Chronic Fatigue Syndrome (CFS) and related illnesses: the potential Role of Thimerosal-containing vaccines (TCVs)

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Abstract

Chronic Fatigue Syndrome (CFS), Fibromyalgia Syndrome (FMS), Gulf War Syndrome (GWS) and related illnesses are insidious diseases that are now recognized to affect millions of Americans. Although having been studied for almost twenty years, they are still considered to be disorders of unknown etiology. An exhaustive review of the literature reporting immunological abnormalities and the physical symptoms of these disorders yielded little of significance with respect to the cause of onset of these illnesses.

In an ongoing study, glutathione, a tripeptide necessary for the body's general wellness, was proved to be more successful in improving patient health than antivirals, antidepressants or antibiotics. In the study to be cited, patients were treated with glutathione, both IM and IV, and with the tripeptide as a complex, glutathione•ATP. Patients receiving the glutathione•ATP complex at a dosage of 300 mg/week (IM) showed an 82% improvement in their physical symptoms. Until now, it appeared that a definitive "cure" for CFS was out of the question, with a recovery rate of only 12% being reported.

In 2002, a review authored by T.W. Clarkson entitled, *The Three Modern Faces of Mercury,* provided the needed information to bridge the gap between mercury in the environment and ethylmercury as potentially causing harm to infants and children (autism) and adults later diagnosed with CFS, FMS or GWS.

It is the author's hypothesis that Thimerosal-containing vaccines (TCVs), genetic polymorphism and stress lead to the onset of Chronic Fatigue Syndrome (CFS) and a number of related illnesses. Data and literature are cited to lend support to this hypothesis. Finally, a proposed treatment program for adults suffering from CFS and its related illnesses is presented.

***Keywords**:* Chronic Fatigue Syndrome (CFS), Fibromyalgia Syndrome (FMS), Gulf War Syndrome (GWS), Thimerosal-containing Vaccines (TCVs), genetic polymorphism, molecular mimicry, orthomolecular medicine, micromercurialism.

1. Introduction

In 1995, Chronic Fatigue Syndrome was defined by the Centers for Disease Control and Prevention as a "debilitating disorder characterized by profound tiredness or fatigue. Patients with CFS become exhausted with only light physical exertion". In addition to these defining characteristics, patients generally reported symptoms including "weakness, muscle aches and pains, excessive sleep, malaise, fever, sore throat, night sweats, tender lymph nodes, impaired memory and/or mental concentration, insomnia and depression [1]". Various additional descriptive and diagnostic terms for Chronic Fatigue Syndrome (CFS) or Chronic Fatigue Immune Dysfunction Syndrome (CFIDS) include Fibromyalgia Syndrome (FMS), Gulf War Syndrome (GWS), Chronic Epstein-Barr Virus (EBV), Yeast-related illness (complex) or systemic (Candidiasis); Myalgic Encephalomyelitis (ME), Environmental illness, Hypoglycemia, Non-paralytic Polio and/or Multiple Chemical Sensitivity (MCS). CFS is an insidious disease of unknown etiology, which is now recognized to affect up to 5% of the U.S. population.

Based on a study completed in 1997, U.S. Gulf War Veterans complained of symptoms similar to those of CFS and FMS. Researchers found that 45% of those who went to the Gulf and 15% of those who did not go complained of symptoms including fatigue, cognitive problems, and muscle pain. Of those sent to the Gulf, 6% complained of severe symptoms whereas just 0.7% of those not sent complained of severe symptoms. It was reported that CFS symptoms were between 4 and 16 (mean = 10) times more common in those that went to the Gulf than the civilian population sampled as a comparison [2].

Finally, in a large number of patients treated, a significant percentage had undergone an incredibly stressful situation before becoming ill. Stress reported included recent deaths of close family members, job-related stress, divorce, problems with children or money and the Gulf War [3]. The suggested CFS pathway proposed consists of 5 steps as follows: (1) vaccination with Thimerosal-containing vaccine (TCV); (2) latent period of symptom-free health; (3) extremely stressful period of time; (4) onset of flu-like symptoms/extreme fatigue; and (5) diagnosis of CFS/GWS.

