[back] Acetaminophen
[back] Glutathione
[back] Drugs depleting nutrients

Tylenol (Acetaminophen) depletes Glutathione (necessary for removal of mercury)

http://www.wellness.com/blogs/DrLauraMarkham/51/what-every-user-of-acetaminophen-needs-to-know/dr-laura-markham
"The danger is that there isn't much difference between a safe, effective dose, and a toxic dose. Just a doubling of the maximum daily dose can be enough to kill, warns Dr. Anne Larson of the University of Washington Medical Center. The other problem is that if you have no food in your stomach, or if you have alcohol in your system, or worse yet, both, (not relevant for your kids unless they're teenagers, but think about that tylenol you took for your hangover last month), the regular dosage can be toxic because of the overload to the liver."

COMMENT
"Glutathione is found in every one of the trillions of cells in the body. It is most abundant in the liver and then the kidneys. These are the detoxifying organs. NAC N-acetyl cystiene is provided to patients in every hospital emergency situation in the USA for acetaminophen overdose. However NAC has many side effects to the health of the individual, but the alternative is certain death. So it is only administered in emergency room situations."

http://www.mercola.com/2005/may/17/tylenol_risk.htm

"Most experts believe Tylenol causes its damage by depleting glutathione. If you keep your glutathione levels up, the damage from the Tylenol may be largely preventable. Even conventional medicine recognizes this, as anyone who overdoses on Tylenol receives large doses of NAC in the emergency room."

http://www.sciencedaily.com/releases/2002/10/021014072451.htm
"An overdose of acetaminophen can cause depletion of glutathione and land a person in the hospital. "Acetaminophen toxicity is the number one cause of hospital admission for liver failure in the United States," he said. "

http://www.benbest.com/nutrceut/NAC.html
"Glutathione detoxifies acetaminophen, but once glutathione is depleted there can be significant cell death in the liver [THE AMERICAN JOURNAL OF MEDICINE; Flanagan,RJ; 91(Suppl C):131S-139S (1991)]. AIDS victims can suffer severe liver and kidney damage by using acetaminophen or alcohol, which severely deplete glutatione [PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (USA); Herzenberg,LA; 94(5):1967-1972 (1997)]. "

http://www.ssafood.com/site/docsTopicsTips/Nourish%20Your%20Eyes.mht
"Glutathione, another antioxidant, also prevents cataracts. In fact, lenses with cataracts contain one-fifteenth (1/15th) of the normal amount of glutathione and one-tenth (1/10th) the normal level of vitamin C. Glutathione is in asparagus, avocado, broccoli, garlic, onions, spinach, tomatoes, watermelon, eggs and walnuts. Abel advises taking alpha lipoic

acid, N-acetyl cysteine and selenium, which contribute to glutathione production. Abel notes that metabolizing and excreting acetaminophen (Tylenol) depletes glutathione. "Tylenol is probably not the best long-term pain reliever for anyone concerned with eye health." He says. "

GOOGLE ON +glutathione+tylenol

http://www.gotdownsyndrome.net/glutathione&acetaminophen.html

Glutathione & The Acetaminophen (active ingredient in Tylenol) Issue:

Glutathione is commonly deficient in individuals with Down Syndrome, due to the extra chromosome & overexpression of the SOD-1 gene. Glutathione is an important antioxidant. It helps scavenge free radicals, deal with oxidative stress & the pro-oxidant state of individuals withÂ Down Syndrome. Acetaminophen (the active ingredient in Tylenol & many other OTC drugs), depletes Glutathione levels in the liver (where it is made & stored). Therefore, for a person with Down Syndrome, this situation is very important. Since Acetaminophen depletes Glutathione (which is already deficient in DS), it is a bad situation - a key antioxidant is being even more depleted. It can then cause more oxidative damage, free radicals & liver damage. Tylenol is known to possibly cause liver damage & failure. It is best if Tylenol (and drugs containing Acetaminophen) be avoided, but if it is necessary, they can be used at times. When you have to use them, just make sure you give more Glutathione or N-acetyl-cysteine to help out! Below, is alot of info on Glutathione & the Acetaminophen issue (since it is an important issue that comes up fairly often on DS lists):

The following is a list of some of the drugs that contain Acetaminophen:

Actifed Plus

Anacin (all products)

Benadryl (Plus and Plus Nighttime)

