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Established heart disease is a huge public health issue, impacts the quality of life for many individuals and is a significant factor in mortality rates. The prevention of second or repeated events is thus a very important problem. In this issue we review the new guidelines from the American Heart Association with emphasis on recommended medications. The viewpoint used in this discussion involves the number of individuals needed to treat with a given class of medication to prevent one adverse event such as a second heart attack or a stroke or death from any cardiovascular cause. This departs from the standard approach of citing relative risk reductions where the patient is typically told that taking this drug, generally for life, will reduce the risk of some event by 20 or 30%, based typically on 5-year studies. Alternatively, perhaps the relative risk reduction numbers are omitted and "significantly" substituted. Either may introduce a false sense of security or even encourage ignoring more important preventive strategies, even if recommended. However, if patients are told that treating 100 patients will benefit only 2 or 3, they may be inspired to aggressively adopt other approaches. In fact, patients may have trouble understanding these two different ways of describing exactly the same clinical trial results.

In this issue we reopen the debate on fat and heart disease thanks to a new study which examined the substitution of saturated fat with a vegetable seed oil very high in omega-6 polyunsaturated fatty acid. Prior studies, and there are quite a few, generally did not examine the effect of individual components of the PUFAs used with the result that there was serious confounding by the presence of omega-3 PUFAs. The study described avoids this and comes up with some surprising results.

We are now being told by some to avoid fructose, which is difficult since sucrose (table sugar) is a molecule containing one glucose and one fructose molecule joined together but dissociated upon ingestion. In this issue, a study is discussed that provides biological plausibility for avoiding fructose based on effects in the brain resulting in impaired signals such that overeating is rewarded or not discouraged. In other words, fructose appears to be a significant driving force behind obesity and this study adds to previous evidence indicating the merits of avoiding sugar-sweetened soft drinks and fruit drinks and sugar-sweetened foods which are a large part of the diets of many, and especially children and teenagers.

Then a recent study of the association between toxic metal loads and autism in children is reviewed. Autism has been discussed several times in IHN. The take-home message was that there are some physicians accomplishing a cure, but not only is this ignored by mainstream medicine but also the protocol used is highly suggestive of the identity of multiple causative factors which are treatable. Correct them and the child frequently returns to either normal or close to it. The North American rate is inching below 1 child in 10 with autism. When will the national crisis be recognized? At 1 in 8 (and we may be close to that now), one in 7, one in 5? The CDC keeps producing incidence numbers, but connecting the dots and taking the indicated action seems to go against the beliefs of evidence based medicine.

Finally, recent results connecting aspirin and age-related macular degeneration is discussed, as well as a new study which helps neutralize the anti-egg dogma and a study viewed as clinically insignificant by your editor which points to risks of kidney failure if one takes two classes of anti-hypertensive drugs plus a non-steroidal anti-inflammatory.

And finally, if you need to restock your supplements, please remember that by ordering through the on-line vitamin store you will be helping to maintain the web site and the publication of IHN. You can find the store at http://www.yourhealthbase.com/vitamins.htm.

Wishing you and your family good health,

William R. Ware, PhD, Editor

Highlights

MANAGEMENT OF HEART DISEASE. A DISCUSSION OF THE MAINSTREAM VIEW

About one in three adults in the US have some form of cardiovascular disease, more than 13 million have coronary artery disease and 9 million suffer from angina. The financial toll is estimated at $150 billion per year in the US, according to an article on Medpagetoday.com.

The American Heart Association (AHA) has recently published extensive practice guidelines for the management of stable coronary (ischemic) heart disease.[1] This involves secondary prevention of adverse cardiovascular events. The medical therapies discussed include management of lipids, blood pressure and diabetes and recommendations provided regarding antiplatelet therapy, physical activity, weight management, smoking cessation and psychological factors. Drug interventions are justified in terms of relative risk reductions, the standard way clinical results are presented and the approach most influential when discussing benefits with patients. To gain a better insight, we will examine some of the critical underlying evidence in terms of numbers of patients needed to be treated in order to prevent one adverse event which naturally also provides the number treated who do not benefit.