2. Immunological Disorders

Researchers have looked at a variety of immune system anomalies and viral infection possibilities. Since CFS patients exhibit signs of chronic inflammation and/or chronic infection, research has been predominately focused on studies of the immune system and the identification of a potential "infecting agent." In research comparing healthy control patients to CFS patients, the most frequent immunological abnormalities found included chronic T cell activation, decreased Natural Killer (NK) cell function, reduction of the $CD8^+$ suppression cell sub-

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set, differences in subsets of $CD4^+$ cells, cytomegalovirus (HHV-5) and/or HHV-6 infections and increased levels of the antibody to the Epstein-Barr virus early antigen (EBV-EA). A significant decrease in intracellular glutathione concentration, a low selenium (Se) concentration in red blood cells and lower concentrations in white blood cells, specifically lymphocytes, deceased levels of the steroid hormone cortisol and increased levels of the pituitary hormone ACTH [4-6].

Chronic Fatigue Syndrome and its related illnesses appear not to be caused by some as yet unidentified viral, nanobacterial, mycoplasmal or yeast-related infection. Most of the evidence points to the reactivation of latent endogenous viruses at the onset of CFS, rather than new, primary infections [7]. However, if one considers strong case-based data reported in environmental medical literature, it seems reasonable to consider sub-acute mercury poisoning or "micromercurialism" [8] as a potential cause of CFS.

While there are no published controlled studies of mercury level testing in CFS patients, several clinicians have reported that many of their patients have exhibited elevated mercury levels [9-15]. In addition, immune testing has shown significantly elevated hypersensitivity to mercury in a significant number of CFS patients [16-18].

The heightened susceptibility of the nervous system to mercury is well established but little is known about factors that modulate sensitivity to repeated low-dose exposure delivered i.m. (*and*) or restricted to postnatal life [19]. The suggested hypothesis is that adults who have a heritable genetic predisposition leading to idiosyncratic reactions to mercury will present CFS symptoms sometime after receiving a Thimerosal-containing vaccine (TCV). A review of mercury and autoimmune reactions is presented by Pelletier et al. [20]. The physical symptoms of CFS and "micromercurialism" are compared in Table 1.

3. Vaccines and Thimerosal

The preservative in numerous vaccines including Hepatitis B, DTaP and Influenza (HiB) vaccine is Thimerosal, sodium 2-ethylmercurythiosalicylate (CH_3CH_2-Hg-S-C_6H_4-COO^-Na^+). Hypersensitivity or "allergy" to mercury may explain a preponderance of the data collected to date with respect to CFS patients as previously disclosed.

Once in the body, Thimerosal releases ethylmercury via a simple hydrolysis step. Ethylmercury once formed, binds tightly, but reversibly to the surrounding thiol ligands in tissue proteins. Recent studies have determined that there is little difference in the neurotoxicity of ethylmercury and methyl mercury [21]. Of greatest concern is the fact that the brain preferentially absorbs 5 to 7 times more ethylmercury than the blood [22].

4. Methods

In a study conducted by Dr. Patricia Salvato of Houston, the efficacy of glutathione, a tripeptide necessary for the body's general wellness, was evaluated in treating the immunological and physical symptoms of CFS patients (n=478). Of the patients in the study, 327 were treated with glutathione•ATP injections weekly. As seen in Table 2, 118 patients (36%) received a dosage of 100mg/week, 160 patients (49%) received a dosage of 200 mg/week and 49 patients (15%) received a dosage of 300 mg/week. This data are compared to data collected from the previous evaluation of antivirals/immune system stimulants, antidepressants, vitamin/mineral supplements and co-enzyme Q10 in Table 3.

Specimens collected for FIA testing were required to be no more than 24 hours old to optimize isolation and growth of the lymphocytes necessary for the Functional Intracellular Analysis. Spectrox™ Total Antioxidant Function including glutathione, L-cysteine and Lymphocyte Proliferation Index were conducted by SpectraCell Laboratories Inc. of Houston, Texas. Recommendations with respect to antioxidants and minerals to be taken by the patient were presented.

