoning usually remains undetected until Alzheimer's disease takes hold. Lead can also be a factor in developing osteoporosis for adult women. It can show up as bone pain, gout, high blood pressure, iron-deficiency anemia, kidney damage and more. Lead is one of the most widely used metals in manufacture of metal products such as cable covering, certain plastics, electric cable insulation. Lead wires, pipes, roof coverings, storage batteries and solder.

Do you remember working on or wrecking out lead cable? I do! The sheath was solid lead and extremely heavy, but I never thought or was told anything about it being dangerous. Solder was 60% lead 40% tin when I started work in 1973 as an outside installer. Solder changed over the years and is now lead free which is good. If you buy it at the local hardware store you will see it is now 95% tin & 5% antimony. The lead is gone but they more than double the tin and both metals are on the list for toxic metals. Splicers used lead sheets to cover splice cases, since it was so pliable. Central Office techs soldered all day long on the racks. If a new phone went in or disconnected it was hard wired to the solder blocks or removed with an iron. There were always several soldering irons spaced out along the racks for easy access. The fumes were just as toxic as handling it. The pins were so close you sometimes had your nose less than a foot away from the soldering iron trying to reach the back pins without hitting another phone line or alarm line, the same way out in the field. There were still some old solder block terminals in Seattle when I left in 1999.

I hope this is causing you to stop and think for a few minutes. Why do I care??? In 1999 after 25 years, 11 months, I was forced to retire from the phone company due to what was diagnosed as an incurable muscle disease called Inclusion Body Myositis. A biopsy was sent to the University of Washington and the results ended my career. I was expected to be totally bedridden or at least confined to a wheelchair within one year and dead within 5 years. A mis-diagnosis!!! This year I was tested for heavy metal toxicity. It has a cure and I had nothing to lose. I am off the scale and set a new record for my doctor. Tin is allowed a maximum count of 10 I am 140, yes 140 (remember to solder?) Lead is rated at 5 maximum I am 23 more then 31/2 times the limit. Aluminum was also elevated. I drank lots of soda in aluminum cans over the years, plus used aluminum cookware and foil. Heavy metals are accumulative and never leave the body. Mercury has a maximum range of 4, mine is 14, 21/2 times the limit. I have an amalgam filling I asked a dentist to remove it, It is considered hazardous waste and required a special license and handling to remove it so he would not touch it, but I can walk around with it in my mouth and body with no problem… Right!! We all believed that one, and the tooth fairy will protect us. NOT!!! Today I am 65 years old and in a wheelchair every day and can only walk with the help of two canes for a short distance. It takes a long time to eat because the throat muscles are very weak and swallowing is difficult. My left hand can barely hold an empty paper cup. My right hand can hold only 3-5 lbs for a minute or two. I am loosing control of bladder bowel functions and my stomach can no longer assimilate food to nourish my body. THIS IS HEAVY METAL TOXICITY!!! You don't want to follow in my footsteps…. If this doesn't alarm you then read Toxic Metal Syndrome that is where a lot of this information came from. You can also do an internet search for heavy metal toxicity there is a lot of information.

Good news is there is a cure. Chelation Therapy, however my body is too far deteriorated to be able to start the therapy currently and will have to build up my digestive system first. I take 55-60 pills a day plus 7 different vitamin shots 3 times a week. If you think you may have been exposed to heavy metals then invest in your future and quality of life and get tested. Don't wait till it's too late for the cure. It cost less then $200.00 yes it can be expensive on a fixed income and

insurance may not cover it. Don't be surprised if your doctor knows nothing about it or how to test for it, mine didn't. The book I mentioned has a list of doctors for most states in the back.

How much do you like living or playing with your children or grandchildren? Mine are already starting to forget when I could run and play or even walk. Will yours?

(name withheld)

Installation/Repair

Oregon?Washington

1973-1999

PO Box ____ Tonasket WA

Posted in April, 2007

A letter to "Dr. Gott" From a Retired (and Worried) Telephone Cable Splicer

"Ask Dr. Gott" is a syndicated column that appears regularly in newspapers across the country. This particular column was published online in the Northwest Herald, Crystal Lake, Ill., on Sunday, April 15, 2007. [iii]

"Dear Dr. Gott: After graduation from high school in June 1940, I was fortunate enough to be hired as a cable splicer's helper (later promoted to an outside supervisor) for the Chesapeake and Potomac Telephone Co., of Virginia.

Those were the days when all outside telephone cables were manufactured by Western Electric Manufacturing Co. and all cables to be placed outside had lead sheaths. After splicing the paper-pulp insulated copper wires together at splice locations, the next step in the process was to make the cable airtight and watertight. This was accomplished by using a section of lead sleeving, actually a length of lead pipe sawed off to the length needed. The next step was to form the lead sleeve around the spliced wires, and the last step was to wipe lead joints to seal the water and air out.

My purpose for relating this information to you was to ask you the following question: As a cable splicer for 11 years and handling lead hundreds of times, what was the effect upon my health and that of my wife and two children (now adults)?

I continue to have health check-ups. So far, my health has been reasonably good. I did, however, about 12 years ago, have two operations on my head for hydrocephalus. I recovered nicely from these, but after about

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www.suburbanchicagonews.com/couriernews/lifestyles/gott/

340416,3_7_GOTT_S1.article

two years, the hydrocephalus returned, and I had two more operations on my head, which I also recovered from."

To which Dr. Gott replied;

Dear Reader: I am publishing your entire letter because your profession is fascinating, and I want my readers to appreciate your commitment to your job.

Hydrocephalus (an increase in pressure within the brain's fluid) is not ordinarily the result of lead exposure of the type you had. Nor should the exposure affect your friends and family.

Nonetheless, to be certain, you should raise this issue with your family physician, who may choose to measure the lead level in your body."

Comment;

No large in-life study has ever been done on the long-term effects of lead poisoning in humans.[iv] Although there may be a number of reasons for this inactivity the biggest one has to be a lack of funding for research. Once federal and state legislation was passed removing lead from gasoline and from most consumer products, government and philanthropic interest in lead poisoning seemed to wane.

Judging from comments made by the writer of this letter he was in his early seventies when he had his initial surgery for hydrocephalus, and he was about seventeen years of age when he began working as a cable-splicer's apprentice. So from the date of his initial exposure to lead until his surgery more than 50 years had passed.

Although it is well known from published studies as far back as 1900 in both experimental animals and in humans, that acute and chronic lead poisoning can have pathological effects on the brain, including inflammation, the question as to whether lead poisoning can cause hydrocephalus in adult humans has never been studied. However hydrocephalus has been produced in experimental animals by feeding them lead, and hydrocephalus is known to occur under certain circumstances in young children with lead poisoning. In both instances inflammation of the meningeal cover of the nervous system is thought to have played a role. In addition, in both adults and children hydrocephalus may be a late-occurring complication of bacterial meningitis, another cause of meningeal inflammation.