HYPERTENSION

Two trials specifically examined the benefits of blood pressure reduction on cardiovascular events. Both were either restricted to secondary prevention or to diabetics with at least one additional risk factor. The number of patients needed to be treated over 4-5 years to prevent one event (NNT) are given along with the relative risk reduction (RRR).

The HOPE trial. Secondary prevention of heart disease by treating of hypertension[2]

EVENT	NNT	RRR (%)
CVD Mortality	50	26
Heart attack (all)	42	20
Stroke (all)	67	32
All-Cause Mortality	56	16

The EUROPA trial. Secondary prevention of heart disease by treating of hypertension[3]

EVENT	NNT	RRR (%)
Composite*	52	20
CVD mortality	166	14
Non-fatal heart attack	71	22
All-Cause Mortality	125	14

**CVD mortality, heart attack or cardiac arrest*

HOPE involved 13655 subjects with coronary heart disease with a mean age of 60. EUROPA had 9297 subjects age 55 or more. In EUROPA, eighty percent had a history of coronary heart artery disease and 10% had a history of stroke. HOPE had a considerably higher incidence rate of acute events which partly accounts for the smaller NNTs. Both were randomized controlled trials.

BLOOD LIPIDS

The most recent meta-analysis of statin therapy for secondary prevention was published in June 2012.[4] This analysis involved only randomized placebo controlled trials which investigated the prevention of recurrent cardiovascular events. There were gender dependent differences in the risk reductions. In terms of numbers needed to treat over about 5 years, the results were as follows for women (F) and men (M):

Results of a meta-analysis of secondary prevention using statin therapy[4]

EVENT	NNT(F)	RRR (F) %	NNT (M)	RRR (M) %
Any CVD event	34	19	28	18
All-cause mortality	166	9	48	21
CVD mortality	50	27	36	27
Any heart attack	33	31	38	27
Any stroke	200	8	111	19

In general these numbers are smaller than found for primary prevention, and illustrate the rationale of critics of the cholesterol hypothesis for admitting that there may be some merit in secondary prevention, but it is not clear the extent to which it arises from non-lipid lowering effects. The relative risk reduction appears to correlate poorly with the NNT, although they are consistent with the common view that very small RRRs (size effects) are associated with large NNT.

ANTIPLATELET THERAPY (ASPIRIN)

The AHA guidelines also deal with antiplatelet therapy, including aspirin. They state that treatment with 75 or 162 mg daily should be continued indefinitely for the purpose of secondary prevention. Therefore the numbers needed to treat to prevent acute events are of interest. Presented are both primary (1 year) and secondary values (2 years).

Results from pooled studies of aspirin for prevention of CVD events. Source: TheNNT.com

EVENT	PRIMARY NNT	SECONDARY NNT
Any CVD problem	1667	50
All-cause mortality	Infinite	333
Non-fatal heart attack	2000	77
Non-fatal stroke	10,000	200

The results for primary prevention indicate strongly that daily aspirin is not indicated since there is risk of bleeding even with low-dose therapy. For secondary prevention, the numbers suggest the therapy is debatable. TV ads give one the impression that an aspirin a day and one is home free.

These tables illustrate how deceptive the relative risk reduction can be. But they are not to be interpreted as suggesting that the therapy is ineffective. It simply depends on what one considers an effective therapy. However, the outcomes are *at best* such that only about 2 to 3 individuals benefit when 100 are treated and for some events it is considerably less than 1 in 100. Some might feel that this is not good enough. Some might even put it in stronger language or feel that they have been deceived regarding the efficacy when applied to one person rather than a population. The point is that this is the

best in terms of preventive medication that modern medicine has to offer in the context of secondary prevention of adverse CVD/CHD events. When 30, 50, 100 or more must receive treatment to prevent one event, the message seems to be that the treatments are not working very well. The numbers should inspire patients with heart disease not necessarily to abandon conventional treatments but to aggressively seek other interventions such as supplements, diet, exercise and help with psychological stress reduction in order to increase their odds of avoiding a second acute event. Reading the views of Dr. Stephen Sinatra may provide some ideas.[5-7]

