Sanford Health

Aunt Cathy's Guide to:

Thinking About OTHER Nutrition Issues in Diabetes

8-13

AUNT CATHY

Cathy Breedon PhD, RD, CSP, FADA
Prenatal/Pediatric Nutrition Specialist
Clinical and Metabolic Nutrition Specialist
Sanford Health (formerly MeritCare)
and UND School of Medicine
Fargo, North Dakota

Introduction:

Blood sugar control is the key focus of nutrition and diet planning for people with diabetes. Historically the most attention has been paid to adjusting the intake of the types and amounts of carbohydrate throughout the day, including the newer concepts of "carbohydrate counting" and the "glycemic index." Additional attention has been paid to total calories and total fat content, plus the content of saturated fat and "trans" fat because of the importance of weight control in diabetes, and the increased risk of cardiovascular disease. **These remain the basis of diet and nutrition interventions for diabetes.**

However, information is becoming available about the special roles of **certain vitamins, minerals, phytochemicals, and some forms of dietary fat** that appear to be useful in various ways in fighting diabetes and its consequences. The nutrition tools described here do not "cure" diabetes – but in many cases they have been shown to have the capacity to **decrease the risk of diabetes developing**. Others have demonstrated that they can be useful for people who already have it by **helping with diabetic control,** or by **making diabetes hurt them less.** This includes reduction in complications like blindness, kidney failure, neuropathy, impaired circulation, and even birth defects.

Some have been shown to be helpful in at least one of seven ways:

1) Enhancing insulin sensitivity.

2) Using antioxidants to minimize secondary damage from poor diabetes control caused by higher than normal production of free radicals.

3) Reducing the severity of inflammation that has been shown to be associated with diabetes in order to decrease risk of developing diabetes complications (including Type I, Type II and gestational forms.)

4) Helping stabilize/improve excessive fluctuations of blood sugar that can sometimes be a problem in spite of following the diet very carefully (e.g. what some people used to refer to as "brittle" diabetes.)

5) Minimizing risk of developing other threats to health and well-being, including conditions that are known to be exacerbated by diabetes.

6) Minimizing the risk of developing diabetes.

7) Discovering the emerging miscellaneous effects of a variety of phytochemicals (plant substances) on the diabetic state.

Happily, all of the diet or nutrient ideas suggested here are also reported to be beneficial for most folks in terms of decreasing risks associated with one or more of a variety of chronic illnesses, including cardiovascular disease, cancer, MS, arthritis (both rheumatoid and osteo,) alzheimers, parkinsonism, depression and osteoporosis.

In other words, the suggestions made here are all health-promoting for the whole family, whether one has diabetes or not … they are just even more important if you DO have diabetes.

It is also extremely important to note that all of the suggestions are based on research reported in the legitimate peer-reviewed scientific literature. None are based on wild claims made on the internet (or elsewhere.) There is no law against fiction in America, and therefore there are quite a lot of false claims made for diet and nutrition in particular, often in the interest of selling products.

Many of the "dietary supplements" sold are in fact not related to nutrition at all. Many are actually pharmaceutical products, but a loop-hole in the FDA laws allows the marketing of substances in the "dietary supplement" category without having to show "safety and effectiveness" as they would if marketed as pharmaceutical products.

Understandably, the existence of so much nutritional quackery in the world has made health professionals hesitant to consider nutrition manipulations to be legitimate adjuncts to management of complex diseases like diabetes. There is a tendency to just "throw the baby out with the bathwater." The purpose of this paper is simply to highlight areas of research that are identifying the real "babies" to watch out for.

All the suggestions included here are generally regarded as safe at this time (I never recommend anything that comes anywhere near to "scary,") and they are often easy to do and inexpensive. In two cases, the amount of the substance that has been shown to be beneficial is somewhat impractical to attempt when using the currently available over-the-counter products. In these cases, a prescription for the appropriate higher dosage product makes it much simpler. Although these two products and one other are also among the more expensive interventions discussed, the prescription form may be covered by insurance even though the over-the-counter form is not.

As always, the information provided here is simply my best interpretation of the research currently available. No claims are made that any of the large official health organizations have approved this message. It is all subject to change, which is why my papers are marked with a date. And as is always the case for everything in the world, "more research is ALWAYS needed," but now there is actually quite a lot of research out there, and certainly for some things there is clearly enough evidence to initiate some safe and cheap changes. It's at the **"won't hurt / really might help"** level.

I am not selling anything – no supplements, no diet plan, no books. My only goal is to try to do some good for people. However, also as always, not all the suggestions are appropriate for every individual, so be sure to **discuss things with your personal health care provider. Feel free to share these materials with him/her.**

References for the research cited in this paper through 2010 are included at the end, divided into subtopics. There are LOTS more that has been published since 2010 in the same direction relative to the recommendations below, but I haven't yet had a chance to write up a nice big bibliography for each topic. However, some of the most up-to-date references are included in the discussion and suggestions in this paper, and they are also available on Public Medline www.pubmed.gov. This website allows anyone to access the scientific reports available in the National Library of Science / National Institute of Health.

On the www.pubmed.gov website, just type the word diabetes and another word of interest in the search box (examples: "diabetes biotin" "diabetes carnitine" "diabetes alpha-lipoic acid") and you will find the references (and abstracts) noted here and also those that have appeared on this topic since 2010. The some data and conclusions from these newer reports have been incorporated into the text of this paper. I just haven't shined up the reference pages to reflect the post-2010 references. I plan to do this, of course, but just reading all this stuff on diabetes and nutrition (and other nutrition topics in my other papers) and thinking about it and then tucking it in where it goes in to make the latest version of the paper is pretty time consuming. It's like writing term papers for school every single day … not a pretty picture! ☹

Also, in the interests of brevity and saving trees, I will sometimes refer you to another of my papers for more complete information about a particular topic. In addition, papers with more details on this topic and others available for free on the Sanford Health website.

