

Autism, The Misdiagnosis of Our Future Generations

US Congressional Sub-Committee Hearing

May 6, 2004

Rashid A. Buttar, DO, FAAPM, FACAM, FAAIM
Vice Chairman, American Board of Clinical Metal Toxicology
Visiting Scientist, North Carolina State University

Over the last 15 years, the incidence of Autism has rapidly increased in the industrialized nations with the United States and the United Kingdom having the sharpest rise. A lot of the attention has been given regarding the link between mercury and autism, with mercury being the possible factor underlying the etiology of this condition. The issue of whether mercury plays a role in Autism or other neurodevelopmental disorders has been the subject of long debate and extreme political discourse but the evidence is overwhelmingly obvious to even the simplest of intellects once the data is objectively reviewed.

The prevalence of mercury in our society is endemic in nature. The association of mercury with chronic disease in the US “medical literature” exists but is very anemic. However, when searching under Toxline under the ATSDR (Agency of Toxic Substances and Disease Registry), a division of CDC, one finds all scientific literature which also includes didactic literature, NOT just the “medical literature”. Not surprisingly to advanced researchers and physicians, the association of mercury to chronic diseases is well documented in the didactic scientific literature.

The search for the association between mercury and cardiovascular disease, the number one killer in the industrialized world, revealed 358 scientific papers exemplifying the relationship. The search for the association between mercury and cancer, the number two killer in the industrialized world, revealed 643 scientific papers exemplifying the relationship. Both of these conditions represent 80% cause of all deaths in the industrialized world, according to the WHO (World Health Organization) as published in 1998. But the association of mercury with neurodegenerative diseases is the most significant, with the references numbering 1445.

The inevitable question is how do we get exposed to mercury? The sources surround us, from mercury amalgams in our teeth, to the contamination of our water sources, inhalation of combustion from fossil fuel, fish that we consume, virtually all vaccinations, and via breast milk, just to name a few. So if mercury is so devastating, why is it allowed to be in our flu shots, vaccines, foods, etc.? This is the “million dollar” question, although it should be evident to the well informed the answer will be somewhere along the money trail.

Increased exposure to mercury through thimerosal containing vaccines is one of the most important issues at hand. Thimerosal (also known as Marthiolate) is the common name of a substance known as ethyl mercurithiosalicylic acid. The overburdening knowledge that thimerosal is converted to ethyl mercury (a substance over a thousand times more destructive than inorganic mercury) in less than one minute after being introduced into the

body should give great concern to those appointed to protect the public. Yet, it is virtually ignored. Why is this highly toxic substance still allowed to be a constituent of our vaccines used to inoculate our precious children, our own future generations?

For example, the MSDS on thimerosal from Eli Lilly, documented on their own letter head as far back as July 13, 1991 clearly states that thimerosal is a “product containing a chemical known to the State of California to cause birth defects or other reproductive harm”. Yet Eli Lilly continues to use thimerosal in the manufacturing process for vaccines. However, the vaccine issue must not overshadow the cumulative mercury exposure experienced by the patient during gestation and early infancy. These additional exposures besides the vaccine history include dietary mercury content, dental amalgam fillings which contribute greatly to the maternal mercury load, Rhogam (immunoglobulin) administration to mother during gestation, exposure to combustion of fossil fuels, water contamination, and mercuric compounds used in skin products.

Mercury’s causes damage by various mechanisms which include: competitive and noncompetitive inhibition of enzyme activity by reversibly or irreversibly binding to active sulfur, binding at the sites off and displacing other divalent cations, like magnesium, zinc, copper, and manganese causing a disruption of enzyme systems, disrupting critical electron transfer reactions, and complexing molecules and inducing a change in structure or conformation which causes them to be perceived as foreign by the body’s immune defense and repair system (haptens reactions) resulting in hypersensitivity that can potentiate or exacerbate autoimmune reactions. Mercury alters biological systems because of its affinity for sulfhydryl groups which are functional parts of most enzymes and hormones. Tissues with the highest concentrations of sulfhydryl groups include the brain, nerve tissue, spinal ganglia, anterior pituitary, adrenal medulla, liver, kidney, spleen, lungs heart and intestinal lymph glands.