There is also the balancing of the benefits of medication against the harms. This is difficult because in the case of industry-sponsored clinical trials there appears, in some cases, to be an understatement of harm or even suppression of adverse results. In post-approval use, reported harmful effect rates are estimated to be only about 1% of the actual number. This is unfortunate since we are talking about the equivalent gambling odds. While many purchase lottery tickets when the odds are terrible, this action carries little risk of side effects unless the practice becomes addictive and health is not on the line.

As regards the harmful effects of statins, in a recent debate with one side titled "Healthy Men Should Not Take Statins" which appeared in the JAMA,[8] the author pointed out the serious shortcomings of studies addressing this issue such as the exclusion during the recruitment or the run-in phase of individuals with comorbidities making them susceptible to side effects, or exclusion of those experiencing adverse results. Also, small randomized trials collect information only on "quantifiable" adverse effects. They point out that observational studies find much higher rates of adverse effects than the 1% to 5% commonly cited. Included are significant risks of moderate or serious liver dysfunction, acute kidney failure, moderate or serious myopathy, diabetes and cataracts. These examples were found in one population studied in Great Britain. In the observational data from the Woman's Health Initiative study there was a 49% adjusted increase in risk of diabetes in healthy women attributed to statins. Finally, he compares the NNT of 50 or 100 to prevent a first MI with the one or more out of a hundred who will develop diabetes and the 20% or more who will experience disabling symptoms including muscle weakness, fatigue and memory loss.

It is well known that statins have a very high rate of early discontinuation of prescribed treatment. Also, the same phenomenon is seen in studies. This implies side effects sufficiently bothersome or worrisome to prompt ignoring the advice that the therapy is necessary and beneficial. For example, fatigue, cognitive problems and muscle pain would no doubt prompt such action. This adds to the suspicion of serious underreporting of adverse side effects.

With regard to blood glucose lowering medications in the context of cardiovascular events in diabetics, the authors of the AHA guidelines recognize the lack of evidence of benefit when normal vs. intensive control is compared and they discuss trials vs. a placebo which were contradictory, but fail to cite the recent meta-analyses suggesting no or marginal benefit (See the December/January 2013 IHN).

POLYUNSATURATED FAT FOR SATURATED FAT AND THE OMEGA-3- OMEGA-6 CONTROVERSY

There is widespread belief that saturated fat (SF) is adversely associated with coronary heart disease (CHD) and cardiovascular disease (CVD). This hypothesis has been incorporated into practice and nutritional guidelines. Specific targets for maximum amounts of SF as a percentage of dietary energy are also a common aspect of guidelines, as is the recommendation to replace saturated fat with polyunsaturated fats. Historically total fat was viewed as the culprit, but when evidence was not forthcoming, the focus shifted to SF. Central to this subject is the syllogism, i.e. SF elevates LDL cholesterol, LDL cholesterol causes heart disease and therefore SF causes heart

disease. This syllogism falls apart when one examines its two critical components. First, there is a remarkably poor correlation between SF intake and LDL cholesterol, especially in the range of low intake to that common in North America. The very small LDL increases can be viewed as clinically insignificant for any given individual. As regards the second component of the syllogism, LDL cholesterol levels are not related to the prevalence or progression of coronary atherosclerosis, independent of age, gender or ethnicity. For asymptomatic individuals, cholesterol is not a significant CHD/CVD risk factor for women, the elderly, or men over about 50 years of age. For younger men, the association may be seriously confounded by issues associated with blood pressure reactivity and stress.