5. Results

The results showed that of the CFS patients receiving glutathione•ATP injections at a dosage of 300 mg/week, 84% reported improvement in symptoms, 86% reported decreased fatigue, 57.1% reported improvement in cognitive ability and 82% reported an overall improvement in functional capacity. Patients on treatment concentrations of 200 mg/week or higher also demonstrated an increase in Natural Killer (NK) cell count and reported relief from the low-grade fever typical of CFS patients. The question to be answered is why the results, although extremely promising, had not essentially provided a cure of CFS patients taking the injections. This group included the author (Table 2) [23].

Life style changes reported as non-pharmacological therapies were also monitored. Patients which experienced extended periods of rest showed an improvement of 86%, stress reduction yielded an 82% improvement and patients experiencing environmental changes showed a 60% improvement. Rest and stress reduction, if one is chronically fatigued, reflects a common sense solution. Typically a 2:1 ratio was instituted wherein two hours of rest were required for one hour of activity. All treatments evaluated are summarized in Table 3.

6. Discussion

6.1 Glutathione

Glutathione (GSH) is a sulfur-containing tripeptide composed of the amino acids cysteine, glutamic acid and glycine as shown in Figure 1. Interestingly, glutathione contains a glutamic acid residue which is an unusual peptide linkage involving its γ-carboxyl rather than the typical α-carboxyl. γ-peptide bonds do not occur in proteins; therefore, these γ-bonded structures protect specialized peptides such as glutathione from proteases [24].

Glutathione plays a number of roles in the body, one of which includes reaction with or neutralization of oxygen-containing free radicals in the body, which can become active during chemical reactions and/or wound healing and ultimately cause cellular damage. GSH also interacts with Vitamin C to reduce it after it has been oxidized in metabolic processes, thereby potentiating the positive effects of the vitamin [25].

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A sometimes overlooked role of GSH is detoxification of heavy metals ($^{No.}M_{wt.}$) in the body. Metals with a molecular weight greater than that of iron ($^{26}Fe_{55}$), such as lead, mercury and cadmium can bind to enzymes that are essential to the body's metabolism and general wellness causing significant stress on the body [26]. The rapid onset of Diabetes Mellitus II, in some CFS patients as example, can be related to mercury's effect on a significant number of carbohydrate metabolism enzymes including those which undertake polysaccharide cleavage and synthesis, disaccharide hexoside and glucuronside metabolism, metabolism of three carbon compounds, reactions with two carbon compounds, acyl activation and transfer, lipases and esterases and enzymes of the citric acid cycle. Some enzymatic interruptions are reversible, but there are more unknowns than knowns in this area [27]. GSH in these cases would be the first line of defense until the intracellular synthesis of glutathione is interrupted itself caused by a significant and measurable drop in the extracellular concentration of L-cysteine [28].

Intramuscular injections of glutathione•ATP have been shown to increase the intracellular levels of glutathione even when compared to glutathione given intravenously. It is theorized that complexing of glutathione with adenosine triphosphate (ATP) enhances the diffusion/transport through the cell wall. It has recently been shown that the distribution of drug compounds in rodents reaches several-logs higher concentration in the organs following intramuscular injections than via oral ingestion [29]. This reported observation is extremely important when considering Thimerosal-containing vaccines, all of which are given intramuscularly.

Methylmercury is known to cross the blood-brain barrier and since recent studies have shown equivalent neurotoxicity when comparing methyl- and ethylmercury [21] therefore, it is not unreasonable to assume both derivatives are transported in the same fashion. In fact, these mercury derivatives are actually carried across the cell membrane and the blood-brain barrier in what is referred to as "an intriguing example of molecular mimicry [8, 30]."