So although the question remains unanswered for the writer of this letter to Dr. Gott, there is no theoretical reason why lead poisoning could not have been the cause.

References:

Courtney JW: "A case of multiple cerebral hemorrhages from chronic lead poisoning with necropsy". Boston Medical and Surgical Journal, 1900, vol cxlii, 136-138.

Hirano A, Mikoto I, "Neuropathology of lead intoxication", pgs 40,47,57; in Handbook of Clinical Neurology, vol 36, p 35-64, Elsevier Publications, 1979.

[iv] A small study of the effects of lead poison on blood pressure, kidney function, and hemoglobin levels in adult subjects who had lead poisoning 50 years earlier as children was published in the American Journal of Diseases of Children , vol 145, June, 1991.

Sharma RR, Chandy MJ, Lad SD.Transient hydrocephalus and acute lead encephalopathy in neonates and infants. Report of two cases. British Journal of Neurosurgery. 1990;4(2):141-5.

Pappas CL, Quisling RG, Ballinger WE, Love LC. Lead encephalopathy: symptoms of a cerebellar mass lesion and obstructive hydrocephalus. Surgical Neurology. 1986 Oct;26(4):391-4.

Mirando EH, Ranasinghe L. Lead encephalopathy in children. Uncommon clinical aspects. Medical Journal of Australia. 1970 Nov 21;2(21):966-8.

Posted in July, 2008

The Hazards of PILC Cable

Visit www. http://www.tci-pcb.com/article.htm to learn why lead exposure and lead poisoning are not the only hazards associated with paper-insulated lead-covered (PILC) cable. This type of cable was used by both the telephone and power industries to transmit telephone calls (in the case of the former) or electricity (in the case of the latter).

After viewing this web site recall that not one word of caution was ever given to Bell System field employees by the regional operating companies regarding the handling of this type of cable.

Posted in January, 2009

"Lead in your telephone" – advertisement in the Saturday Evening Post

The advertisement on the following page appeared in the Saturday Evening Post sometime in 1927.

Lead Poisoning and Stuttering

Anyone over the age of sixty can remember when stuttering was common. Many people knew a child or adult who stuttered, sometimes severely. Today the sounds of stuttering have virtually disappeared. "*To one who has worked in the field of stuttering for many years,"* wrote a leading speech pathologist in 1982, "*it appears that the incidence of stuttering has been declining in the last 30 years … when the author of this text began to practice in 1934 the high schools seemed full of stutterers…this does not seem to be true today*." [1] Five years later that view was echoed in A Handbook on Stuttering [2] in 1987, "… *there is a widespread impression among American clinical workers of long experience that the prevalence of stuttering is considerably less than it was some decades ago* ".[v] Neither writer offered a cogent view as to why that was the case.

However buried in a collection of studies published 50 years ago by the University of Iowa on the origins of stuttering in children, are data that suggest that at least some of those studied had lead poisoning. Although lead has long been known to cause motor speech and language disorders in children, and although there is at least one published report of stuttering in an adult following acute poisoning, no study has ever been published that looked for a connection between lead poisoning and stuttering and then presented data supporting the conclusions.[vi, vii, 3, 4, 5, 6]

[v] A Handbook of Stuttering, p 123.

[vi] See also, www.lead.org/bellsystemleadpoisoning/images/an investigation of circumstances.pdf, footnote # 10.

[vii] A search of the National Library of Medicine's "PubMed" failed to find any publication using "lead poisoning" and "stuttering" as search terms. However reference in the literature has been made at least as far back as 1942 to speech and language problems in children with lead poisoning.

The purpose here however is not to prove that lead poisoned even one child. Proof requires physical evidence of lead in the body, evidence that was never collected by the Iowa investigators. Rather the purpose is to build an argument based on data newly viewed in the light of more than 50 years of lead poisoning research. An argument that poisoning could have been present and likely was.

The Iowa experience began in the early 1930s with the first of three consecutive studies. The last study ended in the late 1950s. Using only the tools of the social scientist - in this case a series of questions of parents of children who stuttered (Experimental Group) and parents of children who did not (Control Group) - investigators set out to explore why children stuttered. The questions (addressing parents, children, or both) dealt with education, upbringing, occupation, social and economic status, behavior and intelligence. Answers were compared and differences tested for statistical significance.[7, 8] The studies were made all the more powerful by the fact that each was concurrently controlled by a group of children who had never stuttered. In Study II for example, *"…each of the fifty families in the Experimental Group was matched with a family having a child of like age and sex who did not stutter",* wrote Dr. Frederic Darley, the lead investigator, *"…a third basis was used for matching, namely, socioeconomic status of the child's family….all mothers and fathers were interviewed separately…".*[viii] Similar procedures were followed in Study III. A brief description of demographics and control procedures for each of the three studies can be found in Table 1.

In the 50 plus years since publication of the Iowa studies, scientists have learned that lead poisoning can lead to Attention Deficit Hyperactivity Disorder (ADHD) with its attendant daydreaming and distractibility; [9, 10, 11] lowered intelligence; [12] juvenile delinquency; [13, 14] and behavioral changes.[15]

Today there is more than enough information available to draw a picture of a typical child with lead poisoning living in the middle of the last century. Compared to a lead-free child such an individual would more likely have been raised in a working class home (and therefore potentially exposed to lead dust brought home from work),[16, 17] been slow as a toddler to acquire speech, [18, 3, 28, 19] had trouble focusing on the task at hand (that is, to be attention deficient), day-dreamed excessively, been considered not as mentally sharp as his or her peers, been a discipline problem (and therefore more at risk for juvenile delinquency), engaged in thumb and finger sucking (and therefore more likely to have ingested lead), [20, 21] suffered from sleep disturbance,[22] and repeated school years. As it happens, this is remarkably close to, if not exactly the same as, the description given by the Iowa researchers of the children who stuttered.

The figures on the following pages summarize the results of a supplemental analysis of the Iowa data. The analysis was done to look for a link between lead poisoning and stuttering. Data supporting the figures (data originally published in the book The Onset of Stuttering: research findings and implications [23] in 1959) can be found in Table 2. In the preamble to the appendix, the book's lead author Dr. Wendell Johnson wrote, "*the main purpose of making the findings* [of Studies II and III] *available in this detailed fashion are to allow essential reference to them in the body of the report and to encourage further evaluation of them and continued development of their implications, not only by students of the stuttering problem but also by…medical investigators and others…".* [ix]

[viii] The Onset of Stuttering, pgs 12, 19.

[ix] The Onset of Stuttering; *explanatory notes to the summary table*, Appendix A.