Lowering LDL with statin drugs in asymptomatic individuals has a near negligible effect on acute event risk (approximately 1% absolute risk reduction) which may be due to pleiotropic effects (non-lipid lowering effects), and there is no impact on mortality. Finally, the association between SF and CHD or CVD has come under intense attack in the past two years. The overall conclusion is that there is no significant evidence for concluding that dietary SF is associated with the risk of CHD or CVD. This conclusion was already evident in 1998 but ignored. In addition, many studies find that SF does not appear to be a risk factor for several common cancers, nor with the risk of type 2 diabetes, and if there is a weak association with insulin resistance, it does not appear to carry over to type 2 diabetes risk. See the June 2009 issue of IHN for details, as well as the update in the November 2010 issue.

Nevertheless, the notion that saturated fat is bad is alive and well today and the advice to substitute vegetable oils rich in polyunsaturated fatty acids (PUFAs) for animal fats is still a cornerstone of worldwide dietary guidelines. But there is a big problem in that the general category PUFA comprises multiple species of omega-3 (n-3) and omega-6 (n-6) PUFAs and that each has unique biochemical actions. In a study just reported, Ramsden *et al* have examined the effectiveness of replacing dietary saturated fat with linoleic acid, the most common omega-6 component of PUFAs. At issue is the effect of this intervention on the secondary prevention of coronary heart disease and death.[9] The data they examine was actually from the Sydney Diet Heart Study which was a randomized controlled trial conduced in 1966-1973. This study replaced saturated fat from animal sources, common margarines and shortenings with safflower oil and safflower oil margarine. This oil contains a high percentage of the omega-6 PUFA linoleic acid. Controls received no specific dietary instruction or study foods.

The surprising results were that the intervention group had higher rates of death than the controls (all-cause mortality, 17.6% vs. 11.8%; cardiovascular mortality, 17.2% vs. 11.0%; and coronary heart disease mortality 16.3% vs. 10.1%). The researchers then conducted a meta-analysis of comparable studies which included this new data. The result was a trend toward increased risk of death for coronary heart disease and cardiovascular disease, but the results failed to achieve statistical significance, whereas the results from the Sydney Diet Heart Study given above did. It was concluded that intervention found no cardiovascular benefit when 4 studies were subjected to meta-analysis and there was evidence that in fact it caused an *increase* in mortality from coronary heart disease, cardiovascular causes and all causes.

The authors comment that increasing dietary linoleic acid in place of saturated fatty acids lowers total cholesterol, primarily by reducing LDL. As discussed in the 2009 IHN research review, the changes seen with such interventions which manipulate saturated fat intake are quite small and of the same magnitude as normal LDL variations. They go on to point out that the traditional heart-diet hypothesis holds that lowering LDL will diminish deposition of cholesterol in the arterial wall, slow progression of atherosclerosis, reduce clinical cardiovascular events and improve survival. Serious problems with this hypothesis have been repeatedly been discussed above.

It is important to emphasize that this study focused on not the general question of replacing saturated fat with PUFAs, but specifically with the omega-6 PUFA linoleic acid, the most common dietary member of this class. The authors point out that at the time of their report there was no clinical trial evidence

relating to issues of the risk of cardiovascular risk or death involving replacing saturated fats with linoleic acid without concurrent increasing omega-3 PUFAs, and this confuses the issue because of the apparent benefits of the latter. This study eliminated 0mega-3 confounding.

For decades, the conventional wisdom has been that the n-3 fatty acids were anti-inflammatory whereas the n-6 acids were inflammatory. Willett and coworkers have argued that adequate amounts of both n-6 and n-3 fatty acids are essential for good health and low rates of cardiovascular disease and type 2 diabetes. Furthermore, the n-6 fatty acids do not inhibit the anti-inflammatory effects of the n-3 fatty acids and the dietary combination of both fatty acids is associated with the lowest levels of inflammation.[10,11] Thus there are some outstanding issues.

It is also interesting that in the study discussed above, Ramsden *et al* found that in fact "*the reduction in saturated fat was not significantly related to any mortality outcome*." This was not mentioned in the abstract and the italics have been added. All of this is brings to mind a recent book by Uffe Ravnskov, MD, PhD, having the heretical title *Fat and Cholesterol are Good for You!* A good balance in all the fat family members appears to be an approach worth considering.

