This is the specific cite proving EDTA binds mercury and it's interaction with brain tubulin. The research below shows mercury chelation must precede EDTA chelation therapy.

There is a vast body of *experience* from chelation doctors that says the opposite. Dr. Garry Gordon of CaEDTA fame and Drs. Mcdonaugh and Cranton of NaEDTA fame have used EDTA for decades without overt or suspected evidence of neurological complications whether amalgams were present or not. My question to these doctors who ignore the presence of mercury in the teeth would involve whether or not their patients who successfully survived cardiovascular deaths, later had Alzheimer's Disease (AD).

While those physicians mentioned above criticize Haley's work because it was done *in vitro* (meaning in the laboratory) as opposed to *in vivo* (meaning in the living body) making his results are invalid. Haley deducted his conclusion by saying:

> After our laboratory demonstrated that tubulin had diminished biological activity in AD brain, and only AD brain, we searched for possible toxicants that might mimic this biological problem. Our finding was simple and straight-forward. After testing numerous heavy metals we observed that only mercury-II cation (Hg^{2+}) could mimic this effect in homogenates of normal brain at concentrations that might be expected to be found under toxic conditions (3,4).

This presents my clinical dilemma. I have an expert with verified scientific research showing the HgEDTA interacts with brain tubulin and actually shows the HgEDTA complex potentiates the effect of mercury.

I have used DMPS for decades with safety and success in lowering the total body burden of mercury. When I first read of Dr. Haley's work, I called him on the phone and we had an extended conversation regarding his findings. It must be realized that much medical scientific work is done in vitro and this is usually scientifically approved. I believe Haley has a valid claim and the last thing I want to save a patient from a cardiovascular event only to have them stricken with Alzheimer's Disease. I have covered the mercury association with AD elsewhere on this site. After you read these two short papers, I think you will understand that there is no dilemma - mercury must be removed from the soft tissue prior to EDTA chelation therapy.

P. Braun, M.D.

http://www.ncbi.nlm.nih.gov/pubmed/8212009

HgEDTA complex inhibits GTP interactions with the E-site of brain beta-tubulin.

E. F. Duhr, J. C. Pendergrass, J. T. Selvin and B. Haley, "HgEDTA Complex Inhibits GTP Interactions with the E-Site of Brain -Tubulin," Toxicology and Applied Pharmacology, 122, 273-288 (1993).

Division of Medicinal Chemistry and Pharmaceutics, College of Pharmacy, University of Kentucky Medical Center, Lexington.

We have found that EDTA and EGTA complexes of Hg2+, which conventional wisdom has assumed are biologically inert, are potentially injurious to the neuronal cytoskeleton. Tubulin, a major protein component of the neuronal cytoskeleton, is the target of multiple toxicants, including many heavy metal ions. Among the mercurials, inorganic mercuric ion (Hg2+) is one of the most potent inhibitors of microtubule polymerization both in vivo and in vitro. In contrast to other heavy metals, the capacity of Hg2+ to inhibit microtubule polymerization or disrupt formed microtubules cannot be prevented by the addition of EDTA and EGTA, both of which bind Hg2+ with very high affinity. To the contrary, the addition of these two chelating agents potentiates Hg2+ inhibition of tubulin polymerization. Results herein show that HgEDTA and HgEGTA inhibit tubulin polymerization by disrupting the interaction of GTP with the E-site of brain beta-tubulin, an obligatory step in the polymerization of tubulin. Both HgEDTA and HgEGTA, but not free Hg2+, prevented binding of [32P]8N3GTP, a photoaffinity nucleotide analog of GTP, to the E-site and displaced bound [32P]8N3GTP at low micromolar concentrations. This complete inhibition of photoinsertion into the E-site occurred in a concentration- and time-dependent fashion and

was specific for Hg2+ complexes of EDTA and EGTA, among the chelating agents tested. Given the ubiquity of Hg2+ in the environment and the widespread use of EDTA in foodstuffs and medicine, these mercury complexes may pose a potentially serious threat to human health and play a role in diseases of the neuronal cytoskeleton.