But most relevant to us for the purposes of this hearing is that mercury has clearly been shown to cause a denudation of the neurofibrils resulting in direct damage to the neuronal cells. In addition, mercury exposure leads to many secondary clinical problems resulting from the aforementioned mechanisms of damage, such as immuno-suppression, allowing for opportunistic infections, allergies, GI dysbiosis, etc. Addressing all other issues in children with Autism is analogous to attempting to put out fires without addressing the cause of the fire itself. The fire will keep re-igniting unless the “spark” is eliminated. It is the elimination of this “spark”, i.e. mercury, for which we now have an easy and effective solution. Along with some supportive therapies, Autism and certain other chronic neurodegenerative diseases such as Alzheimer’s can be fully and permanently reversed if appropriately treated. This is NOT theory. It has already been clinically validated on a repetitive basis.

But first, let us answer the question why some people are affected while others show no manifestations of mercury toxicity, despite living in the same environments. In our case, the discussion will be limited to mercury, which is considered to be the second most toxic metal known to man but this explanation is applicable to most other heavy metals as well. Most individuals exposed to mercury as well as other heavy metals, have the ability to at

least begin the process of eliminating these heavy metal out of their system. But not everyone has this ability and the extent of variability in the ability of an individual to detoxify their systems will determine the severity of the symptoms of toxicity. Slides #10 to #14 show the typical individual who can get rid of mercury with appropriate treatments. Despite having been exposed to severe levels of mercury vapor, this patient named Robin T. was able to detoxify once appropriately treated with DMPS. Her mercury level was almost 22 fold greater or 2200% more than what is considered to be safe but with appropriate treatments, her levels returned to normal and her symptoms of mercury toxicity resolved.

However, patients with impaired detoxification pathways do not show similar results on testing. Their bodies are unable to release the mercury and/or other metals and on testing, the mercury does not appear. The basis of our treatment protocol for children diagnosed with autism was determined by my clinical observation that certain individuals were unable to detoxify mercury like the vast majority of people appear to have the ability to do so. Slides #16 to # 21 show the case of Karen R. who showed no appreciable levels of mercury despite appropriately being “challenged” with DMPS by two different physicians over a year apart. But in Karen R.’s case, she could not detoxify her system effectively despite being treated appropriately with the correct diagnostic methods.

In Karen R’s case, she needed to have persistent treatment for a period of almost 2 years, as seen on slides #16 to #21 but as you will notice, her mercury levels continued to exponentially RISE until her last test which shows the results dramatically drop. What is most interesting is that as the test results revealed an increase in the mercury levels, the patient dramatically began to improve clinically. The reason the levels of mercury actually rose in each subsequent test, is that this testing method only determines how MUCH mercury and/or other metals we are able to remove. As treatment continued, we were effectively able to remove a greater quantity of mercury during each and every treatment.

It is important to note that this patient received treatments every week but the test results were obtained only every 20 weeks. Despite this disparity between treatments and testing, we see a dramatic and steady increase in mercury levels on testing, directly correlated with significant improvements clinically and alleviations of symptoms. In this particular patient, the symptoms for which she presented included glactorhea, ataxia, dysphagia, inability to articulate with a new onset of stuttering, arrhythmia, chest pain, myalgias, arthralgias, hirtuism, cephalgia, insomnia, fatigue, malaise, depression, and anxiety. On presentation, the patient had notified me she had seen 16 other physicians in the previous 5 years and if I could NOT help her, she would “take care” of the problems herself because she could no longer live this way. This patient, Karen D. was 34 years old when she presented to me. The level of mercury measured during each of Karen D.’s tests was inversely proportionate to the amount of mercury remaining in her system.