6.2 Molecular Mimicry

Short-chain alkyl mercury compounds easily chelate with L-cysteine, one of the amino acids required for the body to synthesize glutathione. A certain amino acid carrier then mistaking the alkyl mercury/L-cysteine chelate for L-methionine carries the chelate into the tightly packed endothelial cells that comprise the blood-brain barrier. Inside the endothelial cells, glutathione is required to chelate the alkyl mercury compound and said chelate is "pumped" out of the cells on a glutathione carrier [8]. Problems arise in that any cell that will absorb or transport L-methionine from the extracellular matrix will also welcome in the alkyl mercury/L-cysteine chelate (Figure 2).

In the two-step mechanism described, mercury is transported into the cell or through the blood-brain barrier from the blood-side where it can lead to the death/loss of nerve cells, especially in the "pockets" of the cerebrum and cerebellum. Evidence, though limited, suggests that short-chained alkyl mercury complexes, as discussed, can inhibit protein synthesis in the brain at concentrations as low as 2×10^{-5}M. Further, inorganic mercury can potentially build up in the brain if the alkyl mercury compounds discussed are not transported back out across the blood-brain barrier to be excreted [8]. As a result, it may not be unreasonable to assume that the unidentified bright objects (UBOs) seen in the MRI's of CFS patients are small "pencil point" deposits of inorganic mercury [31]. If this is indeed the fact, significant additional work on the neurotoxicology and potential transport mechanisms of inorganic mercury compounds will be required because inorganic mercury is also reported to cause emotional disturbances, tremors and/or fatigue [26:544–5].

The fact that L-cysteine will chelate the alkyl mercury explains why glutathione levels in CFS patients are found to be extremely low if measurable. The body is unable to produce glutathione because of the mercury/L-cysteine chelate. Supplementing with glutathione injections will clearly correct the glutathione deficiency, however, once the glutathione/alkyl mercury complex reaches the extracellular fluid, it finds its way to the liver, gall bladder and through the bile duct into the bowel by which time it has undergone two hydrolysis reactions and the mercury/L-cysteine chelate is available for re-absorption [32]. This perhaps is why CFS patients report significant improvement once placed on glutathione therapy and why glutathione does not provide a cure.

6.3 Enterohepatic Circulation

Because the L-cysteine/alkyl mercury chelate can disassociate in the bowel, a steady-state enterohepatic circulation mechanism is established [30]. In order to excrete the mercury, glutathione therapy must be supplemented with an additional mercury chelant which will shift the steady-state conditions toward excretion. This could be accomplished by supplementing with a non-absorbable thiol chelant taken orally as recommended by Berlin [33] or by using any of several chelating agents such as 2,3-dimercaptosuccinic acid (DMSA), penicillamine (β,β-dimethylcysteine) or N-acetylcysteine (NAC). Attention must be paid to the potential of significant side-effects such as hypersensitivity to penicillin in the case of penicillamine and BAL (British anti-lewisite; 2, 3-dimercaptopropanol) should absolutely be avoided because it forms a lipid-soluble complex with alkyl mercury compounds, redistributing them into the brain where they can cause greater CNS damage [26:816–7]. Slowly excreting mercury out of enterohepatic circulation loop would theoretically begin to improve all the symptoms aforementioned.

Another indicator which has not been reported on is the L-methionine concentrations in CFS patients. L-methionine can convert serine ($HO\text{-}CH_2\text{-}CH\ (NH_2)\text{-}CO_2H$) into cysteine ($HS\text{-}CH_2\text{-}CH\ (NH_2)\text{-}CO_2H$) via trans-sulfuration. Of further interest is the fact that vitamin B_{12} catalyzes the reversion of homocysteine back into L-methionine. If the cysteine concentration is insufficient, the body may attempt to fill the need for cysteine to synthesize glutathione by converting L-methionine into cysteine. Both L-cysteine and L-methionine contain a sulfhydryl group (-SH) which in layman's terms is a mercaptan, named by early chemists because of this group's impressive ability to "capture mercury".

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6.4 The Chronic Nature of the Illness

A major and overlooked problem is that CFS patients tend to continue to poison themselves. It is all but etched in stone that patients with compromised immune systems should be vaccinated annually against each year's variant flu strain. Referring to Table 4, a 1 cc influenza vaccination re-introduces 50 mcg of mercury into a system that is already undergoing a biochemical/immunological catastrophe [34].