Comtrex (all products)

Dristan (all products except Room Vaporizer)

Drixoral Plus

Excedrin (all products)

Nyquil Nighttime Cold Medicine

Pamprin (all products)

Panadol, Children's and Infant's

Percogesic

Sinutab (all products)

Sominex Pain Relief Formula 1 Tablets

TheraFlu (all products)

Tylenol (all products)

Paracetomal

from:
http://www.mdanderson.org/topics/paincontrol/display.cfm?id=34E2C00E-5D6A-44A1-8317170F0DAB8D54&method=displayFull

This article is one of the best articles I have seen on Glutathione. It talks about the numerous things that Glutathione is involved with. It also talks about Acetaminophen & Glutathione. The article also does not talk with too many "big words" & things that are really hard to understand.
http://www.thorne.com/altmedrev/fulltext/glut.html

Here are a couple quotes from the above article about Acetaminophen (with a few notes of mine):

"Many pharmaceutical products are oxidants capable of depleting GSH [my note: Glutathione] from the liver, kidneys, heart, and other tissues.29 The popular over-the-counter drug acetaminophen [my note: active ingredient inÂ Tylenol] is a potent oxidant [my note: it creates oxidation - free radicals]. It depletes GSH from the cells of the liver [my note: which is where GSH is made & stored], and by so doing renders the liver more vulnerable to toxic damage. "

"The consequences of sustained GSH depletion are grim. As cellular GSH is depleted, first individual cells die in those areas most affected [my note: we have lots of cell death already going on in DS]. Then zones of tissue damage begin to appear; those tissues with the highest content of polyunsaturated lipids and/or the most meager antioxidant defenses are generally the most vulnerable. Localized free-radical damage [my note: which is an issue in DS, due to low antioxidant levels & high oxidative stress] spreads across the tissue in an ever-widening, self-propagating wave. If this spreading wave of tissue degeneration is to be halted, the antioxidant defenses must be augmented."

This is an interesting abstract that says that Resveratrol helps against the toxicity done by Acetaminophen. Resveratrol is a potent antioxidant - it is from the skin of grapes. If you have to give Tylenol, it sounds like it'd be very beneficial to give Resveratrol also:

Protective effects of resveratrol against acetaminophen-induced toxicity in mice.
Marmara University, School of Pharmacy, Departments of Pharmacology, Istanbul, Turkey.

This investigation elucidates the role of free radicals in acetaminophen (AA)-induced toxicity and the possible protection by resveratrol (RVT). BALB-c mice were injected with a single dose of 900mg/kg AA to induce

toxicity, while RVT administred in a dose of 30mg/kg i.p. following AA. Mice were sacrificed 4h after AA injection to determine serum ALT, AST and tumor necrosis factor-alpha (TNF-alpha) levels in blood, and glutathione (GSH), malondialdehyde (MDA) levels, myeloperoxidase (MPO) activity and collagen contents in liver tissues. Formation of reactive oxygen species in hepatic tissue samples was monitored by using chemiluminescence (CL) technique with luminol and lucigenin probe. ALT, AST levels and TNF-alpha were increased significantly after AA treatment, and reduced with RVT. AA caused a significant decrease in GSH levels while MDA levels and MPO activity were increased in liver tissues. On the other hand when RVT administered following AA, depletion of GSH and accumulation of MDA and neutrophil infiltration were reversed back to control. Furthermore increased luminol and lucigenin CL levels in the AA group reduced by RVT treatment. Our results implicate that AA causes oxidative damage in hepatic tissues and RVT, by its potent antioxidant effects protects the liver tissue. These data suggest that RVT may be of therapeutic use in preventing hepatic oxidative injury due to AA toxicity.

Someone asked for some articles/abstracts to take to their doctor to show him the importance of Glutathione in Down Syndrome & the issue of Acetaminophen. So, I figured it'd be good to compile a bunch of abstracts & articles that show the AcetaminophenÂ issue, the pro-oxidant state already present in an individual with Down Syndrome & their deficiency of Glutathione. Below is that info:

Abstracts showing elevated oxidative stress, pro-oxidant state & glutathione deficiency in DS -

Multiple evidence for an early age pro-oxidant state in Down Syndrome patients.

Department of Physiology, University of Valencia, E-46010, Valencia, Spain.