The answer to the question of why some people are able to effectively release mercury and/or show absolutely no manifestations of mercury toxicity despite having lived in the same exact environments and had the same level of exposure to metals while others are severely affected with serious clinical manifestations, is not as difficult to answer as one

would initially believe when the multiple variables are considered, which include the type of exposure, biological individuality and genetic predisposition. Drs. Michael Godfrey, et al, reported one such variable explaining the variability of individuals in detoxifying mercury in a landmark paper published in the Journal of Alzheimer's Disease in 2003, entitle "Apolipoprotein E Genotyping as a Potential Biomarker for Mercury Neurotoxicity".

Apolipoprotein-E (apo-E) genotyping has been investigated as an indicator of susceptibility to heavy metal (i.e., lead) neurotoxicity. Moreover, the apo-E epsilon 4 allele is a major risk factor for neurodegenerative conditions, including Alzheimer's disease (AD). A theoretical biochemical basis for this risk factor is discussed herein, supported by data from 400 patients with presumptive mercury-related neuro-psychiatric symptoms and in whom apo-E determinations were made. A statistically relevant shift toward the at-risk apo-E ϵ 4 groups was found in the patients (...0 001). The patients possessed a mean of 13.7 dental amalgam fillings and 31.5 amalgam surfaces. This far exceeds the number capable of producing the maximum identified tolerable daily intake of mercury from amalgam. The clinical diagnosis and proof of chronic low-level mercury toxicity has been difficult due to the non-specific nature of the symptoms and signs. Dental amalgam is the greatest source of mercury in the general population and brain, blood and urine mercury levels increase correspondingly with the number of amalgams and amalgam surfaces in the mouth. Confirmation of an elevated body burden of mercury can be made by measuring urinary mercury, after provocation with 2,3, dimercapto-propane sulfonate (DMPS) and this was measured in 150 patients. Apo-E genotyping warrants investigation as a clinically useful biomarker for those at increased risk of neuropathology, including AD, when subjected to long-term mercury exposures. Additionally, when clinical findings suggest adverse effects of chronic mercury exposure, a DMPS urine mercury challenge appears to be a simple, inexpensive procedure that provides objective confirmatory evidence. An opportunity could now exist for primary health practitioners to help identify those at greater risk and possibly forestall subsequent neurological deterioration.

We started treating children with Autism first in 1996. By 1997, we were being referred patients by a pediatric neurologist, who was following a mutual patient and observed significant changes in the child's behavior after implementation of our treatments. However, by the end of 1998, taking care of children with special needs proved more than I wanted to handle. Although we had far better success than the traditional approach, our treatments had not been responsible for "normalizing" any children. The emotional component was also overwhelming, just having to deal with the pain and frustration of the parents of these children. As a result, we stopped accepting new patients with the diagnosis of Autism or any type of developmental delay in early 1999.

On January 25, 1999, my son Abid Azam Ali Buttar was born. By the time he was 15 months old, he was saying "Abu" which means father in Arabic, and a few other words such as "bye bye". But by the age of 18 months, my son had not only failed to progress in his ability to speak, but had also lost the few words he had been saying. At the age of 36 months, he had absolutely no verbal communication except for the one syllable that he would utter, "deh", on a repetitive basis. As he grew older, I began to worry more and more that he was suffering from a developmental delay. He exhibited the same characteristics that so many parents with children that have developmental delays have observed, such as stemming, walking on tip toes, and lack of eye contact. Sometimes I would call to him but his lack of response would convince me there must be something wrong with his hearing. Certain sounds would make him cringe and he would put his hands on his ears to block the obvious discomfort he was experiencing. He would spend hours watching the oscillation of a fan. But through all this, when he would make eye

contact with me, his eyes would say, “I know you can do it Dad”. The expression he would give me, for just an instant, would be that of a father encouraging his son.