A "Thimerosal allergic" patient can be easily identified by conducting a simple but telling skin rash test [35-36]. Further, the literature defines a "Thimerosal allergic" as one who presents a polymorphic gene, glutathione-S-transferase. The latter anomaly should be easy to determine because it is reported to be extremely rare for a patient to exhibit two mutations on a single gene. Westphel et al. [37] reported on "homozygous gene deletions of the glutathione-S-transferases M1and T1". This report in concert with others reporting on "toxic" Thimerosal-related reactions, allergy and sensitization [38,39] causes one to stop and reflect upon just how many could potentially become ill because it is recognized that 50% of the Caucasian population has a gene deletion for the glutathione-S-transferase M1 [26:11–34].

Dr. Bonnie S. Dunbar in testimony before Congress disclosed that there were certain groups of patients of Anglo- descent that had shown a high degree of correlation between autoimmune illness and the Hepatitis B vaccine [40], which contains Thimerosal. Dr. Dunbar was concerned about the recombinant methods used in producing the vaccine but once she reported that a medical student working in her lab went blind after receiving the Hepatitis B vaccine, it became one's opinion that the idiosyncratic reaction seen was clearly the result of a toxic like mercury [26:587].

6.5 Differences in Symptoms Observed in Children and Adults

Although U.S. manufacturers of vaccines are quietly racing to remove Thimerosal from their product(s) given to newborns and infants, the differences observed between that group and adults is explainable. The hepatic excretory system is not fully developed in newborns, which is complicated by the fact that they receive 187.5 micrograms of mercury during their first year through vaccinations—25 micrograms of mercury on their first day of birth—and this appears to be the reason why significantly more neurotoxicity is observed in newborns or infants [41]. The development of the hepatic excretory function in newborns and infants may be promoted by administering a microsomal enzyme inducer [42-44].

Even though symptoms may improve, mercury allowed to remain in the system can potentially interrupt the replication process of RNA and DNA which may explain why birth defects have been seen particularly in the offspring of veterans who may have received up to 200 micrograms of mercury through vaccinations and are suffering from GWS. The determination of mercury clearance should be determined by a "provocative urinalysis" based on a six hour collection period to minimize dilution error [45].

The use of growth hormone secretagogues have already been proven useful in the treatment of adult CFS patients by Salvato and Thompson [46]. In adult growth hormone deficiency, Somatomedin C (IGF-1) may be the therapeutic agent of choice.

Once the mercury levels are lowered, the patient can also be placed on a selenium supplement without fear of redistributing mercury as a lipophilic selenide, bisethylmercury selenide ($[CH_3CH_2Hg]_2$ Se).

Finally, the reports of di- or tetrasodium edetate ($Na_{2\text{-}4}EDTA$), calcium disodium edetate ($CaNa_2EDTA$), [47], doxycycline [48-49] and methotrexate [Rubin R. and Miller RF, unpublished data, 2004] being reported as effective in treating CFS are very reasonable and support the premise that mercury is a causative factor. If one considers the structures of the aforementioned drug compounds, it becomes obvious that each is capable of chelating a heavy metal.

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Table 1: Comparison of symptoms of Chronic Fatigue Syndrome and Micromercurialism

Chronic Fatigue Syndrome	Micromercurialism
Excessive sleep	
Weakness Profound tiredness/fatigue Insomnia	General weakness Fatigue Insomnia Anorexia
Irritable bowel syndrome	Gastrointestinal Disturbances
Migraine headaches	Increased excitability Photophobia
Night sweats	Perfuse sweating
Impaired memory	Memory loss
Depression	Shyness
Chronic low-grade fever	
Sore Throat	Frequent colds

Table 2. Results of treatment with Glutathione (I.V. and I.M.) as compared to Glutathione as an ATP complex