Oxidative stress has been associated with Down syndrome (DS) and with its major phenotypic features, such as early ageing. In order to evaluate an in vivo pro-oxidant state, the following analytes were measured in a group of DS patients aged 2 months to 57 years: (a) leukocyte 8-hydroxy-2'-deoxyguanosine (8-OHdG); (b) blood glutathione; (c) plasma levels of: glyoxal (Glx) and methylglyoxal (MGlx); some antioxidants (uric acid, UA, ascorbic acid, AA and Vitamin E), and xanthine oxidase (XO) activity. A significant 1.5-fold increase in 8-OHdG levels was observed in 28 DS patients vs. 63 controls, with a sharper increase in DS patients aged up to 30 years. The GSSG:GSHx100 ratio was significantly higher in young DS patients (< 15 years), in contrast to DS patients aged >/=15 years that showed a significant decrease in the GSSG:GSHx100 ratio ratio vs. controls of the respective age groups. Plasma Glx levels were significantly higher in young DS patients, whereas no significant difference was detected in DS patients aged >/=15 years. Unlike Glx, the plasma levels of MGlx were found to be significantly lower in DS patients vs. controls. A significant increase was observed in plasma levels of UA in DS patients that could be

related to an increased plasma XO activity in DS patients. The plasma concentrations of AA were also increased in young (< 15 years) DS patients, but not in older patients vs. controls in the same age range. The levels of Vitamin E in DS patients did not differ from the values determined in control donors. The evidence for a multiple pro-oxidant state in young DS patients supports the role of oxidative stress in DS phenotype, with relevant distinctions according to patients' ages.

Glutathione metabolism and antioxidant enzymes in children with Down syndrome.

Laboratory of Biochemistry, Molecular Medicine Unit, Children's Hospital and Research Institute Bambino Gesu, Rome, Italy.

Oxidative stress has been proposed as a pathogenic mechanism of atherosclerosis, cell aging, and neurologic disorders in Down syndrome. This study demonstrates a systemic decrease of all glutathione forms, including glutathionyl-hemoglobin, in the blood of children with Down syndrome. Furthermore, we obtained a disequilibrium, in vivo, between the antioxidant enzyme activities.

Diminished glutathione levels cause spontaneous and mitochondria-mediated cell death in neurons from trisomy 16 mice: a model of Down's syndrome.

Institut fur Physiologie der Charite, Humboldt Universitat Berlin, Germany. sebastian.schuchmann@charite.de

It has been suggested that the increased neuronal death in cultures from trisomy 16 (Ts16) mice, a model of Down's syndrome, might result from a diminished concentration of reduced glutathione (GSH). In this study we used microfluorometric techniques to investigate the effect of GSH levels on neuronal survival in diploid and Ts16 cultures. Addition of the GSH precursors cysteine and cystine and the antioxidant tocopherol to the culture medium increased the GSH concentration up to 126.0% in diploid and up to 111.9% in Ts16 neurons. Moreover, we observed a reduced spontaneous neuronal death rate in diploid and Ts16 cultures. Following the application of 50-100 microM glutamate to culture medium, we found a GSH increase in the presence of cysteine, cystine, tocopherol, and cyclosporin A, an inhibitor of mitochondrial permeability transition (diploid, 105.8-110.8%; Ts16, 83.1-96.3%). However, only tocopherol and cyclosporin A had a protective effect on glutamate-induced neuronal death. The results suggest that reduced GSH levels affect the increase of a spontaneous and a mitochondria-mediated, cyclosporin A-sensitive type of neuronal cell death. Therefore, elevating intracellular GSH concentration may have neuroprotective effects in Down's syndrome and Alzheimer's disease.

Glutathione levels and nerve cell loss in hippocampal cultures from trisomy 16 mouse--a model of Down syndrome.

Department of Neurophysiology, Humboldt University, Berlin, Germany.

The tripeptide glutathione (reduced state, GSH) is an important intracellular free radical scavenger protecting cells against oxidative stress. The trisomy 16 mouse is a model of the human trisomy 21 (Down syndrome). Here we demonstrate that cultured hippocampal neurons from trisomy 16 mouse exhibit decreased GSH levels and augmented cell death when compared to diploid cells. Additional lowering of GSH levels led to enhanced cell death in trisomy 16 cells. Based on these results we suggest that a GSH level which is decreased under a specific threshold by increased consumption, reduced synthesis or lack in precursor contributes to cell loss and neurodegeneration in Down syndrome.