PMID: 8212009 [PubMed - indexed for MEDLINE]

This was repeated in 1996 by Haley.

Results from the Boyd Haley Laboratory relating to toxic effects of mercury to excerabation of the medical condition classified as Alzeimer's Disease

Research regarding Alzheimer's disease (AD) in our laboratory has been directed towards detecting aberrancy in the nucleotide binding proteins of AD post-mortem brain versus age matched control brain samples. Basic to all of our findings is the following observation. Two very important brain nucleotide binding proteins, tubulin and creatine kinase (CK), show greatly diminished nucleotide binding ability and they are abnormally partitioned into the membrane fraction of AD brain tissue (1,2). What tubulin and CK have in common is that both have a very reactive sulfhydryl in their active sites that, if modified, inhibits their biological activity (14, 15). Mercury has a very high affinity for sulfhydryls and has been proven to be a potent inhibitor of both of these proteins biological activity.

After our laboratory demonstrated that tubulin had diminished biological activity in AD brain, and only AD brain, we searched for possible toxicants that might mimic this biological problem. Our finding was simple and straight-forward. After testing numerous heavy metals we observed that only mercury-II cation (Hg^{2+}) could mimic this effect in homogenates of normal brain at concentrations that might be expected to be found under toxic conditions (3,4). The observation was that Hg^{2+} at 1-5 micromolar levels could selectively and totally abolish the binding activity of tubulin without any noticeable effect on other proteins. This gave a nucleotide binding profile that was identical to that observed in AD brain (4,5,6). Further, recent results in our laboratory have shown that the addition of Hg^{2+} to control brain homogenates not only caused the decrease in nucleotide interaction but could also cause the abnormal partitioning of tubulin into the particulate fraction as observed in AD brain (7). This was especially effected in the presence of other metals (see below).

The next set of experiments was to determine if mercury vapor, the form that escapes from dental amalgams, could mimic the effect in rats exposed to such vapor for various periods of time (5). Rats are different from humans in that their cells can synthesize vitamin C whereas humans have to ingest vitamin C. Vitamin C is thought to be somewhat protective against heavy metal toxicity and other oxidative stresses. However, we observed that the tubulin in the brains of rats exposed to mercury vapor lost between 41 and 75 percent of the nucleotide binding capability demonstrating a Hg^{2+} induced similarity to the aberrance observed in AD brain (5). Consistent with this was a recent report by Dr. Michael Aschner of Wake Forrest University at the 1998 Spring IAOMT meeting. He stated that Western blot analysis of brains of rats exposed to mercury vapor (as above) showed elevated levels of an enzyme called glutamine synthetase (GS) when compared to non-treated controls. This is consistent with a report published from our laboratory in 1992 where we predicted that the elevation of GS in the cerebrospinal fluid of AD patients had potential as a diagnostic marker for AD (12). This potential value of GS as a diagnostic aid for AD was recently confirmed by German scientists (16). We feel that, while mercury has effects on tubulin, CK and GS and that these proteins are proven to be aberrant in AD, this still does not conclusively prove that mercury exposure causes AD. However, it definitely proves that chronic, daily exposure to mercury would exacerbate the clinical conditions of Alzheimer's disease by the ability of low doses of mercury to inhibit enzymes known to be inhibited in AD brain.

