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This issue features hypothyroidism, a disorder associated with low levels of one or more thyroid hormones. A number of causes have been identified, and some are associated with micronutrient deficiencies. This brings up a general problem. How widespread are deficiencies of vitamins, minerals and other essential chemicals not made by human biochemistry? Conventional wisdom informs us that if we eat a so-called balanced diet, the darling of nutritionists, we will be just fine and should not waste energy worrying about being deficient or any money on nutritional supplements. Those who disagree are regarded with disdain by the card-carrying experts and are regarded as "health nuts" or "health fanatics", or as individuals who have fallen under the influence of junk science and quacks.

Thyroid problems are a good example. Iodine deficiency is ignored even though it is clear that iodized salt is only a patch-job and we are being told to restrict salt, which includes iodine enriched salt. Integrative physicians who pay attention to this deficiency find it prevalent among their patients. They also find hypothyroidism very much under diagnosed. Along with iodine, selenium is necessary for the synthesis of thyroid hormones. In areas where there are iodine deficiencies, there frequently are selenium deficiencies.

There are many other examples that could be cited. It has taken decades for vitamin D deficiency to be reluctantly recognized as a possibility. Scientists who study the role of magnesium in human biochemistry are distressed at how common a deficiency of this mineral is in the general public. Potassium and vitamin K deserve mention. Vitamin C is a classic example. While salt is demonized, low levels in fact can cause big problems.

What does not appear to be appreciated is that many micronutrients are cofactors for the thousands of enzyme-catalyzed human biochemical processes and that in addition there are large variations in individual levels where deficiency become pathological. Thus, multiple deficiencies can produce multiple but frequently subtle disorders that represent the dysfunction of endogenous vital chemistry. The problem of individuality in optimum levels and synergistic associations between micronutrients cannot be ignored. A significant problem involves reasoning from clinical presentations to multiple deficiencies and thus discovering a potential route to a cure. This problem is aggravated by the pharmaceutical orientation and the profound suspicion of supplementation ingrained as dogma in modern medicine.

The failed approach to the diagnosis of thyroid disorders carved in stone in modern medicine serves as a good example of this issue and illustrates how deficiencies and the disabling of a critical enzyme-catalyzed biochemical pathway can result in widespread and unnecessary suffering. Statins provide another example of the inhibition of an important pathway with important downstream implications. This issue includes a news release to British newspapers concerning the movement underway to increase statin use among all age groups. Other topics discussed include rapid detoxification with an organic diet, vitamin D and multiple sclerosis and the poor performance of drugs used to treat Alzheimer's disease.

Finally, if you need to restock your supplements, please remember that by ordering through our on-line vitamin store you will be helping to maintain the web site and 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

PROBLEMS WITH DIAGNOSIS AND TREATMENT OF HYPOTHYROIDISM

Hormones comprise a large class of chemicals essential to the functioning of our biochemistry. The common ones such as estrogens and testosterone have a very high profile. The form of vitamin D that is used as a serum marker for sufficiency is a prohormone and is converted into a hormone which is responsible for its biochemical activity. Melatonin is a hormone excreted by the pineal gland located in the brain and commonly manipulated by supplementation for sleep pattern normalization. The thyroid gland excretes two major hormones, thyroxine (T4) and triiodothyroxine (T3), both of which work inside the cells of the body, mainly influencing metabolism. T3 is much more biochemically active than T4 and is the form of thyroid hormone responsible for stimulating cells to produce energy. T4 incidentally contains four iodine atoms and T3 contains three, underscoring the role of this element in thyroid function.

Hypothyroidism, also termed an underactive thyroid, is the most common disorder associated with the thyroid hormones. It appears to be significantly under-diagnosed and thus the prevalence is controversial, but estimates as high as 40% of the adult US population have been advanced.[1] Even if the prevalence is 20% or 30%, this represents a serious and significant health issue with a large set of symptoms and associated disorders. A sample: cold hands and feet, constipation, depression, hypertension, fatigue, inability to concentrate, infertility, irritability, menstrual irregularities, muscle weakness, nervousness, and poor memory. Some of these symptoms could precipitate a prescription for a psychiatric drug and being labelled with a mental disorder. While many individuals are screened for thyroid disorders during routine physical exams or because several symptoms raise the question, how hypothyroidism should be diagnosed is also controversial. These disagreements generally exist between conventional and holistic or integrative physicians.