Evaluation of superoxide dismutase and glutathione peroxidase enzymes and their cofactors in Egyptian children with Down's syndrome.

Department of Human Genetics, National Research Centre, Cairo, Egypt.

The present work investigated the activity of Cu/Zn superoxide dismutase enzyme (SOD) in red blood cells and glutathione peroxidase enzyme (GPx) in whole blood by spectrophotometric methods. Plasma levels of the cofactors copper and zinc and whole-blood selenium were evaluated using atomic absorption spectrophotometer. The study included a population of 18 Down's syndrome (DS) patients with complete trisomy 21 (group 1), translocations (group 2), and mosaicism (group 3), and their 15 matched controls. The purpose of this work was to study the gene dosage effect of SOD and its consequence on GPx enzyme and the various cofactors, and to find out correlations with developmental fields. Our results showed that in the population with complete trisomy 21 and translocations, SOD and GPx activities were increased, whereas in cases with mosaicism, the enzymes activities were within normal limits. Plasma copper concentrations were increased, whereas whole-blood selenium concentrations were significantly decreased in the three DS groups. Plasma zinc levels were within normal in all patients. We concluded that changes in trace elements and enzyme activities were not related to age or sex. Also, there was no correlation between the enzyme levels and the developmental activities. Our results are useful tools for identifying nutritional status and guiding antioxidant intervention.

Correlations of glutathione peroxidase activity with memory impairment in adults with Down syndrome.

Department of Neuroscience, University of California, San Diego, USA.

BACKGROUND: Down syndrome (DS) is a genetic disorder (trisomy 21 in 96% of cases), associated with an excess of a key enzyme involved with free radical metabolism (FRM), superoxide dismutase-1 (SOD-1), that is encoded by a gene on chromosome 21. Consequently, SOD-1 activity is elevated in DS,

which also occurs in conditions of oxidative stress, and is associated with a compensatory increase in glutathione peroxidase activity (GSHPx). METHODS: This study examined the relationship of memory function with erythrocyte SOD-1, GSHPx and catalase (CAT) activity in 22-51 year old adults with DS. RESULTS: Mean erythrocyte SOD-1 ($p < .02$) and GSHPx ($p < .01$), but not CAT ($p = .76$), activities were significantly greater in the DS group than the controls. In the DS group, erythrocyte GSHPx, but not SOD-1 or CAT activities, was significantly correlated with memory function ($r = .625$, $p < .025$, $df = 13$ for savings score, $r = .631$, $p < .01$, $df = 14$ for intrusion errors) but not with intelligence quotients. CONCLUSIONS: These observations suggest a possible relationship between altered FRM with memory deficits among adults with DS within the age-range in that an age-related increase in the prevalence for Alzheimer's neuropathology is known to be robust before reaching a plateau of 100%.

Glutathione levels and nerve cell loss in hippocampal cultures from trisomy 16 mouse--a model of Down syndrome.
Department of Neurophysiology, Humboldt University, Berlin, Germany.

The tripeptide glutathione (reduced state, GSH) is an important intracellular free radical scavenger protecting cells against oxidative stress. The trisomy 16 mouse is a model of the human trisomy 21 (Down syndrome). Here we demonstrate that cultured hippocampal neurons from trisomy 16 mouse exhibit decreased GSH levels and augmented cell death when compared to diploid cells. Additional lowering of GSH levels led to enhanced cell death in trisomy 16 cells. Based on these results we suggest that a GSH level which is decreased under a specific threshold by increased consumption, reduced synthesis or lack in precursor contributes to cell loss and neurodegeneration in Down syndrome.

Red cell superoxide dismutase, glutathione peroxidase and catalase in Down syndrome patients with and without manifestations of Alzheimer disease.

Department of Obstetrics and Gynaecology, University of Toronto, Mount Sinai Hospital, Canada.