We were interested in the genetic research regarding AD and followed this work to see if it correlated to our results. That is, does susceptibility to heavy metal toxicity have any relationship to AD? When we read the correlation of APO-E4 to age of onset of AD we were intrigued enough to look at the primary structure of this protein and its alleles, APO-E2 and APO-E3. In general, the story is this. Individual with

APO-E2 or combinations of APO-E2 and E3 are much less likely to get AD than are individuals who have inherited APO-E4 genes. Also, APO-E2 appears to be more protective than APO-E3 against AD. What is the basic structural difference between these three alleles? Simply, the protective APO-E2 has two sulfhydryls (cysteines) which can bind mercury or other heavy metals that APO-E4 lacks. For example, in APO-E3, one of these cysteines is replaced by an arginine and in APO-E4, both of the cysteines are replaced by arginine. Therefore, lack of protection against AD follows loss of sulfhydryls from APO-E proteins (6). What does APO-E protein do. It is involved in cholesterol transport and all three forms work reasonably well at this. However, APO-E is classified as a "housekeeping protein". That is, in contrast to tubulin and CK which are meant to stay inside of cells where they are synthesized, APO-E is meant to leave the cell carrying out unwanted material for the body to dispose of. In the brain, APO-E protein leaves brain cells and goes into the cerebrospinal fluid (CSF) and then crosses the blood brain barrier into the blood plasma. It is cleared from the blood by processes that dispose of the unwanted material that it is carrying. It is our hypothesis that while APO-E2 or E3 are leaving the brain cells and traversing the CSF they likely bind any heavy metal or other sulfhydryl reactive toxin that may have made it into the central nervous system (6). APO-E4 could not do this and therefore loses the protective parameters that APO-E2 and E3 have. It is interesting to note that the second highest level of APO-E protein is in the CSF that bathes and protects the brain cells.

There was considerable debate concerning whether or not mercury reaches levels in the brain that could be considered toxic. The determination of the levels of mercury toxicity that could cause neurological disease has been done using animals, such as rats, under tightly controlled laboratory conditions where the diet is carefully monitored to exclude other toxicants. However, humans do not live under such restricted conditions and heavy metal imbalances in AD brains have been reported numerous times. For example, lead (Pb) toxicity is not that uncommon in the inter-city environment or for those exposed to leaded gasoline fumes for many years. **The latest research in our laboratory has shown that one can add various metals to human brain homogenates to levels that do not affect nucleotide binding to tubulin yet the very presence of these metal potentiate the toxicity of mercury.** That is, the presence of Zn^{2+} and Cd^{2+}, at non-toxic levels, decrease the amount of Hg^{2+} required for 50% inhibition of tubulin or creatine kinase viability. When we compare the toxicity of Hg^{2+} in brain homogenates as described above (refs. 3 & 4) the addition of 0, 10 and 20 micromolar Zn^{2+} increases the inhibition of GTP binding to tubulin from 4% to 50% and 76%, respectively (7,13). In other words, mercury is much more toxic in the presence of other metals that compete with mercury for the binding sites on protective biomolecules (e.g., APO-E2 & E3, glutathione, metallo-thionine, ect.). This observation probably explains some observations on the toxicity of solutions in which dental amalgams have been soaked. The most recent publication in this area seems to put an end to the argument as to whether or not mercury from dental amalgams pose a threat to the exacerbation of AD. Olivieri et al. demonstrated that exposure of neuroblastoma cells to 36×10^{-9} molar Hg^{2+} caused an increased secretion of ß-amyloid protein 4 to 6 hours later and increased phosphorylation of the microtubulin protein Tau (17). Both of these biochemical changes are uniquely observed in AD brain tissues and ß-amyloid protein makes up the 'amyloid plaques' that are the hallmark diagnostic marker for AD on brain pathology. A very strong component of AD researchers believe that amyloid protein is the cause of AD. Therefore, mercury exposure at nanomolar levels causes neuroblastoma cells to produce a protein that is believed to be involved directly in AD. However, this data supports the initial contention from the Haley laboratory that mercury first inhibits enzymes like tubulin, creatine kinase and glutamine synthetase and dramatically affects metabolism. After these inhibitions occur the cell responds to the cytotoxicity by producing and secreting amyloid protein which forms the amyloid plaques observed on brain pathology and used to substantiate the AD diagnosis. To the point, amyloid plaques are the result of AD, not the cause. The cause is exposure to environmental toxicants like mercury.