Screening and diagnosis for hypothyroidism generally involves measuring the serum level of a regulatory hormone known as the thyroid stimulating hormone or TSH. This hormone is secreted by the pituitary gland and causes the thyroid to release the thyroid hormone T4. In standard practice, TSH is the only marker measured. The physician then compares the result with the reference range and on this basis decides whether or not to pursue the question further. However, there is controversy over the reference range. A typical reference range is 0.4 to 4.5 mIU/L and thus a diagnosis of hypothyroidism is triggered by a TSH ≥ 4.5 mIU/L. As is common with reference ranges, this one encompasses values found in the population

between the 2.5 and 97.5 percentiles. Such a system may be fine for rare disorders, but not for those with high prevalence. Thus under-diagnosis automatically occurs since large numbers of individuals with hypothyroidism are included in the laboratory reference range.

Dr. David Brownstein, one of the leading critics of the current reference range, has proposed revision to 0.3 to 2.0 mIU/L.[1] This proposal is based on many years of treating thyroid disorders diagnosed in a much more comprehensive manner, and in fact, he makes the point that it is inappropriate to diagnose hypothyroidism by relying only on blood tests. Rather, he uses the following: blood tests, medical history, basal body temperature and physical examination.

The objective of this discussion is to alert readers to the reference range problem and to encourage them to demand a comprehensive thyroid blood test panel. The results of the blood tests plus a simple basal body temperature measurement made at home can then provide the basis for a discussion with one's physician concerning the question of hypothyroidism. Given what appears to be rampant under-diagnosis and the simplicity of treatment, this is a serious matter, although how to treat is also controversial.

The need for more comprehensive blood tests involves the basic biochemistry of T4 and T3. Measuring TSH provides an indication of the T4 level, an inverse association. However, T3 is not excreted separately but is produced (converted) from T4 by an enzyme catalyzed reaction. Since T3 is really the important thyroid hormone, anything that interferers with this conversion can result in hypothyroidism. However, there is great resistance in modern medicine to measure anything other than TSH, thus missing those with low T3. The point that strangely seems to be missed is that a normal T4 does not imply a normal T3.

In his book *Overcoming Thyroid Disorders,*[1] Dr. Brownstein lists twenty factors that can block or decrease T4 to T3 conversion. These include chronic illness, smoking, drugs, heavy metal toxicity, high levels of stress, low adrenal status, malnutrition, mineral and vitamin deficiencies, advanced age and non-fermented soy ingestion. Among the drugs are birth control pills, estrogens, beta-blockers, antidepressants, antimanic agents such as lithium, chemotherapeutic drugs, and iron supplements if taken with thyroid medications. Among the toxins are fluoride, mercury, lead and bis-phenol A (BPA). Vitamin and mineral deficiencies implicated include selenium, and vitamins A, B6 and B12.

There is another problem. T4 can be converted into reverse T3, rT3. This is in part a response to too much T3. According to Dr. Brownstein the most common cause of an elevated rT3 is iodine deficiency. It leads the body to slow metabolism by converting T4 into rT3. Checking rT3 is rare in spite of the fact that it provides additional information about thyroid function.

It has been known for over a century that there is an association between iodine and thyroid function. Iodine deficiency can be caused by low levels in the soil yielding low levels in food. The classical goiter belt phenomenon which motivated the addition of iodine to table salt nicely illustrates the problem. However, it is far from clear that this has solved the problem and the current movement to severely limit salt merely adds to it. Both selenium and iodine are required for the enzyme catalyzed reaction that converts T4 to T3. In some cases, symptoms of hypothyroidism disappear after only addressing deficiencies in these two elements. Furthermore, in areas where there is a soil deficiency in iodine, there is also frequently a deficiency in selenium, and the stage is set for T3 deficiency.

Rather than worry about the factors that inhibit the T4 to T3 conversion, it makes more sense to simply determine by a blood test if this is a problem. Brownstein cites reference ranges for T4 and T3 as 80-180 µg/dL and 55-120 ng/dL respectively. Individuals can have the signs and symptoms of hypothyroidism even with normal TSH and T4 levels if their T3 is low,

simply because it is the most active of the thyroid hormones. Modern medicine for the most part appears to ignore this.