The activities of red blood cell enzymes that scavenge the superoxide radical and hydrogen peroxide were measured in severely to profoundly retarded adult Down syndrome (DS) patients with and without manifestations of Alzheimer disease (AD), and control individuals matched for sex, age, and time of blood sampling. Cu,Zn superoxide dismutase (SOD-1) and glutathione peroxidase (GSHPx) activities were significantly elevated (1.39-fold and 1.24-fold, respectively) in DS individuals without AD. When an adjustment was made for the SOD gene dosage effect, DS patients with AD manifestations had significantly lower SOD levels than the matched control individuals. In contrast, DS patients with and without AD had a similar elevation in GSHPx (an adaptive phenomenon). The mean catalase (CAT) activity was no different in DS and control individuals; however, in a paired regression analysis, DS patients without AD had marginally lower CAT activity than control individuals, whereas DS patients with AD had slightly but not significantly higher CAT activity. Thus, AD manifestations in this DS population are associated with changes in the red cell oxygen scavenging

processes.

Abstracts showing that Acetaminophen depletes Glutathione:

Typing in "Acetaminophen & Glutathione" in PubMed (www.pubmed.com) brings up 54 pages of results - lots of stuff there! Below are just several abstracts I thought were very good.

Full text at: http://www.jbc.org/cgi/content/abstract/M605143200v1

"Acetaminophen overdose is a leading cause of drug related acute liver failure in the United States. Glutathione, a tripeptide antioxidant protects cells against oxidative damage from reactive oxygen species and plays a crucial role in the detoxification of xenobiotics, including acetaminophen."

Acetaminophen Hepatotoxicity: An Update

Acetaminophen is a widely used nonprescription analgesic and antipyretic agent. It is also a dose-related hepatotoxin that can cause fulminant liver failure when taken in massive overdoses or, much less commonly, at therapeutic doses in susceptible individuals. Persons who regularly consume alcohol or persons who have been fasting may be more susceptible to this hepatotoxicity. This liver injury is due not to the drug itself but to the formation of the toxic metabolite N-acetyl-p-benzoquinine imine generated through the cytochrome P-450 drug-metabolizing system. Normally, hepatic stores of glutathione combine with the toxic metabolite and prevent liver cell injury. When glutathione stores are depleted by overproduction of this metabolite, however, the reactive metabolite binds to liver cell proteins and causes hepatic necrosis. P-450 2E1 is induced by alcohol consumption and possibly starvation, and glutathione depletion can occur due to the inadequate nutrition occurring in chronic alcohol use or in starvation. Recent studies have shown that activated Kupffer cells and their secreted toxic agents such as cytokines may also play a role in this liver injury. This liver injury is characterized by extremely high levels of serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (> 1000), and bad prognostic signs include severe prolongation of the prothrombin time, renal dysfunction, and, most importantly, acidosis. N-acetylcysteine is a highly effective antidote when given early (within 15 hours) of overdose. Some patients may develop such fulminant liver injury that they require transplantation. Unfortunately, many such patients have a course so rapid that a donor liver may not become available in time. Thus, both the medical community and the general public require a heightened understanding of this clinical problem in order to initiate prevention measures and to implement early therapeutic measures if an overdose situation occurs.

This is a really good abstract on Acetaminophen, at what dose it depletes Glutathione, over how long it is active, & what to give to stop that depletion (note the bolded part at the end):

Dose-dependent pharmacokinetics of acetaminophen: evidence of glutathione

depletion in humans.

The time course of excretion of acetaminophen and its metabolites in urine was determined in eight healthy adults (seven men and one woman) who ingested 1 gm of the drug and collected timed urine samples for 24 hours. The mean time of peak excretion rate was 1.3 to 3.7 hours for acetaminophen, its glucuronide, sulfate, cysteine, mercapturate, and methoxy metabolites but 13.5 hours for methylthioacetaminophen. The mean half-life of acetaminophen was 3.1 hours and the mean half-life of the metabolites other than methylthioacetaminophen ranged from 4.1 to 5.7 hours. The half-life of methylthiometabolite could not be determined because of its very late peak time. In a second study the effect of dose on the clearance of acetaminophen was determined in nine healthy adult subjects (eight men and one woman) who received doses of 0.5 and 3 gm acetaminophen on separate occasions, separated by 4 to 10 days. The renal clearance of acetaminophen and the formation clearances of the sulfate, glutathione, and catechol metabolites were lower (by 38%, 41%, 35%, and 46%, respectively) at the higher dose. The renal clearance of acetaminophen sulfate and glucuronide conjugates were not different between doses. In a third study (10 men), 10 gm N-acetylcysteine was found to increase the formation clearance of the sulfate conjugate by 27% and that of the glutathione conjugate by 10%. The data suggest that the hepatic supply of reduced glutathione and 3'-phosphoadenosine 5'-phosphosulfate begins to be depleted over the range of 0.5 to 3 gm acetaminophen and that the depletion is overcome by the administration of N-acetylcysteine.