Wataha et al. (8) reported that extracts of the amalgam material (trade name, Dispersalloy) "was severely cytotoxic when Zn release was greatest, but less toxic between 48 and 72 hours as Zn release decreased". Zn is an essential metal needed for health and many times recommended by physicians to be taken in supplemental form. It is my opinion that the increased toxicity was not caused by direct Zn toxic effects. Rather, enhanced toxicity was due to the Zn potentiated toxicity of mercury caused by Zn^{2+} occupying biomolecule chelation sites resulting in a higher concentration of free Hg^{2+} capable of inhibiting the activity of critical nucleotide binding proteins such as tubulin and CK. This raises the question if

mercury is released from amalgams under similar conditions. Chew et al. (9) tested the "long term dissolution of mercury from a non-mercury-releasing amlagam (trade name Composil)". Their results demonstrated "that the overall mean release of mercury was 43.5 +/-3.2 micrograms/cm^2/24hr, and the amount of mercury released remained fairly constant during the duration of the experiment (2 years)". In my opinion, this is not an insignificant amount of mercury exposure if one considers the number of years a 70 year old individual living today may have been exposed. In the Haley laboratory we soaked amalgam fillings in distilled water and then tested the resulting solution for toxicity. The results were obvious, the water was now extremely toxic and when added to brain homogenates dramatically inhibited the viability of tubulin and creatine kinase, exactly as observed when we added mercury cation. The bottom line is that mercury toxicity is enhanced by the presence of other heavy metals and both are released from dental amalgams. Additionally, when one considers the toxicity of a certain body level of mercury it is somewhat meaningless unless the body level of other heavy metals is also considered.

Many recent literature and popular press reports state that the presence of periodontal disease raises the risk factor or exacerbates the condition of several other seemingly unrelated diseases such as stroke, low birth weight babies, cardiovascular disease (See October 1996 issue of Periodontology). The anerobic bacteria of periodontal disease produce hydrogen sulfide (H_2S) and methyl thiol (CH_3SH) from cysteine and methionine, respectively. This accounts for the "bad breath" many individuals have. However, in a mouth that produces H_2S, CH_3SH (from periodontal disease) and Hg^o (from amalgam fillings) the very likely production of their reaction products, HgS (mercury sulfide), CH_3S-Hg-Cl (methyl thiol mercury chloride) and CH_3S-Hg-S-CH_3 (Dimethylthiol mercury) has to occur. This is simple, un-refutable chemistry whose presence is supported by easily observable amalgam tattoos. These tattoos are purple gum tissue surrounding certain teeth where the gum and tooth meet and caused by HgS as determined by mercury analysis of such tissue. HgS is one of the most stable forms of mercury compounds and is the mineral form of mercury, called cinnabar, from which mercury is mined from the earth). All of these compounds are classified as extremely toxic and the latter compound, dimethylthiol mercury is very hydrophobic and it solubility similar to dimethyl mercury. Dimethyl mercury was the compound that was recently in the press where only a small amount spilled on the latex gloves of a Dartmouth University professor caused severe medical problems and finally death. Logic implys that anyone with periodonatal disease, anaerobic bacterial infected teeth and mercury containing fillings would be exposed daily to these very toxic compounds. In the Haley laboratory we synthesized the two methylthiol-mercury compounds and tested them. They are extremely cytotoxic at 1 micromolar or less levels and are potent, irreversible inhibitors of a number of important mammalian enzymes, including tubulin and CK.

To determine if toxic teeth could have an effect on the enzymes/proteins of human brain we have done the following study. Several very toxic teeth were incubated for 1 hour in distilled water. Aliquots of these solutions were then added to control human brain homogenates and the resulting samples tested for enzyme viability. The result showed that several of the solutions, but not all, in which toxic teeth had been incubated inhibited the viability of the same enzymes that are found to be inhibited in AD brain. Therefore, depending on the type of anerobic bacterial infection in avital teeth it is possible to have a toxicant production that would exacerbate the condition classified as AD.