The oceans of tears that I cried and the hours that I spent trying to figure out what was happening to my son are no different than that of any other parent in the same situation. The only difference was that I was one of only a 190 doctors through out the US board certified in clinical metal toxicology. And if this was metal related, I should know how to fix this problem. I tested him and re-tested him and tested him again, searching for mercury. Slides # 23 to 27 show the results of my son’s test and how his system showed no appreciable levels of mercury. But the older he became, the more obvious it became that my son was not developing as he was meant to be developing. My son was not meant to be this way and that was the only one thing that I knew for certain.

About the same time while desperately searching for the cause of the same ailment that had afflicted so many of my own patients previously, I had been invited to present a lecture regarding some of our research on IGF-1 and the correlation with cancer. I had notified the conference that I was too busy to present this lecture but when I learned that Dr. Boyd Haley was also scheduled to present at this conference, I changed my schedule and agreed to lecture just so I could meet and discuss my son’s situation with Dr. Haley. That meeting turned out to be one of the key elements which resulted in our development and subsequent current protocol for treating children with autism, autism like spectrum and pervasive developmental delay. My son was the first one who went through this protocol once safety had been established. Dr. Haley told me of a study that had at the time, not yet been published.

Just before the turn of the century, Holmes, Blaxill and Haley did a study assessing the level of mercury measured in the hair of 45 normally developing children versus 94 children with neurodevelopmental delays diagnosed as Autism using DSM IV criteria. The finding showed that the Autistic children had 0.47 parts per million of mercury in their hair where as the normally developing children had 3.63 parts per million, more that 7 times the same level of mercury as the Autistic children. Opponents of the mercury-neurodegeneration camp used this opportunity to state that this study clearly showed that mercury had NOTHING to do with Autism or any other neurodegenerative condition. However, they completely missed the point of the study. For the reader, the conclusion of the study is obvious, and in part, is reproduced below.

“The reduced levels of mercury in the first baby haircut of autistic infants raise clear questions about the detoxification capacity of a subset of infants. Despite hair levels suggesting low exposure, these infants had measured exposures at least equal to control population, suggesting that control infants were able eliminate mercury more effectively. In the case of autistic infants, those in our sample were exposed to higher levels of mercury during gestation, through dental amalgams or Rho D immunoglobulin injections in the mother. The addition of multiple postnatal exposures to mercury in childhood vaccines would have more severe consequences in infants whose detoxification capacity is reduced or who may be closer to a dangerous threshold exposure. In the case of control infants, mercury hair levels were strongly affected by exposure levels, suggesting that detoxification and excretion played an important role in ensuring normal development in children with elevate toxic exposure relative to peers. If reduced overall mercury elimination is related to hair elimination, then autistic infants will retain significantly higher

levels of mercury in tissue, including the brain, than normal infants. In light of the biological plausibility of mercury's role in neurodevelopmental disorders, our study provides further insight into one possible mechanism by which early mercury exposures could increase the risk of autism.”

These findings were published in the International Journal of Toxicology in 2003. Understanding these findings, along with my clinical experience with the case of Karen D. as previously detailed, led me to the conclusion that a more aggressive method of treatment was necessary compared to the DMSA and various other treatments I had to date employed in the attempt to document high levels of mercury in my son, which up to this point, had not been successful. The first two attempts with DMPS as a challenge treatment were unsuccessful, the first due to difficulty catching the urine since Abie was only 2 years old at the time, and the other due to loss of the urine specimen while being delivered to the laboratory. The third try with DMPS, which represented the 6th test we did on my son with all previous tests showing no appreciable levels of mercury, resulted in the findings on slide #29, the results that were reported to me on his 3rd birthday. His mercury level was over 400% that of safe levels. It is important to note that this level was only indicative of what we were able to “elicit or sequester” out of him. His actual levels were far greater.