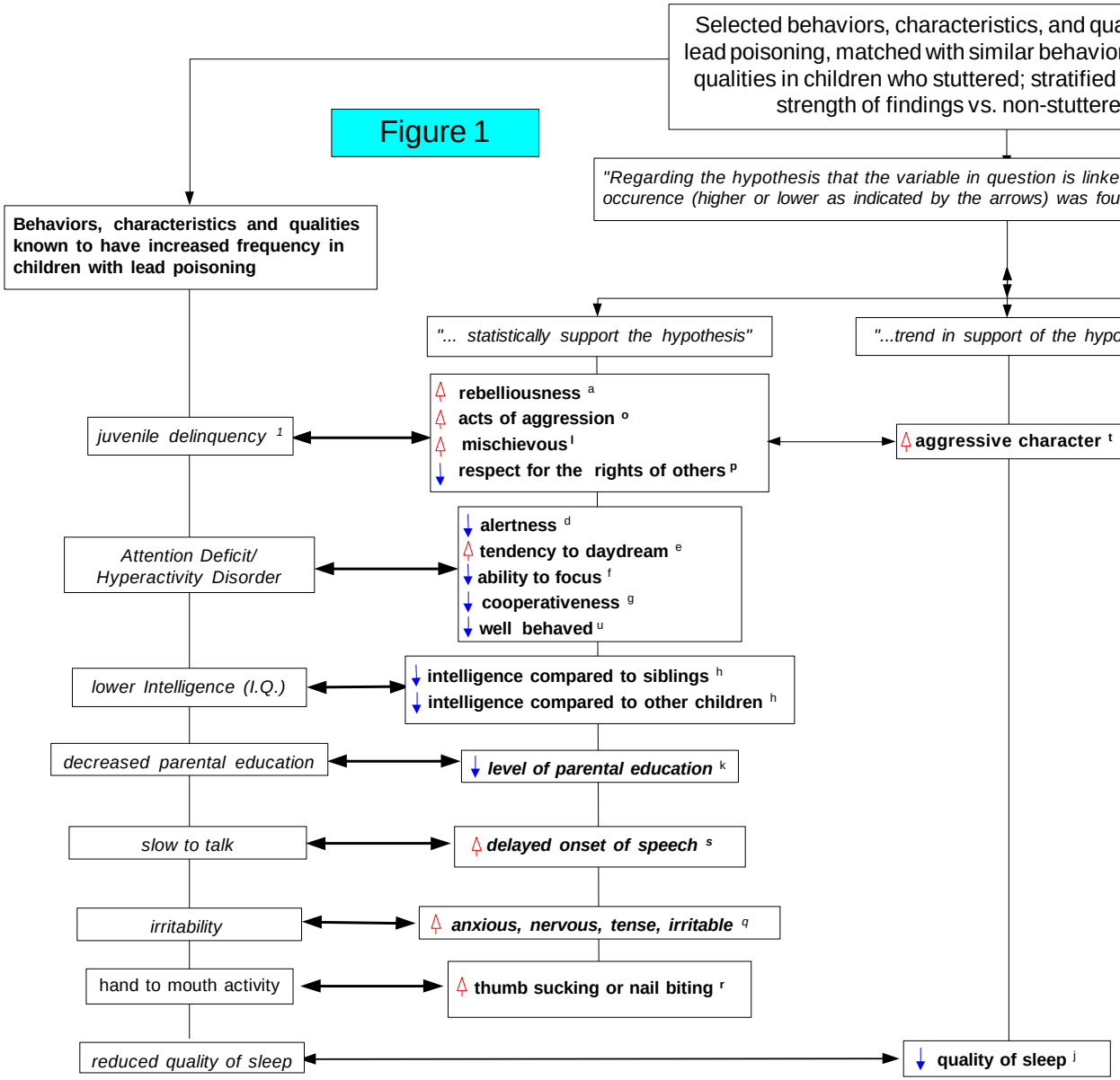
Table 1

A brief description of research carried out at the University of Iowa comparing children who had a stuttering speech dysfluency with children who did not; 1934 to 1957

	Study I [24]	Study II [25]	Study III
Dates conducted	1934-'40	1948-'52	1952-'57
Subjects enrolled;			
• Stutterers (Experimental Group)	32 boys 14 girls	39 boys 11 girls	107 boys 43 girls
• Non-stutterers (Control Group)	33 boys 13 girls	39 boys 11 girls	107 boys 43 girls
Ages of subjects;			
• Stutterers (range)	27 mos - 9 yrs, 3 mos (median 4 yrs, 2 mos)	28 mos - 14 yrs, 4 mos (mean 8 yrs, 8 mos) 24 mos – 14 yrs (mean 9 yrs)	27 mos - 8yrs (mean 5 yrs) 28 mos – 8 yrs, 7 mos (mean 5 yrs)
• Non-stutterers (range)	27 mos – 9 yrs, 10 mos (median 4 yrs, 5 mos)		
Non-stutterers matched with stutterers on variables	"Sex, age, intelligence level"	"Sex, age (± 6 mos) and socioeconomic status"[x]	"sex, age, socioeconomic status"
Definition of stuttering	As defined by a parent, other family member, or teacher	As defined by both parents, school health official, or state health nurse	As defined by one or both parents

[x] Use was made by Johnson and his colleagues of the classification system described in Social Class in America: a manual of procedure for the measurement of social status; W.L.Warner, M. Meeker, K. Eells. Peter Smith (pub); Glouster, MA. 1957.