In summary, the data on the effects of mercury on the nucleotide binding properties and the abnormal partitioning of two very important brain nucleotide binding proteins first suggested that mercury must be considered as a contributor to the condition classified as AD. This is strongly supported by the recent finds that nanomolar levels of mercury causes neuroblastoma cells to secrete ß-amyloid protein and increase phosphorylation of the microtubulin associated protein Tau, both major biochemical observations related to AD. Consideration of mercury as an exacerbating factor is especially relevant when mercury is present in combination with other heavy metals such as zinc (Zn) cadium (Cd) and lead (Pb). Bluntly, the determination of safe body levels of mercury by using animal data where the animals have not been exposed to other heavy metals is no longer justifiable. Mercury is much more toxic to individuals with other heavy metal exposures. As I have been sent numerous lab reports on levels of elements in the hair and other tissues of suspected mercury toxic patients I have noticed that many have exceedingly high Pb, Cd, Cu, Zn, etc. levels. It is my opinion that one of the major questions left to be answered concerning the toxic effects of mercury is "does the combination of mercury with different heavy metals lead to different clinical observations of toxicity?" There can be little doubt that the elevated

levels of other heavy metals increases the toxicity of mercury. Further, the reaction of oral mercury from amalgams and the reaction of this mercury with toxic thiols produced by periodontal disease bacteria very likely enhances the toxicity of the mercury being released. This makes any claim regarding the determination of safe levels of mercury as obtained under controlled conditions (e.g. in a system where other heavy metals are excluded) very suspect when discussing toxic mercury effects in the uncontrolled environment that humans are exposed to.

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Secretion and Tau Phosphorylation in SHSY5Y Neuroblastoma Cells. J. Neurochemistry 74, 231-231, 2000.

The following are Haley links regarding mercury and disease, particularly ASD.