The basal temperature measurement indirectly checks thyroid function by the normality of an individual's metabolism. The procedure is straightforward. Morning temperature, either underarm or oral (if there is no upper respiratory infection) is taken on five successive days before getting out of bed. For women who are menstruating, the temperature should be taken starting on the second day of menstruation. If thyroid function is normal, the underarm temperature should range between 97.8° and 98.2°F (36.6° to 36.8°C), the oral between 98.8° and 99.2°F (37.1° to 37.3°C), Lower values than these suggest hypothyroidism.

The most common physical manifestations suggestive of hypothyroidism, according to Dr. Brownstein, are poor eyebrow growth especially in the outer third of the eyebrow and swelling under the eyes, so-called puffy eyes. The list of signs and symptoms given above also provides a rough guide.

Individuals who believe that measuring TSH is not enough and request a more comprehensive assessment may experience very strong resistance or even hostility. Generations of medical student have been taught that TSH is all one needs to measure and that the reference ranges are definitive. In some health care systems, even ordering T3 and T4 in the presence of a "normal" TSH can result in problems for a physician, especially as concerns grow over controlling expenses. Some doctors who test for T3 and T4 have been called "quacks." Patients may need to pay for the T3 and T4 tests themselves. Or they may have to find a physician who disagrees with what is a well-established orthodoxy.

When TSH is above the upper reference range limit, the conventional approach is to prescribe a synthetic (translation—patentable) form of T4. This brings down TSH and the therapy is regarded as a success, since by definition the hypothyroidism is gone. All medical students are presumably taught this. From what has been outlined above, this conclusion is not correct if the problem is poor T4 to T3 conversion. The traditional alternative therapy is a desiccated thyroid preparation derived from pigs. These go by the trade names Armour Thyroid, Nature-Throid, or Westhroid, and the preparations contain T4 and T3 and also compounds that improve thyroid function. This approach addresses the T4 to T3 conversion problem, and if it is effective, it is generally obvious to the patient almost immediately and confirms the diagnosis. Mainstream medicine regards using this natural product as old fashioned, and incorrectly believes that the quality control is poor for these products while ignoring repeated problems with quality control for the synthetic T4 products. However, the natural products have been used successfully for decades.

Note that long-term thyroid hormone use in the presence of an iodine deficiency increases the risk of breast cancer. It is in fact important when addressing thyroid problems to make sure that there is no deficiency in iodine. There are mixtures of iodine and potassium iodide available over the counter or at compounding pharmacies which make it easy to maintain an adequate iodine level. The classical preparation is called Lugol's Solution. Readers are referred to another book by Dr. Brownstein, *Iodine. Why you need it .Why you can't live without it."*[2]

As mentioned above, conventional physicians rely only on synthetic T4, called levothyroxine sold by prescription as Synthroid or levothroid. However, there is also a synthetic T3 called liothyroine sold as the prescription drug Cytomel. There is also a synthetic preparation that contains both T4 and T3 (Thyrolar). Compounding pharmacies also offer custom combination preparations of T4 and T3. Note that totally bioidentical thyroid hormones are not available. Those derived from pig thyroid glands are very close, as are the synthetic versions, and all appear to possess acceptable biological activity.

A recent review of studies of combination therapy (T3 + T4) concluded that while meta-analyses failed to find clear benefits, continued interest is warranted due to methodological deficiencies in the majority of studies. The authors comment that experimental and clinical evidence suggests that a TSH level within the reference range is not a sufficiently optimal marker of adequate thyroid replacement therapy in hypothyroid patients, a comment that must also apply to diagnosis.[3]

We are discussing a serious matter. Whatever the prevalence of hypothyroidism is, it is high enough to present a significant public health problem. One area of concern involves the association of depression with hypothyroidism. While this is a controversial subject, a recent review stated categorically that hypothyroidism has been linked to depression by irrefutable evidence and depressive patients have a higher frequency of hypothyroidism.[4-6] Hypothyroidism may only be rarely considered in the diagnosis of depression in a short office visit to a general practitioner. There are many individuals being treated for depression with psychiatric drugs having serious adverse side effects when their depression may be the result of hypothyroidism which could have been easily and quickly cured with a natural product entirely free of side effects with the added bonus of probably relieving a number of additional symptoms that had a negative impact on the quality of life. These individuals probably all had "normal" TSH levels. Even if they did not, treatment with T4 therapy might have failed and the antidepressant settled upon as a solution. In this context, it is very interesting that antidepressants block T4 to T3 conversion. It is also interesting that there are a number of reports in the literature where patients were resistant to antidepressants and especially SSRIs, and the addition of T3 produced large, positive benefit.[5]

Drug treatments of some of the other symptoms of hypothyroidism may be doomed to failure because they only address symptoms and leave the cause intact. Targeting symptoms is understandable when no one has a clue as to how to achieve a cure of some disorder, an unfortunately common situation. In the case of hypothyroidism, it borders on malpractice.