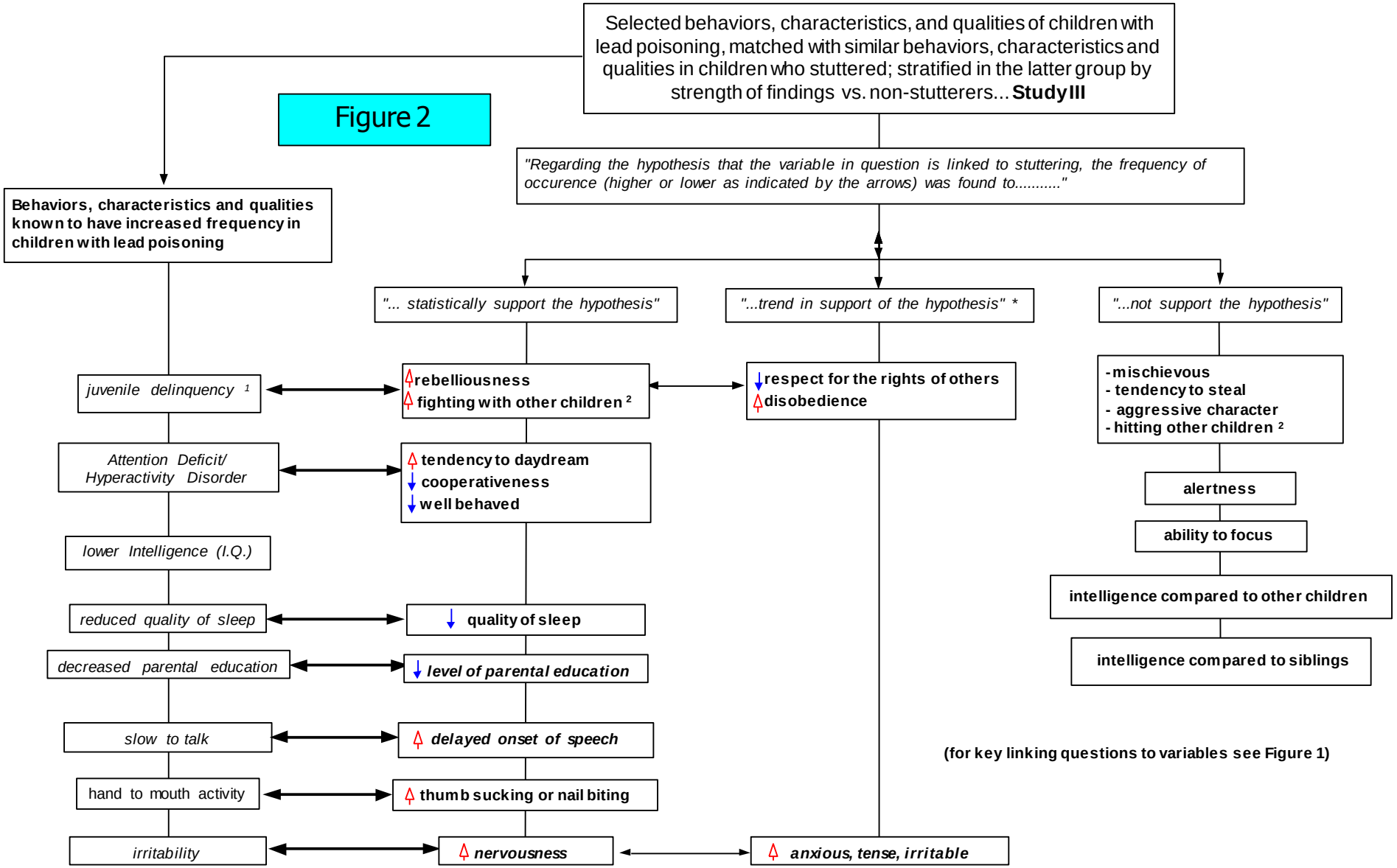
Figure 1



Variables keyed to questions asked of parents by the Iowa investigators. The numbered questions can be found in Table 2.		
Footnote lettered..	Variable	Question number(s)
a	"rebelliousness"	6
d	"alertness"	2
e	"tendency to daydream"	3
f	"ability to focus"	8
g	"cooperativeness"	18
h	"intelligence"	4, 17,16
j	"quality of sleep"	15,20
k	"level of parental education"	1
l	"mischievous"	5
m	"tendency to steal"	10
n	"disobedience"	11
o	acts of aggression	12,13
t	"aggressive character"	19
p	"respecting the rights of other"	23
q	"irritable, tense, nervous,etc."	22, 14
r	"thumb sucking, nail biting"	9
s	"delayed onset of speech"	25,24
u	"well behaved"	7

** Empirically a trend was said to be present when the frequency of occurence (higher, or lower where appropriate) although failing to reach statistical significance vs. non-stutterers never-the-less favored the hypothesis by a small but measureable amount.*

[1] Although children in the Experimental Group in Study II were reported as being significantly more likely to hit and fight with other children (questions # 12, 13; in Study III only to fight not to hit), they were also reported as being either no (Study III) or only slightly more (Study II) aggressive than children in the Control Groups (question # 19). Darley administered the Rogers Test of Personality Adjustment (see endnotes 25 and 31) to 28 stutterers and 18 non-stutterers in Study II. Nine stutterers (32%) and 5 non-stutterers (28%) rated "high" on the Family Maladjustment score (ref 25, p 133), while 18 stutterers (64%) and 8 non-stutterers (44%) rated "high" on the Social Maladjustment score (ref 25, p 132).



** Empirically a trend was said to be present when the frequency of occurence (higher, or lower where appropriate) although failing to reach statistical significance vs. non-stutterers never-the-less favored the hypothesis by a small but measureable amount.*

[1] *see footnote Figure 1*

[2] *In Figure 1 combined under the heading 'acts of aggression'*

It is clear from the figures that results from the two studies are not the same. In both studies all case material (that is, children who stuttered) came from referrals. This usually meant that "…*the fact that his family had been concerned enough about his nonfluencies to label them and to seek professional help was sufficient warrant for use of the case* [in one of the two studies] " [xi]. In Study II, "family" meant that both parents agreed that the child stuttered, in Study III it meant one or both parents. In an effort to avoid selection bias it was only after enrolment that an evaluation was made of the child's stuttering. As a result, 47 of the 150 children (31%) enrolled in the Experimental Group in Study III, and 7 of the 50 children (14%) enrolled in the same group in Study II were found not to be "clinical" stutterers.[xii, xiii] In addition, while there were a total of three interviewers in Study II, in Study III there were seven, injecting a greater (albeit unavoidable) degree of variability into the interviewing process. As a consequence, and contrary to any expectation that a larger study would lead to better results, the data from Study III exhibited a greater degree of distortion than data from Study II.[xiv] All of this makes the results of Study III more difficult to interpret and any interpretation more prone to error.

There were other important differences between Studies II and III. Children in Study II were, on average, three and a half years older than children in Study III. While 81% of Study II children had entered first grade, only 21% of Study III children had done so (ref 23, Appendix A, p 186). Because of this more parents in Study II would have had the opportunity to witness their child's academic performance than would have parents in Study III. This might help explain the difference in perceived intelligence between the two groups of children. Also, children enrolled in Study II would have on average reached toddler age (an age at which children put things in their mouths, things contaminated with lead dust brought home from work on clothing) in 1942. Children enrolled in Study III would have reached the same age in 1949, again on average. Throughout the 1940s but especially during the war years there was intense use of lead in American industry, use that was largely unregulated. Together these factors made the '40s the most dangerous decade of the last century for occupational lead exposure.