CONNECTING FRUCTOSE CONSUMPTION AND OBESITY

Fructose and glucose are both so-called monosaccharaides and are sugars of similar molecular structure. However, they are metabolized differently. Compared to glucose, fructose only very weakly stimulates insulin secretion, and insulin acts in the brain to increase satiety and blunt the reward value of food. Fructose also attenuates increases in circulating levels of satiety hormones and does not attenuate levels of an appetite stimulating hormones. Thus high fructose diets are thought to facilitate weight gain and insulin resistance and increased sugar intake has been implicated in the obesity epidemic.

There are three sources of fructose, common table sugar (sucrose) is a molecule composed 1:1 of fructose and glucose and digestion produces the free sugars. High fructose corn syrup (HFCS) contains roughly a 50-50 mix of free glucose and fructose and the slight excess of fructose does not appear relevant. Finally, fruit sugar is fructose. Fructose consumption and obesity are increasing in parallel. While some like to point to HFCS, the issue is really simply sugar consumption from any source, and the Western diet is very high in sugar from many sources, both obvious and hidden. High sugar consumption from either sucrose or HFCS implies high fructose consumption. Thus the interest in its metabolism and its influence on satiety and overeating.

A recent study has enhanced the biological plausibility to the postulated dangers associated with fructose.[12] Functional MRI as well as a technique called arterial spin labelling were used to map out the regional cerebral blood flow (CBF) response to either glucose or fructose and the impact on functional connectivity between the various sections of the brain.

It was found that glucose produced a reduction in CBF within the thalamus, striatum and other regions of the brain that act together to read the metabolic status on an individual and control motivation and reward. A different response to fructose was observed, where fructose failed to diminish the desirable hypothalamic activity but rather induced small transient increase in activity in this region. Furthermore, fructose did not result in deactivation of the striatum. Hypothalamic and striatal deactivation occur when the initially hungry individual reaches satiety. Also, fructose ingestion reduced CBF in the region called the hippocampus, a region that influences emotional food intake response and inhibits appetite.

The changes in CBF acted to change the inter-regional connectivity. Glucose consumption increased functional connectivity between the hypothalamus and the thalamus and the striatum. Fructose also increased connectivity between the hypothalamus and the thalamus, but not to the striatum. The net

result of these and other differential changes observed between glucose and fructose in changing CBF, regional activity and connectivity had an effect on the appetite and reward regions of the brain such that glucose supported the feedback system which involves satiety and appetite whereas fructose did not and as a result fructose acted to contribute to the stimulation of overeating.

Glucose alone is not a satisfactory food and beverage sweetener since it is not sweet enough. Sucrose and HFCS are the common sweeteners and of necessity involve fructose. Thus this study adds biological plausibility to the beneficial action of limiting the intake of either sucrose or HFCS or both. Artificial sweeteners have the potential for adverse effects, some very serious. There appear to be inadequate long-term safety studies of artificial sweeteners, even for those considered natural such as stevia. There are very serious issues with aspartame due to the pathway involving methanol leading to formaldehyde, the latter being highly toxic and carcinogenic. The aspartame problem was discussed in detail in the July/August 2012 issue of IHN.

This study and other evidence implicate fructose in the obesity epidemic. This is of particular interest in connection with juvenile obesity, since that age group is notorious for heavy consumption of sugar- sweetened beverages and sugar-sweetened foods. But the justification for severe limitation of sugar intake involves a much broader area of health concerns for all age groups, especially in the context of diabetes and cancer. The radical view that sugar is a toxic substance was discussed in the March 2013 IHN. Sugar permeates the food system. Sucrose and HFCS are added to sodas, energy drinks and sports drinks as well as to juice drinks, the latter consumed not only by adolescents and adults but by infants and toddlers. Added sugar can be found in snacks of all sorts, meats, sauces, baked goods, and many other foods consumed by all age groups. The economic issues are huge and involve farm subsidies, healthcare costs, and if the general public started to significantly limit sugar consumption, it would send shock waves across the entire industry.