I started his treatments on his 3rd birthday, using a rudimentary version of the current TD-DMPS (DMPS in a transdermal base) that my partner, Dr. Dean Viktora and I had played around with a few years previously. By the age of 41 months, 5 months after initiating treatment with the TD-DMPS, my son started to speak, with such rapid progression of his speech that his speech therapist was noted to comment how she had never seen such rapid progress in speech in a child before. Today at the age of 5, Abie is far ahead of his peers, learning prayers in a second language, doing large mathematical calculations in his head, playing chess and already reading simple 3 and 4 letter words. His attention span and focus was sufficiently advanced to the point of being accepted as the youngest child into martial arts academy when he was only 4. His vocabulary is as extensive as any 10 year old's, and his sense of humor, power to reason and ability to understand detailed and complex concepts constantly amazes me. This was the preliminary basis for our study that we initiated, which came about as a result of the extraordinary results obtained in the treatment of my son, Abie.

The Autism study consisted of 31 patients with the diagnoses of autism, autism like spectrum, and pervasive developmental delay. Inclusion criteria was simple, including an independent diagnosis of the above mentioned conditions from either a neurologist or pediatrician, and the desire of the parent to try the treatment protocol using TD-DMPS. All patients were enrolled sequentially as they presented to the clinic and only those who did not wish to participate in the TD-DMPS were not included.

All 31 patients were tested for metal toxicity using four different tests: urine metal toxicity and essential minerals, hair metal toxicity and essential minerals, RBC metal toxicity, and fecal metal toxicity, all obtained from Doctor's Data Laboratory. These tests were performed at baseline, and repeated at 2 months, 4 months, 6 months, 8 months, 10 months, 12 months, and then every 4 months thereafter. All 31 patients showed little or no level of mercury on the initial baseline test results. Slide #37 shows an example of a baseline test

result of one participant in the study showing very little mercury. In addition, all study patients had chemistries, CBC with differentials, lipid panels, iron, thyroid profiles and TSH drawn every 60 days. Further specialized testing also included organic acid testing (OAT test) from Great Plains Laboratory and complete diagnostic stool analysis (CDSA) from Doctor's Data Laboratory. If indicated, IgG mediated food allergy testing was also obtained but was not routinely performed.

Compared to the baseline results all 31 patients showed significantly higher levels of mercury as treatment continued. Slide #39 shows significantly higher mercury levels in this same study patient after two months of treatment with the TD-DMPS, with results showing approximately a 350% increase from previous baseline levels. The improvements in the patients in the study correlated with increased yield in measured mercury levels upon subsequent testing. Essentially, what was noted was that as more mercury was eliminated, the more noticeable the clinical improvements and the more dramatic the change in the patient.

The manifestations of this evidence for clinical improvements included many observations but were specifically quantifiable with some patients who had no prior history of speech starting to speak at the age of 6 or 7, sometimes in full sentences. Patients also exhibited substantially improved behavior, reduction and eventual cessation of all stemming behavior, return of full eye contact, and rapid potty training, sometimes in children that were 5 or 6 but had never been successfully potty trained. Additional findings reported by parents included improvement and increase in rate of physical growth increased, as well as the child beginning to follow instructions, becoming affectionate and social with siblings or other children, seeking interaction with others, appropriate in response, and a rapid acceleration of verbal skills. The results in many of these children has been documented on video and other physicians involved with this protocol have been successfully able to reproduce the same results.

DMPS, or dimercaptopropane – 1 sulfonate, is a primary chelator for mercury and arsenic. Slide 42 shows the chemical structure of DMPS. DMPS has pitfalls as well as advantages. The pitfalls include oral dosing which is the usual recommended dosing because it is approximately 50% to 55% absorbed by the gastrointestinal mucosa. As a result of already compromised gastrointestinal function and dysbiosis noted in most of these children, there is also be a decreased absorption of the DMPS when dosed orally, and with the severe gut vacillations prevalent in our society, DMPS by mouth becomes impractical. Most of the children that have taken the DMPS orally for more than 1 week continuously, begin complaining of abdominal pain, cramping and other GI distress. We tried the oral DMPS for almost 6 weeks before eliminating it as a possible therapeutic method. Intravenous methods of application were not an option in children so young, although is the preferred method I have used in my clinical practice for my adult patients with mercury toxicity.