Within a 100-mile radius of Iowa City, encompassing an area from which most referrals would have originated, there was heavy war-related manufacturing, first because of World War II and then the Korean War. A prime example of this is the Iowa Army Ammunition Plant near Burlington, Iowa, less than 70 miles from Iowa City where most of the research on stuttering took place. The plant, one of the largest production facilities of its kind in the world, and even today employing thousands of workers, was undoubtedly a major consumer of lead.[xv] According to Wikipedia [xvi] the plant had a period of peak production between 1941 and 1945 when production stopped. Production then ramped up again sharply beginning in 1950. It would have again slowed or stopped in 1953 with the Korean Armistice. Another example would have been the Collins Radio Company located in Cedar Rapids, just 30 miles from Iowa City. The largest supplier of aviation communication equipment during WWII, Collins would have been a major consumer of lead wire solder for many of the years in question. All

[xi] The Onset of Stuttering, p 12.
[xii] Data from all enrolled children were included in the analysis of results.
[xiii] The Onset of Stuttering, Appendix A, question # 614, p.194.
[xiv] One measure of distortion is skewness. In Study II (that is, Study II data in Table 2) skewness of the father's responses ranged from .95 to 1.26, while the mother's ranged from .91 to 1.12. In Study III (that is, Study III data in Table 2) the same measures ranged from 1.30 to 1.38 for the fathers and 1.27 to 1.35 for the mothers. In all cases the fathers' responses demonstrated greater skewness than the mothers'.
[xv] See "Exhibits" page
[xvi] http://en.wikipedia.org/wiki/Iowa_Army_Ammunition_Plant

of this points toward children enrolled in Study II as having had more opportunities to be exposed to higher levels of lead for longer periods of time than children in Study III.

There are other clues in the Iowa data that support the occurrence of lead poisoning in children who stuttered. In Study III information was gathered on the birth order of study subjects (similar information was not obtained in Study II). Compared to the Control Group, children who stuttered were more often the oldest child in the family. The difference between groups was statistically significant. This meant that the child who stuttered was more likely to have reached toddler age earlier in the 1940s – a time of rampant and uncontrolled occupational lead exposure – than children who did not stutter. Also Darley stated that stutterers showed a significantly greater tendency than non-stutterers to be held back in school (ref 25, p 91).[xvii] Academic failure, including having to repeat a school year, has also been reported for children with lead poisoning. [26, 27, 28]

Admittedly it can be argued that there could be reasons other than lead poisoning for some of the observations. For example the stuttering child's acts of aggression might have been solely in response to playground taunts given because of the stuttering. Or perhaps the stuttering itself caused the child to be nervous, tense, and rebellious. On the other hand there are observations that cannot be explained by the stuttering, such as delayed onset of speech, behavior suggestive of an attention deficit, an inability to focus, level of parental education, and perceived intelligence. These and other findings would have to be accounted for in any alternate explanation.

Taken as a whole the data are remarkably consistent and paint a picture of a life under stress for a child who stuttered. That picture in all its particulars except stuttering is interchangeable in whole or in part with any number of published descriptions of children with lead poisoning.[xviii] Furthermore, if Dr. Charles Van Riper's belief, that by 1982 stuttering had been in decline for 30 years, can be taken literally, then turning the calendar back 30 years one arrives at 1952. And in 1952 there was a seminal event in the history of occupational lead exposure. For in that year the American lead industry publicly acknowledged for the first time that unprotected occupational lead exposure was dangerous, and that steps were being taken to curb the worst abuses.[29] Thus the incidence of stuttering as well as that of lead poisoning following occupational exposure began their slow declines hand-in-hand.

It has long been accepted that neurological dysfunction is one cause of stuttering. In the middle of the last century surely one reason for that dysfunction was lead poisoning.

[xvii] Darley stated that almost four times as many stutterers as non-stutterers were held back in school (ref 25, p 91). Intelligence tests were administered to the 15 stutterers forced to repeat all or part of a school year. Five of the 15 were found to have below normal I.Qs. (p. 95). The remaining 10 fell into the range of "average" or above. No intelligence tests were administered to non-stutterers.

[xviii] Since human physiology continues to develop and mature throughout childhood, the detrimental effect of lead in a child is a function not only of the dose (or doses) of lead, but also the age of the child - down to the month – at which the dose is absorbed, whether the child is fed or fasting at the time of lead ingestion, and the cumulative amount of lead in the child's body. As a result children in seemingly identical circumstances can have very different experiences following exposure to lead.

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures.		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.											
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
1. Level of education [of parents]? (only father's response) [xx, 30]	≥ 1 yr of postgraduate work	7	15	17	36	X			X		
	graduate of 4yr college or equivalent	6	9	22	25						
(#13, p.2)	college ≤ 2 yrs	5	6	25	14						
	h.s. grad only	19	9	60	58						
	attended h.s. did not graduate	4	4	15	14						
	completed 3rd to 8th grade only	9	7	9	3						
	"Uncertain;don't know;can't say"	0	0	0	0						
	(missing responses)	0	0	2	0						
2. As compared to other children, how alert is your child?	Much > than average	5	13	44	40	X				X	
	Somewhat > than average	34	48	113	126						
	About average	53	32	134	132						
(#377, p. 128)	Somewhat < average	8	6	9	2						

[xix] U = *"unknown" or "unsure"*

[xx] That is, when compared to the fathers of control subjects, are the father's of stutterers less educated on the whole? The difference in education between control and experimental Study II mothers was not significant, while the difference between control and experimental Study III mothers was significant at p=.03 (chi-square). Men were often granted deferment from the military draft if they were employed in a critical defense industry.