1. Professor Boyd Haley
   "Dr **Boyd Haley** is a chemist from the University of Kentucky who is obsessed with the terrible dangers of mercury. Because of this, he is a darling of both ..." www.ratbags.com/rsoles/comment/haley.htm
2. Boyd Haley PhD
   "**Boyd Haley**, Ph.D., a biochemist at the University of Kentucky, is probably one of the world's top experts on mercury toxicity. Hear this fascinating review ..." www.whale.to/v/haley.html
3. Letter by **Boyd Haley**, PhD, is in response to an article on the ADA ...
   "**Boyd** E. **Haley**. Professor and Chair. Department of Chemistry. University of Kentucky. REFERENCES:. 1. a. Duhr, E.F., Pendergrass, J. C., Slevin, J.T., ..." www.whale.to/m/haley.html
4. Dr. **Boyd Haley** on Mercury toxicity & Autism: Part 1
   "Dr. **Boyd Haley** is a professor and chair of the chemistry department at the University of Kentucky. In this interview, Dr. **Haley** discusses me...all » Dr. ..." video.google.com/videoplay?docid=4115912987954370615
5. Boyd Haley - Wikipedia, the free encyclopedia
   "**Boyd** E. **Haley**, PhD (b. September 22, 1940, Greensburg, Indiana), is a professor at the University of Kentucky, where he has been the chairman of the ..." en.wikipedia.org/wiki/Boyd_Haley
6. Research Interests of the **Haley** Group
   "**Boyd** E. **Haley**. Professor of Chemistry Bioorganic Chemistry. Office: ASTeCC A057 Phone: (859) 257-2300 x246 FAX: (859) 323-1069 Email: be**haley**@uky.edu ..." www.chem.uky.edu/research/haley/
7. Boyd E. **Haley** Publications
   "**Boyd** E. **Haley**'s Selected Publications. R. R. Drake, R. K. Evans, M. J. Wolf and B. E. **Haley**, "Synthesis and Properties of 5-Azido-UDP-Glucose: Development ..." www.chem.uky.edu/research/haley/cv.html
8. AGE OF AUTISM: **BOYD HALEY** RESPONDS TO CA AUTISM NUMBERS
   "13 Jan 2008 ... Thanks to www.vaccinationnews.com for allowing us to link THIS LETTER by Dr. **Boyd Haley** of the U. of Kentucky Chemistry department. ..." www.ageofautism.com/2008/01/boyd-haley-resp.html
9. AGE OF AUTISM: **BOYD HALEY** LETS 'EM HAVE IT
   "(Editor's Note: **Boyd Haley**, the University of Kentucky chemistry .... Listed below are links to weblogs that reference **BOYD HALEY** LETS 'EM HAVE IT: ..." www.ageofautism.com/2008/03/boyd-haley-lets.html
10. ToxicTeeth.net -- Dr. **Boyd Haley's** Curriculum Vitae
    "Briggs, F. Norman, Al-Jumaily, Walid and **Haley**, **Boyd**. Photoaffinity Labeling of the (Ca+Mg) ATPase of Skeletal and Cardiac Sarcoplasmic Reticulum with ..." www.toxicteeth.org/old_web_site/haley-CV.html
11. Consumers for Dental Choice - working for mercury-free dentistry ...
    "Dr. **Boyd Haley**'s Rebuttal to the ADA: Letter written in response to May 11th response by Robert M. Anderton, D.D.S., J.D., LL.M. and President of the ADA, ..." www.toxicteeth.org/natCamp_ADAresponds_haleyrebuttal.cfm
12. Autism Blog - **Boyd Haley** and Mark Geier: Experts? | Left Brain ...
    "7 Jul 2006 ... Autism Blog - In case you didn't know, as well as vaccines, the mercury militia also think that the thiomersal in RhoGAM given to pregnant ..." leftbrainrightbrain.co.uk/?p=393
13. Respectful Insolence (a.k.a. "Orac Knows"): Salon.com flushes its ...
    "RFK Jr. cites Professor **Boyd Haley**, Chairman of the Department of ... This is the same **Boyd Haley** who got into trouble last year for labeling autism as "Mad ..." oracknows.blogspot.com/2005/06/saloncom-flushes-its-credibility-down.html
14. Haley Boyd, 18 : Obituaries : Kitsap Sun
    "**Haley** Lynne **Boyd**, 18, a resident of Port Orchard, died November 19, 2007 in Port Orchard due to an asthma attack. She was born to N. Denny **Boyd**-Poindexter ..." www3.kitsapsun.com/news/2007/nov/21/haley-boyd-18/
15. VACCINE RISK AWARENESS NETWORK -"INTERVIEW WITH DR. **BOYD HALEY** ON ...
    "Interview with Dr. **Boyd** E. **Haley**: Biomarkers supporting mercury toxicity as the major exacerbator of neurological illness..." www.vran.org/vaccines/mercury/merc-tox.htm
16. VACCINE RISK AWARENESS NETWORK -"Affidavit Of **Boyd** E. **Haley** ...
    "Vaccine Risk Awareness Network-Thimerosal or merthiolate is a derivative of thiolsalicylate where ethyl mercury is attached though the sulfur." www.vran.org/vaccines/mercury/mer-haley.htm
17. 040406 Teri Small w.Boyd Haley
    "Interview with Dr. **Boyd** E. **Haley**: Biomarkers supporting mercury ... I'm pleased to be joined by Dr. **Boyd Haley**. **Boyd Haley**, ..." www.spdalberta.org/Articles/News/boyd_haley_biomarkers2.pdf
18. YouTube - Part 1: Mercury Toxicity & Autism
    "Mercury Toxicity & Autism Part 1Dr. **Boyd Haley** is a professor and chair of the chemistry department at the University of Kentucky." www.youtube.com/watch?v=GQYISvsgq6s
19. YouTube - **Boyd Haley**, PhD Speaks at Mercury-Free Vaccines Rally
    "10 Jun 2007 ... **Boyd Haley**, PhD, Chair of the Dept. of Chemistry at Univ. of Kentucky, speaks about the damage done to a generation of children by mercury ..." www.youtube.com/watch?v=aU47LbCxEnI
20. Tobacco Science and the Thimerosal Scandal
    "Robert F. Kennedy Jr. Telephone Interview with **Boyd Haley**, April 9, 2005. ..... According to **Boyd Haley**, this critical conclusion clearly implicates ..." www.nsasa.org/TobaccoScience.pdf
21. Researcher Refutes ADA in Congressional Testimony - Articles
    "Dr. **Haley** postulated that this could be a potential cause of autism and Alzheimer's disease." I stand by my statement as a sensible concern based on ..." articles.mercola.com/sites/articles/archive/2001/06/09/amalgam-safety.aspx