NEWS BRIEFS

ASPIRIN USE AND AGE-RELATED MACULAR DEGENERATION

Two studies have just appeared which associate risk of age-related macular degeneration (AMD) with regular use of aspirin. This is relevant to the above discussion where the AHA recommends regular aspirin use for secondary prevention of acute cardiovascular events.

The first study by Liew *et al* followed approximately 2400 subjects for a 15-year period.[13] Aspirin use was determined by a questionnaire and AMD graded by retinal photographs. At issue was the prevalence of wet AMD also called neovascular AMD. Regular aspirin use was defined as ≥ once a week, occasional < once a week and non-regular group comprised occasional and non-users. When non-regular users were compared with regular users, the number of regular users needed to produce one case of wet ADM was 91 for 5 years of use, 19 for 10 years, and 9 for 15 years. When the reference was non-users, at 15 years, the odds ratios for occasional and regular users were 1.26 and 2.46, but only the latter was statistically significance although the trend was. The odds ratios were adjusted for a number of variables considered confounders.

The second study was cross-sectional in nature (a snapshot of a group where cases were compared to non-cases).[14] There were 96 cases with wet AMD which were compared with 2035 non-cases, all with full data on confounders. The odds ratio for daily use was 2.26 adjusted for all potential confounders and was statistically significant, whereas use for at least once a week but less than daily, the ratio was 1.30 and not statistically significant. No information was available regarding history of use. For early AMD, there was no significant association with aspirin use.

These two studies taken together suggest serious implications for millions of individuals using aspirin therapy. These studies come after others which have examined this question with inconsistent findings.[13]

EGG CONSUMPTION, GOOD OR BAD?

Shortly after the birth of the hypothesis that dietary fat and dietary cholesterol were bad, eggs were demonized, characterized as a toxic food, and true believers took to eating only the egg white. The reason—an egg contains about 200 mg of cholesterol. This was a disaster for the egg industry since egg whites were not that popular, and also unfortunate for the general public since eggs are an inexpensive and low calorie source of important nutrients, including proteins, unsaturated fatty acids and minerals. As most readers know, it turned out that fat was not bad in the context of cardiovascular disease according to many studies up to the present, all of which seem to be ignored, and most humans can eat quit a lot of cholesterol without any impact on their blood levels.

The latest study just-published employed a meta-analysis (study of pooled, weighted studies) in order to address the association between egg consumption and the risk of coronary heart disease and stroke.[15] The studies involved were prospective, i.e. cohort follow-up studies, and dose response was an issue. The meta-analysis included eight published studies with 17 reports (nine for heart disease and eight for stroke). They involved over 3 million person years follow-up for heart disease and 4 million person-years for stroke.

The results are simple to relate. Higher consumption of eggs (up to one per day) was not associated with increased risk of coronary heart disease or stroke. Dose response showed no increase in risk with increased consumption, although there was no data for greater than 10 eggs/week for heart disease and 7 eggs per week for stroke.

Those who hold fast to the cholesterol fat is bad hypothesis will no doubt now want studies covering wider range of egg consumption, and might even point out that one egg a day meets the Americans Heart Association guidelines for cholesterol consumption. However, it must also be pointed out that many individuals can eat two or more eggs a day with no impact on their blood cholesterol levels, and the ideal study would include both 2-3 eggs per day and serum monitoring. The endpoints would have to be clinical and large cohorts needed to obtain meaningful results. It is clear that this will probably never happen.

Uffe Ravnskov, MD, PhD, describes an experiment he conducted on a sceptical Swedish doctor (himself), where he measured blood cholesterol levels after eating from zero to 8 eggs per day. There was no trend at all.[16] There are a number of similar studies which reveal that responders represent only a small percentage of random populations and that for most, the level of blood cholesterol is independent of the dietary consumption over a significant range. Therefore the results of the meta-analysis should not come as a surprise. For the small minority who experience significant cholesterol elevation with dietary consumption, we are back to the problem that serum cholesterol is a very weak risk factor in CVD evens and LDL is not involved in coronary atherosclerosis.