It can be concluded that anyone who suspects they suffer from hypothyroidism should demand blood tests of at least TSH, T4 and T3 and should rely on interpretation only from someone experienced in this field and without bias against measuring T4 and T3, i.e. someone who recognizes that there are patients who are poor converters for any of a number of reason and that the conventional approach is not satisfactory. The number of therapeutic options is clearly large and the need for professional guidance seems clear.

Readers having any suspicion of thyroid problems should read Dr. Brownstein's book. It is the third edition so it is right up to date.[1]

RAPID DETOXIFICATION SIMPLY WITH ORGANIC DIET

Organophosphate pesticides are widely used and are more acutely toxic than the older organochloride chemicals such as DDT which they replaced. Humans, among other living creatures, are exposed to these toxins through ingestion, inhalation, and dermal absorption. Occupational contact and proximity to farms can result in serious exposure. There is no question that low levels exist in food and that authentic organically grown food is relatively free of this important class of toxin. Body loads can be estimated by urinary levels of the metabolites of organophosphate pesticides and this can be used to advantage in examining the success of detoxification.

A study from Australia just published in the journal *Environmental Research* examined the hypothesis that something as simple as changing to a diet of organically grown food has the potential to flush out organophosphates.[7] The study involved 13 participants who were randomly assigned to consume an organic diet (at least 80% organic) or a conventional diet

for 7 days. The crossover design used then had the two groups switch for a second 7 day period. Urine levels of metabolites of the most common organophosphates were determined.

During the organic phase, the participants consumed an average of 93% organic food of which 83% was certified and 10% likely. The amazing result was that urinary metabolite levels were 89% lower when participants consumed organic vs. conventional foods and that this drop occurred over only 7 days. In other words, significant amounts of ingested organophosphates pesticides are metabolized and excreted within a week of ingestion, a result the authors point out is consistent with previous studies on children that demonstrated reductions in the body burden of organophosphates driven by the consumption of organic foods. The children were aged 2-5. Children consuming a conventional diet had nine times the urinary levels of metabolites compared to the children consuming an organic diet.

These results suggest that at least for this class of toxin, there is a good chance of reducing the body load to near zero by prolonged near total avoidance. However, it is important to recognize that toxins differ significantly in how and where they are stored and in addition, there are big differences in how toxins are ultimately eliminated through the body's various mechanisms. This was discussed in the March 2014 issue of IHN (www.yourhealthbase.com/ihn245.pdf) where a variety of detoxification interventions were described, including avoiding the toxins in the first place.

CONSUMERS UNION TAKES A POSITION ON ALZHEIMER'S MEDICATIONS

Consumers Union, publisher of *Consumer Reports,* includes drugs in their field of activity and has recently issued a report on Alzheimer's disease (AD) medication in search of so-called best buys, a judgment based on benefit, risk and cost. The report is available on the internet. Their evaluation was primarily based on an independent scientific review by a team from the Oregon Health & Science University and as well took into account the 2010 report from the US federal Agency for Healthcare Research and Quality and an evaluation conducted by the US Department of Veterans Affairs.

The conclusions:

- Medications to treat AD are not particularly effective. When compared to a placebo, most who take such medication will not experience meaningful benefit.
- It is the rare person who has a significant delay in the progression of symptoms over time.
- There is no way to predict who will experience any benefit at all.
- Costs range from $177 to $400 per month.
- Of the five drugs they reviewed, one (Cognex) is now rarely used because of the risk of liver damage. The other four can cause several side effects, but most are minor such as nausea, vomiting, dizziness, loss of appetite, muscle cramps, tremors and weight loss. If these persist, some may find them intolerable. More serious but rare side effects such as slow heart beat, heart block, gastrointestinal bleeding and ulcers and possibly seizures and convulsions have been reported.

No drug was recognized as a "Best Buy" although the authors of the report indicated they realized many patients and caregivers will want to try this approach.