Drug Compound	Concentration Time Frame	Total no. of patients (%)	Improved Karnofsky (%)	Activity Diary / ADL (%)	Symptom Severity Scale (%)	Fatigue Impairment Scale (%)	Cognitive Screening (%)	Functional Capacity (%)	NK Cell No. Temp
Glutathione I.V.	200mg 2/month	27 (53%)	4 (14.8%)	5 (18.5%)	4 (14.8%)	6 (22.2%)	3 (11.1%)	3 (11.1%)	Inc./NC
	400 mg 1/month	24 (47%)	2 (8.3%)	2 (8.3%)	3 (12.5%)	4 (16.6%)	1 (4.1%)	2 (8.3%)	NC
Subtotal		51 (100%)							
Glutathione I.M.	100 mg/week	16 (100%)	1 (6.2%)	3 (18.7%)	2 (12.5%)	1 (6.2%)	0 (0%)	0 (0%)	Inc. / NC
Subtotal		16 (100%)							
Glutathione-ATP I.M.	100 mg / week	118 (36%)	23 (19.5%)	26 (22%)	25 (21.2%)	27 (22.9%)	18 (15.3%)	24 (20.3%)	Inc./ NC
	200 mg /week	160 (49%)	64 (40%)	68 (42.5%)	63 (39.3%)	61 (39.3%)	42 (26.2%)	58 (36.3%)	Inc./Dec.
	300 mg/week	49 (15%)	39 (79.6%)	44 (90.0%)	41 (84%)	42 (86%)	28 (57.1%)	40 (82%)	Inc./Dec.
Subtotal		327 (100%)							
Total Patients		**478**							

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Table 3. Comparative results of benefits reported: select drug compounds vs. vitamins and supplements vs. non-pharmacological treatments

Treatment	Number of patients	Uncertain total (%)	Worse total (%)	No benefit total (%)	Slight benefit total (%)	Major benefit total (%)	Enormous benefit total (%)
Immune System Adjuncts							
Zovirax (Acyclovir)	123	14 (11)	9 (7)	40 (33)	27 (22)	24 (20)	6 (5)
Gamma Globulin i.m.	130	8 (6)	7 (5)	43 (33)	34 (26)	33 (25)	5 (4)
Gamma Globulin i.v.	51	2 (4)	3 (6)	14 (28)	19 (37)	8 (16)	5 (10)
Oral Interferon	98	15 (15)	12 (12)	40 (41)	18 (18)	6 (6)	7 (7)
Kutapressin	61	3 (5)	7 (12)	32 (53)	9 (15)	8 (13)	2 (3)
Ampligen	8	0 (0)	0 (0)	3 (38)	0 (0)	4 (50)	1 (13)
SPV-30 Boxwood Extract	27	5 (29)	0 (0)	3 (18)	2 (12)	2 (12)	15 (56)
Diflucan	43	6 (14)	3 (7)	8 (19)	11 (26)	13 (30)	2 (5)
Allergy Shots	53	6 (11)	6 (11)	10 (19)	12 (23)	12 (23)	7 (13)
Antidepressants/anxiety							
Prozac	92	10 (11)	19 (21)	22 (24)	13 (14)	23 (25)	5 (5)
Zoloft	50	5 (10)	10 (20)	13 (26)	9 (18)	8 (16)	5 (10)
Sinequan	56	2 (4)	15 (27)	10 (18)	16 (29)	11 (20)	2 (4)
Klonopin	57	4 (7)	6 (11)	8(14)	17 (30)	12 (21)	10 (18)
Florinef	5	0 (0)	2 (40)	0 (0)	2 (40)	0 (0)	1 (20)
Orthomolecular Remedies							
DHEA	137	7 (5)	6 (4)	11 (8)	36 (26)	40 (29)	38 (28)
Co-enzyme 10	88	21 (24)	0 (0)	40 (46)	14 (16)	11 (13)	2 (2)
Vitamins/Minerals	159	5 (3)	0 (0)	33 (21)	59 (37)	48 (30)	14 (9)
Dietary Changes	118	15 (13)	1 (1)	19 (16)	42 (36)	31 (26)	10 (9)
Preliminary GSH-ATP i.m.	35	1 (4)	2 (8)	5 (20)	2 (8)	12 (34)	13 (52)
B12 injection 1cc/week	134	13 (10)	6 (5)	38 (28)	48 (36)	21 (16)	8 (6)
B12 injection 3-5cc/week	41	3 (7)	2 (5)	9 (22)	17 (42)	8 (20)	2 (5)
Non-pharmacological Treatments							
Environmental Changes	50	6 (12)	7 (14)	7 (14)	19 (38)	7 (14)	4 (8)
Medical Acknowledgement of CFIDS	151	12 (8)	1 (1)	13 (9)	22 (15)	57 (38)	46 (31)
Stress Reduction	89	5 (6)	0 (0)	11 (12)	23 (26)	36 (41)	14 (16)
Obtaining S.S. Benefits	81	5 (6)	1 (1)	9 (11)	8 (10)	27 (33)	31 (38)
Joining a Support Group	77	0 (0)	2 (3)	14 (18)	23 (30)	29 (38)	9 (12)
Therapy/Counseling	71	2 (3)	1 (1)	15 (21)	16 (23)	27 (38)	10 (14)
Massage Therapy	79	2 (3)	6 (8)	7 (9)	31 (39)	23 (29)	10 (13)
Rest	168	7 (4)	0 (0)	15 (9)	58 (35)	54 (32)	34 (20)