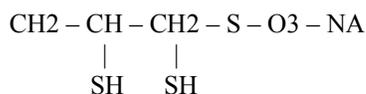
All study patients were also monitored for renal function, and mineral depletion. The key to success with this study was the constant and continuous “pull” of mercury by being able to dose it every other day and the compliance of patient and parents. Each patient was put on a protocol consisting of the transdermal DMPS (TD-DMPS). Transdermal DMPS is

DMPS conjugated with a number of amino acids, delivered in highly specialized micro-encapsulated liposomal phospholipid transdermal base with essential fatty acids. The frequent dosing is one of the most important components of the TD-DMPS. It is important to note that DMPS is highly oxygen reactive and is very unstable when exposed to air. This and many other issues of delivery, stabilization, and oxidation have all been successfully identified and resolved over the last two years with the final result now pending patent. In addition, certain other components have been added to the TD-DMPS to potentiate the efficacy of treatment, such as the addition of various amino acids and glutathione.

There are a number of agents that have been demonstrated to have clinical utility in facilitating the removal of mercury from someone who has demonstrated clinical signs and symptoms of mercury toxicity. The most important part of this systemic elimination process, however, is the removal of the source of mercury. Once this has been completed, treatment for systemic mercury detoxification can begin. The following is a summary of the most effective agent as well as the most commonly used agent that have been documented in the peer-reviewed literature.

A. DMPS

1. The chemical name is Sodium 2,3 dimercaptopropane-1-sulfonate, this water soluble dimercaprol has 2 active sulfhydryl sites that form complexes with heavy metals such as zinc, copper, arsenic, mercury, cadmium, lead sliver, and tin.
2. The chemical structure of DMPS is:



3. DMPS was developed in the 1950's by the Soviets as an antidote for the chemical warfare agent Lewisite.
3. It became commercially available in 1978, being produced by the German pharmaceutical company Heyl.
4. There has been extensive research in both safety and effectiveness of this drug in the 50 years of its existence and it is now considered to be the most effective therapy for the treatment of mercury toxicity, as mercury is bound to sulfur groups throughout the body and is therefore difficult to remove. The sulfur groups on this compound readily unseat the mercury from its attachment to sulfur in our tissues, then this compound is excreted through the kidneys unchanged.
5. DMPS is widely available throughout the United States as a compounded bulk drug and has been recognized by the FDA in that capacity.

6. DMPS is very safe when used properly. Side effects are very rare, but may include allergic reactions such as skin rashes. Most important is to monitor and supplement with appropriate doses of zinc and copper as these minerals are bound readily by DMPS in the same way as it binds mercury. This should be done prior to commencement of any DMPS treatment regimen, then periodically throughout the process.
7. DMPS can be taken orally, as over 50% is absorbed. Most trained chelation physicians in the United States utilize intravenous challenges, whereas most European physicians will challenge with oral DMPS.
8. Currently, the only professional medical organizations that teach and certify physicians in chelation therapy are the International College of Integrative Medicine and the American College for Advancement of Medicine. Both of these organizations periodically conduct workshops on mercury toxicity specifically with emphasis on both basic science knowledge and clinical evaluation and treatment.
9. With the increased concern of mercury toxicity as an environmental health threat and in recognition of the need to increase basic science research and clinical treatment of heavy metal toxicity, the American Board of Clinical Metal Toxicology was recently formed as an evolution of the American Board of Chelation Therapy. This Board will now expand greatly the educational opportunities for physicians interested in this health problem and offer certification procedures that will expand even further the work that has already been done.
10. As a result of the work of these organizations, a general protocol for the use of DMPS has been established which most certified physicians follow.