The below abstract is also a very good abstract talking about GSH & different drugs (particularly Acetaminophen):

Therapeutic doses of acetaminophen stimulate the turnover of cysteine and glutathione in man.

In spite of the importance of glutathione (GSH) in the detoxification of toxic metabolites of drugs, virtually nothing is known about the regulation of hepatic GSH homeostasis in man. In order to estimate the turnover of hepatic GSH and to assess the effect of different doses of acetaminophen (paracetamol) on the synthesis of GSH in man, [3H]cystine and varying doses of acetaminophen were administered to healthy volunteers, and the time course of the specific activity of the cysteine moiety of N-acetylcysteinyl-acetaminophen excreted in urine was followed. The fractional rate of turnover of the tracer in N-acetylcysteinyl-acetaminophen increased significantly from 0.031 +/- 0.007 h-1 after doses of acetaminophen ranging from 50 to 300 mg to 0.045 +/- 0.011 and 0.121 +/- 0.027 h-1 following 600 and 1200 mg of acetaminophen, respectively. The data indicate that therapeutic doses of acetaminophen markedly stimulate the rate of turnover of the pool of cysteine available for the synthesis of GSH, most likely due to an increased rate of synthesis of GSH which is required to detoxify the toxic metabolite of acetaminophen. Patients who are not able to respond to a similar demand on their stores of GSH by increasing the synthesis of GSH may be at higher risk of developing hepatic injury from drugs that require GSH for their detoxification.

Paracetamol:

"Conclusions In febrile children, treatment with repeated supratherapeutic doses of paracetamol is associated with reduced antioxidant status and erythrocyte glutathione concentrations. These significant changes may indicate an increased risk for hepatotoxicity and liver damage." Abstract at: http://www.blackwell-synergy.com/doi/abs/10.1046/j.1365-2125.2003.01723.x

Glutathione & Thimerosal Neurotoxicity:

This abstract says that Glutathione can defend against Thimerosal neurotoxicity. This study was done on children who do not have DS, therefore, it would make the case all the more to not give a child with Down Syndrome Acetaminophen if he has been vaccinated recently. Also, it would be beneficial to give a child with Down Syndrome extra Glutathione if they are being vaccinated, to help prevent neurotoxicity from Thimerosal - since their levels ofÂ Glutathione are diminished, they would have a potentally greater risk of having neurotoxicity from Thimerosal if they are not supplemented with Glutathione.

Thimerosal neurotoxicity is associated with glutathione depletion: protection with glutathione precursors.
Department of Pediatrics, University of Arkansas for Medical Sciences and Arkansas Children's Hospital Research Institute, Little Rock, AR 72202, USA. jamesjill@uams.edu

Thimerosol is an antiseptic containing 49.5% ethyl mercury that has been used for years as a preservative in many infant vaccines and in flu vaccines. Environmental methyl mercury has been shown to be highly neurotoxic, especially to the developing brain. Because mercury has a high affinity for thiol (sulfhydryl (-SH)) groups, the thiol-containing antioxidant, glutathione (GSH), provides the major intracellular defense against mercury-induced neurotoxicity. Cultured neuroblastoma cells were found to have lower levels of GSH and increased sensitivity to thimerosol toxicity compared to glioblastoma cells that have higher basal levels of intracellular GSH. Thimerosal-induced cytotoxicity was associated with depletion of intracellular GSH in both cell lines. Pretreatment with 100 microM glutathione ethyl ester or N-acetylcysteine (NAC), but not methionine, resulted in a significant increase in intracellular GSH in both cell types. Further, pretreatment of the cells with glutathione ethyl ester or NAC prevented cytotoxicity with exposure to 15 microM Thimerosal. Although Thimerosal has been recently removed from most children's vaccines, it is still present in flu vaccines given to pregnant women, the elderly, and to children in developing countries. The potential protective effect of GSH or NAC against mercury toxicity warrants further research as possible adjunct therapy to individuals still receiving Thimerosal-containing vaccinations.

Full text at: http://www.icdrc.org/pdf/Neurotoxarticle.pdf

Home