KIDNEY FAILURE FROM COMBINING BLOOD PRESSURE AND ANTI-INFLAMMATORY DRUGS

A study just published in the *British Medical Journal* has found an increased risk of acute kidney injury (kidney failure) associated with a common practice of combining blood pressure drugs and non-steroidal anti-inflammatory medications (NSAIDs).[17] As one ages, what seems to be inevitable is polypharmacy which may include these two classes of medication, and while NSAIDs are known to be implicated in acute kidney injury, there is limited information about drug interactions.

This study used data from the UK contributed by general practices (a database of longitudinal records from primary care) combined with a national database of hospital episodes. Data was assembled for patients who received anti-hypertensives between January 1997 and December 2008. Patients were then “followed” looking for the first hospital admission for acute kidney injury or initiation of dialysis. Up to 10 controls were randomly selected and matched to each case. For the purposes of the

analysis, they defined double therapy as concurrent use of either, diuretics, angiotensin converting enzyme inhibitors (ACE inhibitors), or angiotensin receptor blockers (ARBs) with NSAIDs. Triple therapy involved concurrent use of drugs from both classes of anti-hypertensives plus NSAIDs.

It was found that only the use of triple therapy increased the risk of acute kidney injury, and this was by a factor of about 1.3 (a risk ratio). However, the incidence rate for hospitalization for acute kidney injury is given by the authors as 5 per 10,000 UK residents. Numbers needed to treat to harm one individual can be derived from these case control studies, but the population incidence is sufficient to estimate the magnitude which is in the order of 1 in 1500.[18] This number seems so big that the actual magnitude of the increased risk is irrelevant and the refined calculation unnecessary. Thus, the issue raised by this study does not seem clinically important or suggest significant concern is justified.

TOXIC METALS AND AUTISM

Studies suggest that children with autism have decreased ability to excrete toxic metals, partly due to low levels of glutathione and the impact of antibiotic use on gut flora. A new study compared the toxic metal levels in normal and autistic children. Analysis involved levels in red blood cells, whole blood and urine.[19] It was found that compared to controls, autistic children had higher levels of lead in the red blood cells and higher urinary levels of lead, thallium, tin and tungsten. When a statistical analysis examined the association of metals with the severity of autism measured by three different scales, it was found that cadmium and mercury were consistently and strongly associated with severity.

As discussed recently in IHN (November, 2012), an essential aspect of the protocol used by the rare physician who attempts to actually cure autism is the removal of toxic metals. An oral chelator, called DMSA for short, is used which is actually sold over the counter (and internet). The safety and efficacy of oral DMSA was already well established in 2009 and reported in two papers in *BMC Clinical Pharmacology*.[20,21] This chelation therapy was also found to normalize red blood cell glutathione in almost all cases, to greatly improve abnormal platelet counts and was also found to decrease inflammation. All this happened with just three days treatment. Details can be found in *Children with Starving Brains* by Jacquelyn McCandless, M.D.[22] Unfortunately, the approach to autism which intends to cure involves dealing with much more than toxic metal load. McCandless describes in detail those parts of the protocol which can be done without active supervision by a physician, but the chances of overall success generally depend on addressing issues which can only be handled by a physician. Furthermore, the order in which these issues are addressed turns out to be critical. DMSA may be an option for anyone concerned about toxic metal overload.

Dr. Robert Jay Rowen reports in his February 2013 newsletter *Second Opinion* of having great success in mercury detoxification with 1 g vitamin C, 200 micrograms of selenium, 300 mg of alpha lipoic acid and 1 g of N-acetyl cysteine split between two doses daily. These are the main active ingredients in Rowen's recommended Advanced Detox Formula. To this is added a clove of garlic. Rowen determines load and progress with a urine test done after a challenge with a chelating agent taken immediately after the first morning voiding. The treatment duration depends on the mercury load. Measured in micrograms of mercury per gram of creatinine, his target is 3, and he sees initial numbers as high as 60! One to three rounds of treatment lasting three months were found to be required.

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