The reader may want to compare the pharmaceutical approach reviewed by Consumers Union with the intervention using medium chain triglycerides including coconut oil even though the latter is based only on favourable anecdotal reports, incidentally along with considerable biological plausibility. See the September 2012 issue of IHN

(www.yourhealthbase.com/ihn230.pdf) for a detailed discussion. Incidentally, justified by a large number of positive anecdotal reports, a significant clinical trial of medium chain triglycerides plus coconut oil is now underway at a U.S. university.

VITAMIN D AND MULTIPLE SCLEROSIS PROGRESSION

Growing evidence suggests that vitamin D deficiency may be one of the most important factors in the development of multiple sclerosis (MS).[8] Higher MS risk has been reported in individuals with low vitamin D intake or lower circulating 25(OH)D levels and was well, an inverse correlation has been observed between vitamin D status and MS activity. This suggests that vitamin D is associated with the disease process that leads to MS and perpetuates it. According to the Cleveland Clinic website, the prevalence of MS in the US is about 350,000 cases (1.5 per 1000 adults) and worldwide, 2.5 million.

Multiple sclerosis patients frequently present with what is called a *Clinically Isolated Syndrome* (CIS) which can evolve into MS. CIS describes a first episode of neurologic symptoms that last at least a day and is caused by inflammation and demyelation in one or more sites in the central nervous system. It can be caused by one or more lesions and termed monofocal or multifocal. When CIS has occurred, there is a high likelihood of a second event and progression to MS.

A very well designed follow-up study recently published examined the association between vitamin D status and the progression of CIS to MS.[9] The study group was actually involved in a randomized trial of an interferon drug and because of a long open label phase at the end, most of the participants were on the drug during part of the trial. Measurements of 25-hydroxyvitamin D were available for a number of participants which allowed the examination of the association between vitamin D status and new active lesions, increased lesion volume and brain volume changes on MRI imaging as well as MS relapses and disability.

The 25(OH)D levels in 464 participants ranged from 19 to 98 nmol/L, i.e. from highly deficient to definitely sufficient. At the end of a five-year follow-up, the general conclusion was that low vitamin D status early in the disease course was strongly related to the risk for long term MS activity and progression. Patients with 25(OH)D levels ≥ 50 nmol/L had a 4 times lower change in lesion volume, a 2-fold lower rate of brain atrophy, and lower disability than those with levels below 50 nmol/L. When the results were stratified by quintile of season-adjusted 25(OH)D (baseline, 6 and 12 months) a definite dose dependence was seen in the most sensitive MRI outcomes. The authors comment that while there was a clear dose dependence observed, there was no evidence of levelling-off and it is possible that potential benefits of vitamin D were not reached because of the moderate levels observed in the highest quintile (median 69 nmol/L or 27.6 ng/mL) Individuals taking adequate supplemental vitamin D frequently reach levels well above 100 nmol/L. A plot of the probability of converting to clinically diagnosed MS over 5 years showed a 10% absolute difference between those with 25(OH)D ≥ 50 nmol/L and those below this threshold. But again, the critical unanswered question is what if there has been a significant number with this marker ≥ 100 or even 120 nmol/L?

These results are consistent with another study published about the same time.[8]

STATINS FOR EVERYONE?

The following press release was sent to all British newspapers on June 10, 2014. This news release reflects growing unease and concern among a minority of highly qualified healthcare professionals and medical scientists concerning the notion that large numbers of individuals still are at risk because of statin drug deficiency and that the thresholds must be lowered to save them from cardiovascular disease. NICE is the British National Institute for Health and Care Excellence. Here is the press release.

An open letter has been sent to the chair of NICE and the secretary of state for health, Jeremy Hunt, from leading doctors in primary care, secondary care and academia. They reject the recent draft guidance from NICE to reduce the threshold for prescribing statins to those with a 10% risk of cardiovascular disease (potentially treating an additional five million healthy individuals).

The letter is signed by a number of leading figures in healthcare including the president of the Royal College of Physicians, Sir Richard Thompson, Professor Clare Gerada, Past President of the Royal College of General Practitioners and Professor Simon Capewell, Clinical Epidemiologist at the University of Liverpool.

Other signatories include Professor David Haslam, Chair of the National Obesity Forum, GP Dr Malcolm Kendrick, London Cardiologist Dr Aseem Malhotra and Professor David Newman, Emergency Medicine physician and Director of clinical research and Mount Sinai School of Medicine, New York.