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

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Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
	Much < average	0	1	0	0						
	"Uncertain;don't know;can't say"	0	0	0	0						
	(missing responses)	0	0	0	0						
3. As compared to other children, how much does your child daydream? [31]	Much > than average	7	3	4	2	X			X		
	Somewhat > than average	20	15	29	25						
	About average	45	41	189	147						
(#399, p.133)	Somewhat < average	15	39	56	78						
	Much < average	3	2	8	24						
	"Uncertain;don't know;can't say"	9	0	14	23						
	(missing responses)	1	0	0	1						
4. How do you think your child compares in intelligence with neighborhood children?	Much > than average	7	11	24	22	X				X	
	Somewhat > than average	27	42	104	111						
	About average	59	41	160	160						
	Somewhat < average	6	1	8	2						
(#597, p. 188)	Much < average	0	2	0	0						
	"Uncertain;don't know;can't say"	1	0	3	4						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures. *In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.*		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
	(missing responses)	0	3	1	1						
5. As compared with other children, how mischievous is your child? *(#373, p.127)*	Much > than average	2	2	12	9	X				X	
	Somewhat > than average	19	10	47	58						
	About average	52	48	175	161						
	Somewhat < average	22	31	53	53						
	Much < average	5	9	13	18						
	"Uncertain;don't know;can't say"	0	0	0	1						
	(missing responses)	0	0	0	1						
6. As compared with other children, how rebellious is your child? *(#382, p. 129)*	Much > than average	2	0	13	13	X			X		
	Somewhat > average	22	11	65	49						
	About average	60	56	189	192						
	Somewhat < average	12	30	24	36						
	Much < average	4	3	7	10						
	"Uncertain;don't know;can't say"	0	0	2	0						
	(*missing responses*)	0	0	0	0						
7. As compared with other children, how well	Much > than average	6	15	8	11	X			X		
	Somewhat > average	21	32	72	71						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures.		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.											
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
behaved do you think your child is?	About average	64	49	191	209						
	Somewhat < average	8	1	27	8						
(#535, p. 177)	Much < average	1	0	1	1						
	"Uncertain;don't know;can't say"	0	0	0	0						
	(*missing responses*)	0	3	1	0						
8. As compared with other children, how able is your child to concentrate?	Much > than average	3	8	25	16	X			X		
	Somewhat > average	14	27	61	58						
	About average	59	52	178	199						
	Somewhat < average	21	12	30	18						
(#384, p. 130)	Much < average	1	1	1	0						
	"Uncertain;don't know;can't say"	0	0	5	9						
	(*missing responses*)	2	0	0	0						
<u>How often is the following behavior occurring:</u>											
9. Thumb sucking or nail biting? (*this combines what was originally two separate questions,*	Very often	NA	NA	43	30	X			X		
	Quite often	NA	NA	50	23						
	Occasionally	NA	NA	60	51						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures.		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.											
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
thumb sucking and nail biting. For that reason 'n' is doubled)	Never	67	76	406	458						
	Often	25	16	NA	NA						
(#s 438,444; pgs 144, 145)	Seldom	8	8	NA	NA						
	"Uncertain;don't know;can't say"	2	0	1	0						
(*Note: change in 'n' for this question.*)	(*missing responses*)	0	0	0	0						
	n	100	100	300	300						
10. Stealing? (in Study II asked only of the mothers)	Quite often	NA	NA	1	1		X			X	
	Occasionally	NA	NA	21	16						
	Never	45	46	276	273						
(#456, p. 148)	Seldom	5	4	NA	NA						
	"Uncertain;don't know;can't say"	0	0	0	0						
	(*missing responses*)	0	0	0	0						
11. Disobedience?	Very often	NA	NA	10	6		X			X	
	Quite often	NA	NA	43	37						
(#462, p. 150)	Occasionally	NA	NA	221	216						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures. *In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.*		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
	Never	2	4	26	28						
	Often	13	14	NA	NA						
	Seldom	35	32	NA	NA						
	"Uncertain;don't know;can't say"	0	0	0	1						
	(*missing responses*)	0	0	0	0						
12. Hitting other children?	Very often	NA	NA	6	5	X					X
	Quite often	NA	NA	39	29						
	Occasionally	NA	NA	179	196						
(#426, p. 140)	Never	4	23	70	44						
	"Uncertain;don't know;can't say"	0	0	0	2						
	Often	7	4	NA	NA						
	Seldom	39	23	NA	NA						
13. Fighting?	Very often	NA	NA	7	8	X			X		
	Quite often	NA	NA	38	27						
	Occasionally	NA	NA	198	171						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures. *In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.*		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
(#428, p. 141)	Never	5	19	57	72						
	"Uncertain;don't know;can't say"	0	0	0	2						
	Often	9	8	NA	NA						
	Seldom	36	23	NA	NA						
14. Nervousness?	Very often	NA	NA	28	6	X			X		
(in Study II question asked only of the mother)	Quite often	NA	NA	68	21						
	Occasionally	NA	NA	133	98						
	Never	5	17	66	149						
(#406, p. 135)	"Uncertain;don't know;can't say"	0	0	2	1						
	Often	32	15	NA	NA						
	Seldom	13	17	NA	NA						
	(missing responses)	0	1	0	0						
15. Nightmares?	Very often	NA	NA	2	1		X		X		
(in Study II questions asked only of the mother)	Quite often	NA	NA	3	0						
	Occasionally	NA	NA	113	58						
(#410, p.136)	Never	22	27	144	165						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures. *In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.*		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
(Note:change in 'n' for this question)	"Uncertain;don't know;can't say"	0	0	0	3						
	Often	1	3	NA	NA						
	Seldom	27	20	NA	NA						
	n	50	50	262	224						
16. Which of your children do you consider the brightest? *(#606, p. 191)*	Present case	11	14	60	39						
	Other than present case	37	21	67	97	X				X	
	No difference	22	48	90	99						
	(no response, only child)	22	10	50	30						
	"Uncertain;don't know;can't say"	8	7	33	35						
	(*missing responses*)	0	0	0	0						
17. Which of your children do you consider the slowest (dullest)? *(#607, p. 191)*	Present case	28	12	50	51						
	Other than present case	20	15	69	72	X				X	
	No difference	21	47	90	100						
	(no response, only child)	22	10	50	30						
	"Uncertain;don't know;can't say"	9	16	41	47						
	(*missing responses*)	0	0	0	0						
18. As compared with	Much > than average	5	15	16	12	X			X		

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures.		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.											
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
other children, how cooperative is your child?	Somewhat > average	25	29	68	86						
	About average	50	44	167	175						
	Somewhat < average	16	11	46	24						
(#379, p. 128)	Much < average	4	1	1	2						
	"Uncertain;don't know;can't say"	0	0	2	1						
	(*missing responses*)	0	0	0	0						
19. As compared with other children how aggressive is your child?	Much > than average	6	4	24	18		X			X	
	Somewhat > average	18	18	68	85						
	About average	46	45	156	141						
	Somewhat < average	22	29	46	49						
(#385, p. 130)	Much < average	7	4	4	2						
	"Uncertain;don't know;can't say"	1	0	2	5						
	(*missing responses*)	0	0	0	0						
20. How well does the child sleep? (asked only of the mother)	Very well	35	41	173	244		X		X		
	Average,fair	11	9	97	47						
	Poorly	4	0	24	6						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures.		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.											
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
	Variable	NA	NA	6	3						
(#170, p. 48)											
21. Birth order of child being studied	Oldest	NA	NA	56	41	-	-	-	X		
	Youngest	NA	NA	45	53						
(#726, p. 219)	Second	NA	NA	16	27						
22. What is the usual mood of the child? *(parents would sometime describe the child using more than one category. For that reason responses are expressed here as percent of total # of responses for that category.) (#176, p.50)*	Usually happy, affectionate, good-natured, jolly, pleasant, cheerful, contented, easygoing….	37%	55%	81%	81%			X			X
	Cranky, sensitive, impatient, moody, quick-tempered, teasing, devilish, irritable, etc.	17%	7%	10%	8%						
	Very excitable, nervous, tense, energetic,	34%	8%	3%	2%						
	Neutral, even-keel, quiet, reserved, daydreamy, serious	12%	31%	7%	8%						
23. As compared with other children, how much does your child respect the rights of	Much > than average	4	4	7	8	X				X	
	Somewhat > average	14	26	32	39						
	About average	62	52	212	211						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures.		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.											
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
others?	Somewhat < average	16	7	43	37						
	Much < average	2	0	1	2						
(#401, p. 134)	"Uncertain;don't know;can't say"	0	1	4	3						
	(*missing responses*)	2	10	1	0						
24. Do you consider the child to have been slow in beginning to talk in comparison with other children?	Much faster than average	2	4	34	25	X			X		
	Somewhat faster than average	14	25	50	69						
	About average	46	52	139	150						
	Somewhat slower than average	24	14	46	49						
	Much slower than average	12	5	30	5						
(#182, p. 51)	"Uncertain;don't know;can't say"	2	0	1	2						
25. When did the child speak his first words (in months)? *	Minimum	6	6	4	5	-	-	-	-	-	-
	Maximum	18	24	30	30						
	Mean	10.9	9.9	10.9	10.8						
(#180, p. 51)	Median	11	-	11.4	10.7						