B. DMSA

1. 2,3 dimercaptosuccinic acid is also a dithiol, like DMPS, and therefore is more effective than EDTA in removing mercury.
2. Structure:

$$\begin{array}{c} \text{HOOC} - \text{C} - \text{C} - \text{COOH} \\ | \quad | \\ \text{SH} \quad \text{SH} \end{array}$$
3. This chelator is an oral agent that is reportedly effective in removing both lead and mercury and is used frequently to treat children.
4. DMSA removes mercury both by way of the kidneys, through urine, and the liver, through bile and then the intestines.
5. DMSA has several disadvantages but also some advantages relative to DMPS:

- a. DMPS remains in the body for a longer time than DMSA, therefore it is able to more thoroughly bind to mercury and eliminate greater amounts per treatment.
 - b. DMPS acts more quickly than DMSA.
 - c. DMPS is given intravenously, intramuscularly, or orally while DMSA is strictly an oral preparation.
6. DMSA is now thought to be potentially harmful if used in patients with excessively high levels of mercury. Therefore, DMSA is recommended for use only late in the mercury elimination process after the peripheral tissue load of mercury has been reduced by DMPS.

In our observation, DMSA did not show efficacy in removing mercury. Slides #26 and #29 show a comparison in the effect of pulling out mercury, completed less than 30 days apart in my son's case. The yield of DMPS compared to DMSA for removal of mercury in this example was 10 to 1. There is an intriguing explanation provided by Boyd Haley, DSc, to support my clinical observations to the lack of efficacy observed with the use of DMSA in treating children with autism and developmental delays. DMSA stands for dimercapto-succinic acid. Succinic acid is a major substrate in the citric acid cycle and DMSA is an analog of succinic acid.

Therefore, DMSA would most likely act as an inhibitor of the enzyme in the citric acid cycle that uses succinic acid as a substrate. This would result in DMSA actually acting as a competitive inhibitor of succinic acid and in turn, would lead to a slowing down of, or inhibition of the citric acid cycle. Succinate produces FADH₂ which is directly coupled to the electron transport chain and leads to ATP production. The competitive inhibition of this succinic acid by DMSA would thus, eventually result in an inhibition of ATP production leading to decreased energy utilization causing a significant burden and impaired ability of the physiological system to function correctly.

In our clinical experience, the only effective method that has resulted in the consistent removal of mercury resulting in the elimination of this "spark" in the pediatric population is the TD-DMPS that was originally formulated only for the purposes of treating my son's developmental delay. Since its implementation, we have now successfully treated scores of patients, many of whom have completely recovered but all of whom have improved since the implementation of this treatment. These results have been duplicated by other physicians involved with the care of patients with neurodegenerative disease processes.

Children with Autism (mercury toxicity) have many resulting imbalances in their systems, including but not limited to significant allergies, systemic candidiasis, hormonal imbalances, gastrointestinal dysbiosis, immune dysfunctions, nutritional deficiencies, etc. However these are what I refer to as the "fires" of autism. All these, and other "fires" of autism result from one "spark". Mercury! Successfully addressing many or all of these "fires" will accomplish transient improvement but until the "spark" that constantly re-ignites these "fires" has definitively been eliminated, any improvement will be short lived at best. Mercury is NOT the fire. It is however, the spark that ignites and constantly re-ignites these "fires". In addition, this particular patient population seems to have antibodies

to mercury binding fibrillarin, confirming the fact that mercury is the cause. But it's the spark, not the fire. Until the spark is eradicated, the fire will continue to re-start and damage the brain and other vital areas such as the immune system. Mercury is the underlying common denominator of all the problems from which these children suffer.