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Table 4. Thimerosal concentration and Mercury (mcg/0.5 cc) per vaccine and manufacturer

Vaccine	Brand Name	Manufacturer	Thimerosal concentration	Mercury (mcg/0.5 cc)	Date Approved or Change Made	Page No. 2003 PDR
Anthrax	Anthrax vaccine	BioPort Corp.	0	0		
DT	All Products		0.01%	25		
Hepatitis A	Havrix	GlaxoSmithKline	0	0		1536
	Vaqta A/P	Merck	0	0	3/29/1996	2105
Hepatitis B	Engerix-B, P.F.	GlaxoSmithKline		<1.0	3/28/2000	1505
	Engerix-B	GlaxoSmithKline	0.005%	12.5	No Longer Produced	
	Recombivax HB, P.F.	Merck	0	0	8/27/1999	2083
	Recombivax HB	Merck	0.005%	12.5		
Influenza	All Products		0.01%	25		
Pneumococcal	Prevnar	Wyeth-Ayerst	0	0	2/17/2000	3455
	Pnu-Imune 23	Wyeth-Ayerst	0.01%	25		3437
	Pneumovax 23	Merck	0	0	1986	2061
Typhoid	Typhoid Ty21a	Vivotif Berna	0	0	04/01/97	2108
Typhoid Fever	Typhim Vi	Pasteur Merieux	0	0	6/1/1995	818
	Typhoid Vaccine	Wyeth-Ayerst	0	0	2/9/1994	
Yellow Fever	YF-VAX	Pasteur Merieux	0	0	5/1/1996	821

Figure 1. Chemical Structure of Glutathione

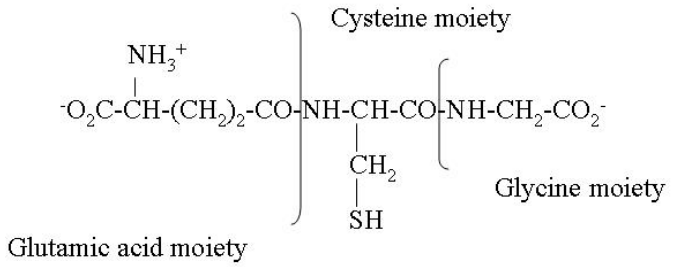
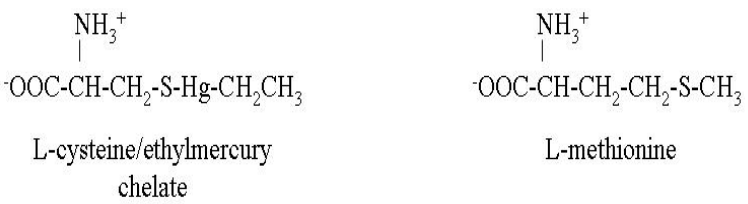


Figure 2. L-cysteine/ethylmercury chelate vs. L-methionine



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