Table 2

Two case-controlled studies, of children 14 years of age or younger who allegedly stuttered, conducted by the University of Iowa Department of Speech Pathology, 1948-1957; frequency of response to selected questions of parents

Orange color-coding identifies data analyzed as primary response variables. See endnote # 7 for an explanation and discussion of analysis procedures. *In some instances responses were not coded identically in both studies. Where this occurred "NA" appears for the non-applicable study.*		Study II For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Study III For each category parent's responses are summed except when noted. When summed, cell frequencies reflect [n x 2]		Regarding the hypothesis that stuttering and the variable are linked, do the response frequencies support the hypothesis in a way that is statistically significant?					
Question *(# and location of source question, appendix A, ref 23)*	Response coded as…	Stutterers (n=50)	Non-Stutterers (Control) (n=50)	Stutterers (n=150)	Non-Stutterers (Control) (n=150)	Study II			Study III		
						Yes	No	U [xix]	Yes	No	U
	90th percentile			17.1	16.7						
(*Note: change in 'n' for this question)*	n	3	5	137	131						

Exhibits

Lead paper weight embossed with the logo of Mason and Hanger, Inc. and offered for sale on an Internet auction site. Seller stated that the weight was from the Iowa Army Ammunition Plant. Mason and Hanger, an engineering, architectural and planning firm, operates the Plant for the US Army as it has since the 1940s. Lead azide is (or was) a component of explosive ordinance and is itself explosive. What looks like a potted plant embossed to the left of the logo is actually a stylistic representation of an early bomb.

Acknowledgement and Disclaimer

The text of this particular edition of LEAD Action News was originally in the form of three articles published at http://www.bellsystemleadpoisoning.com/ *but with the demise of that website, we are very grateful to have been given permission by its author to re-publish all the articles on it at* http://www.lead.org.au/bellsystemleadpoisoning/Lead%20exposure%20and%20Lead%20poisoning%20in%20the%20Bell%20Telephone%20System.htm *and we are particularly keen for our readers to read the three articles reproduced in this newsletter.*

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End Notes for Lead Poisoning and Stuttering

[1] Charles Van Riper, The Nature of Stuttering, 2nd ed., p 49; Waveland Press, Prospect Heights, Illinois, 1982.
[2] Oliver Bloodstein, A Handbook on Stuttering, National Easter Seal Society, 1987.
[3] S. Mayfield, "Language and speech behaviors of children with undue lead absorption: a review of the literature". Journal of Speech and Hearing Research, 26; pp 362-368, 1983.
[4] C. D. Jenkins, "Lead poisoning in children: a study of forty five cases", AMA Archives of Neurology and Psychiatry, 77 (1), pp 70-78, 1957.
[5] R.B. Mellins, C.D. Jenkins, Epidemiological and psychological study of lead poisoning in children". Journal of the American Medical Association, 158(1); pp. 15-20; 1955.
[6] "Epidemiologic Notes and Reports Lead Poisoning Among Sandblasting Workers -- Galveston, Texas, March 1994". Morbidity and Mortality Weekly Report, 44(3); pp 44-45, January, 1995.
[7] According to the authors, data from Studies II and III were entered into contingency tables and tested for statistical significance using Chi-square (ref. # 23, p.26). The supplemental analysis reported here used similar procedures. In both cases Chi-square (or Fischer's Exact, see below) was chosen because the analysis compares two independent variables.

For this report data were first entered into contingency tables. Any table containing an expected cell populated with fewer than five observations was analyzed using Fischer's Exact test (VassarStats.com) if the number of such cells in the table exceeded 20% of the total number of expected cells (see ref # 8, p. 20). Otherwise analysis was by Chi-square (CHITEST, Microsoft Excel 2000). In some cases where Fisher's Exact test was called for the total population of a table exceeded the software program's ability to calculate the test. In that case sparsely populated cells were merged with adjacent cells in the direction of the table's central tendency until the requirements for Chi-square were met.

There has been criticism leveled toward the merging of cells in a contingency table for any purpose (see ref # 8, p. 11). The criticism is along the lines that merging can lead to spurious associations where in fact none exist. Intuitively it seems logical that merging could lead to a loss of information, although in this instance it would seem to work against rejecting the null hypothesis (that is, the hypothesis that stutterers and non-stutterers come from the same population) and instead increase the likelihood that the hypothesis would be falsely accepted. For that reason merging would seem to be conservative rather than radical. The ordering of response, from "*much greater than average*" to "*much less than average*" for example, could be viewed as a tool designed to coax a response from a reticent parent rather than as a functional scale. For example if a parent were to be asked whether they thought their child's intelligence was either "*average or greater*" or "*less than average*" they might be hesitant to respond at all if they thought their child's intelligence was clearly better than average. The researcher's interests however might lie in exactly such a dichotomous response, knowing that it would be more difficult to define, quantify, or explain the difference between "*much less than average*" and "*somewhat less than average*", for example, than a simpler scale. Never-the-less in the interest of parsimony merging of cells in this study was done only to meet the operational requirements of either Chi-square or Fischer's Exact test and for no other reason. Darley gave much this same reason for merging cells in his original analysis (ref 25, p.85).

Both Fischer's Exact and Chi-square are of course nondirectional tests. Data entered into a contingency table and analyzed by either will have the same result whether the matched data are ordered or not. In the event that either test rejects the null hypothesis a direction must be established. This is accomplished through the use of percentage. For example, in question # 8, Chi-square does not tell us if children who stutter are able to concentrate better or worse than children who do not. All that is known is that the two groups do not concentrate the same. However 35% of non-stutterers are able to concentrate better than average compared with only 17% of stutterers. This procedure was followed throughout the report for all variables.

Finally, an analysis of missing data was done for the variables in Table 2. Missing data occurred when a parent did not know what answer to give, and therefore gave no answer at all, or when data were missing for unknown reasons. In the Study II Control Group, fathers on 18 occasions and mothers on 23 occasions failed to respond to a question. In the Experimental Group fathers on 17 occasions and mothers on 16 occasions failed to respond (chi-square; $p = 0.139$ for the difference between groups). In the Study III Control Group, fathers on 75 occasions and mothers on 67 occasions failed to respond. In the Experimental Group fathers on 60 occasions and mothers on 60 occasions failed to answer a question (chi-square; $p = 0.05$ for the difference between groups). For no missing variable in either Study was the difference in response frequency between control and experimental statistically significant.