Children diagnosed with autism suffer from acute mercury toxicity secondary to huge exposure while in utero (maternal amalgam load, dietary factors, maternal inoculations, Rhogam injections, etc.) and early on in life (vaccinations preserved with thimerosal, etc.). Adults diagnosed with Alzheimer's suffer from chronic, insidious mercury toxicity secondary to exposure over a long time (amalgam load, inhalation of mercury vapors, combustion of fossil fuels, dietary factors, etc.). By addressing and eliminating the mercury "spark", these secondary "fires" become far easier to manage clinically and the improvements realized from treatment of the resulting imbalances become easier to maintain.

Mercury directly causes damage to the neuronal cell resulting in denudation of the neurofibrils. In addition, mercury results in secondary problems as discussed such as immuno-suppression, allowing for opportunistic infections, allergies, GI dysbiosis, etc. Addressing all other issues such as immuno-suppression in children with Autism without addressing the issue of mercury, is analogous to attempting to put out multiple fires without addressing the arsonist. The fire will keep re-igniting unless the "spark" is eliminated. It is the elimination of this "spark", i.e. mercury, for which we now have an easy and effective solution. Along with some supportive therapies, autism and certain other neurodegenerative diseases can be fully and permanently reversed. This is NOT a theory but rather, a protocol that has already been clinically validated and the evidence is irrefutable.

The reason for some individuals to have severe damage from mercury where others do not have serious adverse neurological deficits extends due to various factors which include biological individuality and genetic predisposition. In addition, what type of toxicity exposure the individual was exposed to, was it inhaled, ingested, or exposed on their skin? What type of mercury exposure did the individual receive? Was it organic or inorganic mercury? If it was organic, was it ethyl mercury or methyl mercury? How frequent was the exposure to the source of toxicity? Was there a significant maternal load present prior to birth? Was the situation exacerbated by the mother being inoculated, or having Rhogam administration. How many administrations took place and over what period of time? What about the diet? How about the proximity to industrial sites, and exposure to combustion of fossil fuel? As you can see, the variables are extensive. But the treatment is essentially the same. The only difference is the extent of continuity of treatment.

Slide 47 shows a newspaper article in the Charlotte Observer with a picture showing one of my patient's mother administering transdermal DMPS to her son's forearms. Slide 48 gives more information on metal toxicity and represents the focus of the majority of my post graduate medical career revolving around the issue of the effective clinical treatment of heavy metal toxicity.

Summary:

The underlying common denominator in chronic neurodegenerative disease seems to be either decreasing vascular supply (less blood to the brain) or accumulation of heavy metals, specifically mercury. The inability of an individual to eliminate toxic metals, especially mercury, is directly related to the level of neurodegeneration experienced. In the young patient population suffering from Autism or Pervasive Developmental Delay, the vascular supply is not an issue. The underlying pathology of children with autism and the geriatric population with Alzheimer's is of the same etiology, specifically mercury toxicity.

Both these patient populations suffer from the inability to excrete mercury as a result of a genetic predisposition resulting from the Apo E allele. This allele appears to be associated with the inability to get rid of mercury from the system. If these patient populations inhabited a complete mercury free environment, they would not have the problems associated with autism or Alzheimer's. When the mercury is successfully removed from their systems, these individuals begin to significantly improve due to a cessation of the destruction and denudation of the neurofibrils, as evidenced by steady improvement in cognitive function.

Mercury is the "spark" that causes the "fires" of Autism as well as Alzheimer's. Autism is the result of high mercury exposure early in life versus Alzheimer's is a chronic accumulation of mercury over a life time. A doctor can treat ALL the "fires" but until the "spark" is removed, there is minimal hope of complete recovery with most improvements being transient at best. However, once the process of mercury removal has been effectively started, the damage is curtailed and full recovery becomes possible and enhanced by utilizing various additional therapies including nutrition, hyperbarics, etc.

Rashid A. Buttar, DO, FAAPM, FACAM, FAAIM
drbuttarclinic@aol.com

Full submission of testimony with supporting data and references to follow.