22. neurodiversity weblog: Unchelated Autistics "Just Lost"???
    "In his December 23 article, Gold Salts Pass A Test, Dan Olmsted quotes **Boyd Haley**'s callous, gratuitous, unsubstantiated statement, "these older kids are ..." neurodiversity.com/weblog/article/66/
23. neurodiversity.com | petition to defend the dignity of autistic ...
    "We have recently become aware of a presentation made by Dr. **Boyd Haley**, Chairman of the Chemistry Department at the University of Kentucky, at the June 2004 ..." www.neurodiversity.com/mothers_for_dignity.html
24. Boyd Haley: Tagging Toxins for Better Health
    "**Boyd Haley**: Tagging Toxins for Better Health. by Jeff Worley. Why do we eat? Simple hunger? Because our favorite foods taste so good? Nope. ..." www.research.uky.edu/odyssey/exclusive/summer03/haley.html
25. Letter from Professor **Boyd Haley** concerning Vaccination News
    "Thank you, **Boyd**. You can't imagine how much it means to have helped you in the extraordinary work you are doing. - Sandy. About Professor **Boyd Haley** ..." www.vaccinationnews.com/Boyd_Haley_letter.htm
26. Vaccination Liberation Information
    "Sandy: I have been working with **Boyd Haley** since September 2000 who testified at the recent Congressional hearing. Your source is incorrect. ..." www.vaclib.org/news/mmrandmercury.htm
27. Boyd Haley, PhD Speaks at Mercury-Free Vaccines Rally on Technorati
    "**Boyd Haley**, PhD, Chair of the Dept. of Chemistry at Univ. of Kentucky, speaks about the damage done to a generation of children by mercury in vaccines at ..." technorati.com/videos/youtube.com%2Fwatch%3Fv%3DaU47LbCxEnI
28. Adventures in Autism: Mark Blaxill and **Boyd Haley** Respond to ...
    "19 Mar 2008 ... Comment from **Boyd Haley**: ...Attached is a letter Mark Blaxill and I prepared and sent to the Pediatrics Journal in response to a Pichichero ..." adventuresinautism.blogspot.com/2008/03/mark-blaxill-and-boyd-haley-respond-to.html
29. Adventures in Autism: USAAA: Q&A with Dr. Boyd Haley
    "28 Oct 2006 ... Dr. **Boyd Haley**: Vaccines are not the only exposure to mercury or thimerosal. Remember merthiolate, or methyl mercury from fish? ..." adventuresinautism.blogspot.com/2006/10/usaaa-qa-with-dr-boyd-haley.html
30. Presentation: **Boyd** E. **Haley**: Immunization Safety Review ...
    "Presentation: **Boyd** E. **Haley**: Immunization Safety Review: Thimerosal." www.iom.edu/?id=7536
31. Immunization Safety Review: Meeting 9: **Haley** Presentation ...
    "Immunization Safety Review: Meeting 9: **Haley** Presentation." www.iom.edu/?id=18420
32. Dr. **Boyd Haley** Exposes the Phony Science on Amalgam "Safety"
    "Dr. **Boyd Haley**, Chair and Professor of the Chemistry Department at the University of Kentucky exposes the phony science that was the basis of NIH, FDA, CDC, ..." www.mercurypoisoned.com/research/phony_science.html
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