[8] For this report extensive reference was made to: H.T. Reynolds, Analysis of Nominal Data, quantitative applications in the social sciences, 2nd ed. Sage Publications, 1984.
[9] J. Biederman, "Attention deficit/hyperactivity disorder: a selective overview", Journal of Biological Psychiatry 2005; 57, p. 1215-1220.
[10] D.M. Fergusson, et. al, "A longitudinal study of dentine lead levels, intelligence, school performance and behavior: part III, dentine lead levels and attention/activity", Journal of Child Psychology and Psychiatry 1988; 29 (6), pp 811-824.
[11] J.M. Braun, et.al., "Exposure to environmental toxicants and Attention Deficit Hyperactivity Disorder in U.S. children", Environmental Health Perspectives 2006; 114 (12), pp 1904-1909.
[12] S. Pocock, "Environmental lead and children's intelligence: a systematic review of the epidemiological evidence", British Medical Journal 1994; 309, 5 November, pp.
[13] H. Needleman, "Bone lead levels and delinquent behavior", Journal of the American Medical Association (JAMA), 1996; 275(5), pp 363-369.
[14] K.N Dietrich et.al., "Early exposure to lead and juvenile delinquency", Neurotoxicology and Teratology, 2001; 23, pp 511-518.
[15] D.C. Rice, "Behavioral effects of lead: commonalities between experimental and epidemiological data". Environmental Health Perspectives", 104, suppl. 2; 1996.
[16] W.I. Manton, et.al., "Acquisition and retention of lead by young children", Environmental Research; 82 (section A), pp 60-80, 2000.
[17] D.P.H. Laxen et.al., "Children's blood lead and exposure to lead in household dust and water – a basis for an environmental standard for lead in dust"; The Science of the Total Environment, 66 (1987), pp 235-244.
[18] J. Schwartz, D. Otto, "Blood lead, hearing thresholds, and neurobehavioral development in children and youth", Archives of Environmental Health 42(2); 153-160, 1987.
[19] In Studies II and III two questions, # 23 and # 24, seek the same information about speech development. This was part of a stated attempt by the authors to validate information through the use of interlocking questions. In question # 23 in both studies the impression of both parents in the Experimental Group was that the child was slow to acquire speech compared to control. All parents in both studies answered question # 23. In question # 24 however in both studies many data are missing. In Study II this is to such an extent as to make any conclusion based on question # 24 meaningless. In Study III most of the data are present and in support of delayed speech in stutterers.
[20] M. Galvez M.D. et.al. "Childhood Lead Poisoning from Commercially Manufactured French Ceramic Dinnerware: New York City, 2003". Mobidity and Mortality Weekly Report; 53(26); pp 584-585, July 9, 2004.
[21] P.A. Baghurst et.al., "Determinants of blood lead concentrations to age 5 years in a birth cohort study of children living in the lead smelting city of Port Pirie and surrounding areas", Archives of Environmental Health: 47(3); pp 203-210, 1992.
[22] http://www.mgh.harvard.edu/children/adolescenthealth/articles/aa_lead_poisoning.aspx
[23] Wendell Johnson and Associates, The Onset of Stuttering: research findings and implications, University of Minnesota Press, Minneapolis, 1959.
[24] Data from Study I have not been provided in sufficient detail for comment with one exception. The exception is scores from standardized I.Q. tests administered to stutterers and non-stutterers, the only one of the three studies to conduct I.Q. tests in both groups. Despite the fact that researchers sought to empirically match the groups for intelligence, the results showed a lower I.Q. in children who stuttered (median 114; range 80 to 159) than in children who did not (median 116; range 95 to 158). The disparity in perceived intelligence between the groups can also be found in Studies II and III in that it was the impression of parents that their children were not as intelligent as their peers or siblings. It is also consistent with Stuart Pocock's review of the results of I.Q. tests administered to over 2000 lead poisoned children in 7 different published cross-sectional epidemiological studies (British Med. Journal, 309; pp 1189-97; 5 Nov 1994). Although Pocock found a mean deficit of 1 to 2 I.Q. points for roughly every 10ug/dL of blood lead, in some of the studies the deficit ranged much higher, as it did for some of the affected children in Study I.
[25] A full description of Study II has been published as Chapter 4 in Stuttering in Children and Adults: thirty years of research at the University of Iowa, Wendell Johnson, (ed.) University of Minnesota Press, 1955.
[26] H.L. Needleman et.al., "Bone lead levels and delinquent behavior". Journal of the American Medical Association: 275(5); pp 363-369;1996.
[27] D.C. Bellinger, et.al. "Low-level lead exposure, intelligence, and academic achievement: a long-term followup study". Pediatrics, 90(6); pp. 855-861; 1992.

[28] R. Byers, et. al. "Late effects of lead poisoning on mental development". American Journal of Diseases of Children, 66 (5); pp 471-494; 1943.
[29] "Lead in Modern Industry: manufacturer, applications and properties of lead, lead alloys, and lead compounds", Lead Industries Association, NY, NY 1952.
[30] In the scheme put forth in Social Class in America, the book's authors wrote that at first in order to assign social class the "*amount of income and education were used in addition to the four factors already discussed* [that is, occupation, source of income, house type, and dwelling area]. *In the revision…these factors proved unnecessary..and, as this information was more difficult to obtain, they were eliminated*." (chap 2, p. 44) In The Onset of Stuttering, the authors made use of this classification system and wrote that "*the two groups of families* [that is, the Control and Experimental Groups in Studies II and III,] *were matched in socioeconomic status* [using the classification system found in Social Class in America]". The system uses a weighted average of the four factors mentioned with no attempt to match occupations directly. Given that the Iowa researchers failed to achieve balance between the two groups for educational achievement, it suggests that in any social class where members of the class do manual labor, those at an educational disadvantage are more likely to wind up with the dirty dangerous jobs. In the middle part of the last century that meant working with lead more often than not. Further, a lack of education made these workers unprepared to protect themselves (and their families) from hazards arising from occupational lead exposure.
[31] Darley administered standardized social, behavioral, and intelligence tests to some stutterers and non-stutterers in Study II. The tests (see C.R. Rodgers, A Test of Personality Adjustment; Manual of Directions. New York, Association Press, 1931.) included a measure of a child's fantasy life (the 'Daydreaming score'). 25% of tested stutterers and 5% of tested non-stutterers scored "high" on the test (ref 25, p.133). Excessive daydreaming is frequently found in ADHD children. ADHD is a known outcome of lead poisoning.