They address six major concerns and call on NICE to refrain from any final recommendations on reducing the threshold for statin guidance until these are fully addressed.

These six key areas are:

1. *The medicalization of millions of healthy individuals*
2. *Conflicting levels of adverse events*
3. *Hidden data*
4. *Industry bias*
5. *Loss of professional confidence*
6. *Conflicts of interest*

The group state the benefits of statins in a low risk population do not justify putting millions of extra people on a drug which then has to be taken lifelong.

They also express serious concerns that the data driving the latest guidance comes almost entirely from pharmaceutical-sponsored studies; because extensive research reveals that industry-sponsored trials systematically produce more favourable outcomes than non-industry sponsored ones. Industry trials also grossly underestimate adverse effects, partly by removing patients who fail to tolerate the drug in the selection process. They state "relying on these studies alone will not represent those patients taking the drug in the real world."

The group cite important findings from non-industry sponsored studies which include a 48% increased risk of developing diabetes in middle aged women taking statins, while a robust randomized controlled trial revealed that 40% of women had reduced energy and fatigue. Other patients experienced psychiatric symptoms or erectile dysfunction.

They call on the Cholesterol Treatment Trialists Collaboration who has commercial agreements with the pharmaceutical industry to release all data on statins which is currently being concealed for review by independent researchers to help explain major discrepancies in several industry-sponsored studies of statin adverse effects.

The leading doctors also mention that GPs feel that greater prescribing of statins to healthy people is a "step too far" citing the General Practitioners committee's rejection of NICE guidance a few weeks ago until it is supported by evidence derived from complete public disclosure of all clinical trial data"

The group express "serious concerns" that 8 of 12 of NICE's panel of experts on the latest statin guidance have direct financial ties to the companies that manufacture statins. They instead emphasize that parties with industry conflicts should NOT be participants in generating recommendations on drug use across the population.

The leading doctors call on NICE "to withdraw the current guidance on statins for people at low risk of cardiovascular disease until all the data are made available."

They conclude: "The potential consequences of not withdrawing this guidance are worrying: harm too many patients over many years, and the loss of public and professional faith in NICE as an independent assessor. Public interests need always to be put before other interests, particularly Pharma."

Professor Simon Capewell, Professor of Clinical Epidemiology at the University of Liverpool said,

"Two decades of research has confirmed the obvious: doctors receiving drug industry funding produce recommendations favouring the industry. It also represents a further embarrassment for NICE. NICE urgently need to develop a better mechanism for controlling these conflicts of interests. The recent statin recommendations are deeply worrying, effectively condemning all middle aged adults to lifelong medications of questionable value. They steal huge funds from a cash-strapped NHS [National Health Service], and they steal attention from the major responsibilities that government and food industry have to promote healthier life choices for ourselves and our children"

London Cardiologist Dr Aseem Malhotra said,

"Although there is good evidence that the benefits of statins outweigh the potential harms in those with established heart disease, this is clearly not the case for healthy people. For example a doctor wouldn't give chemotherapy to a patient who didn't have cancer or prescribe insulin to someone without diabetes. When you add up hospital appointments, unnecessary suffering for those who experience side effects that interfere with the quality of life, the illusion of protection of taking a drug that won't reduce the risk of death in healthy people- and the increasing burden of chronic disease which is predominantly lifestyle related- prescribing statins to millions of healthy people would increase costs to the NHS, not reduce it. I became a doctor to practice medicine that's best for patients based upon all the available evidence, independently evaluated, not medicine that's purely eminence based or corporate influenced."

Dr Malcolm Kendrick, GP and member of the British Medical Association's General Practitioner's sub-committee said,

"Who knew that millions of people in the UK now suffer from statin deficiency syndrome? Mass statination is the triumph of statistics over common sense. Treating millions at a cost of

billions all based on data we are not allowed to see is another example of the corporatization of medicine and will result in a public health disaster."

Dr David Newman, Assistant Professor of Emergency Medicine and Director of clinical research at Mount Sinai School of medicine, New York said,

"I am always embarrassed when I have to tell patients that our treatment guidelines were written by a panel filled with people who stood to gain financially from their decisions. The UK certainly appears to be no different to that of the United States. The truth is for most people a statin will give them diabetes as often as it will prevent a non-fatal heart attack—and they won't live any longer taking the pill. That's not what patients are looking for."

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