Part One: Enhancing insulin sensitivity and decreasing incidence of diabetes.

Magnesium and chromium are two minerals which have specific roles in carbohydrate metabolism. **Both are frequently suboptimal in the diets of the majority of Americans** (based on data from the National Health and Examination Survey – NHANES) for a variety of reasons and

assuring adequacy has been shown to be critical for insulin sensitivity. For more details, references, and specific recommendations, please see "Aunt Cathy's Guide to Nutrition: Magnesium" and "Aunt Cathy's Guide to Nutrition: Chromium."

Chromium

Chromium is a component of "Glucose Tolerance Factor." It has roles in the metabolism of carbohydrate, protein and fat. Supplementation of chromium picolinate (a well-absorbed form) has been shown to be helpful in blood sugar control and weight loss among people with Type II diabetes on sulfonylurea medication. However, at least in the amounts studied, it may not show all the same benefits among obese people on insulin whose type 2 diabetes is poorly controlled. But just supplementing at the Advisable Intake levels has also been shown to have a role in managing dyslipidemias like high triglycerides and high LDL cholesterol.

As expected, additional **supplementation is unlikely to add further benefit if one's needs are already met**. However, the assumption of chromium adequacy is clearly erroneous for most Americans and standard care typically does not include asking questions about intake of chromium-rich foods or chromium supplements. In fact, many folks, including many health professionals, are not familiar with which foods are the richest sources. It has been generally "not in our radar." This continues to a be a problem when interpreting studies providing supplemental chromium are conducted showing no apparent benefit with outcomes like "developing diabetes" or changes in "insulin resistance," but actual chromium status at the onset is not evaluated.

Chromium is not the only player on the team, but it is definitely one of them. It also has roles in cholesterol and triglyceride metabolism, heart disease and possibly atypical depression. Improved cognitive-cerebral function has been demonstrated in older adults with chromium supplementation. Inadequacy is common and definitely not benign. References follow at the end of this paper.

For more information please see my handout: "Aunt Cathy's Guide to Nutrition: Chromium."

Magnesium

Magnesium has many roles in metabolizing carbohydrate, protein and fat, including being an essential cofactor in the production of ATP via the TCA Cycle. [ATP is a major form of energy used by the cells … we make by burning carbohydrate, fat or protein fuel.] In fact, **magnesium is known to play a role in over 300 chemical reactions in our bodies** including nerve activity and managing heartbeat, so it is not a surprise that it is a very important nutrient, and that inadequacy can be an issue in a broad range of health conditions.

Magnesium is specifically important for the functioning of the insulin receptors on cells, so magnesium inadequacy is a known contributor to **insulin resistance**, a key feature of Type-2 diabetes.

In a very large prospective study from Harvard University it was found that **women with the poorest magnesium intake from food compared with the highest were 25% less likely to have developed diabetes over a 16 year period.**

Detail: They found that women who ate about an ounce of nuts or peanuts four times a week or more were 25% less likely to have developed Type II diabetes in the 16 years of data analyzed, compared with the women who ate these foods rarely.

They then re-evaluated the data looking at magnesium intake from ALL sources, and they found the very same step-wise relationship: dividing magnesium intake into four groups, there was a stepwise relationship found in the incidence of Type II diabetes.

The higher the magnesium intake group, the lower the incidence of developing diabetes.

Two more major studies with important contributions on this topic:

In a Framingham study, magnesium intake was associated with insulin sensitivity in a threshold fashion, with a threshold seen at 325 mg magnesium daily.

And, an important finding in the huge National Nutrition and Health Examination Survey (NHANES) from the Center for Disease Control was that the MAJORITY of Americans take in less than 2/3 of the normally recommended amount of magnesium.

But in spite of that very eye-opening NHANES finding, still we (including health professionals) assume that folks are just fine in the magnesium intake department. This is because people who are deficient in magnesium do not "look funny" … at least not for that reason. ☺ So, we just conclude that the person "looks well-nourished."

The problem is that when 300 metabolic pathways are compromised by inadequate magnesium, the consequences are too general to easily pick them out. If there was just one system down we have a chance of recognizing the problem. With so many systems down, the best description I can come up with for the effect is a sort of "global crummy-ness" … very serious health problems but too general to pin down to recognize all the causes.

But what the NHANES study DOES mean is that if I met you on the street and had to place a bet on whether your magnesium intake was at the recommended level or not for healthy people, I would win my bet far more often if I said that your intake failed to meet that amount.

For that reason, one of my key operating rules is to assume that magnesium intake is fairly crummy unless I ask some specific simple questions and am assured that they are likely doing just fine. I don't assume adequacy, I assume INadequacy until proved otherwise.

Here's how to do that simply and easily:

Magnesium Status Evaluation Rule of Thumb: "Baby Plants"

The terrific magnesium food sources are the parts of plants that will become "baby plants" … nuts, seeds, legumes and the germ of grains.

That is, if it is a seed, it's a good source. If it's a nut it is a good source. If it is the germ of grain it is a good source. If it is a legume (bean, pea, lentil, peanut, etc.) it is a good source. Are we seeing a pattern here?

So, if they say they eat them often, the odds are good that they are in the generally OK range for healthy people.

If they eat them rarely, the odds are good that they are not taking in optimal amounts of magnesium even for healthy people. This is also WAY below the optimal intake for most folks with diabetes.

One really nice thing about this rule is that you can easily get a picture of someone's usual magnesium intake just by asking specifically about eating these foods, and you also know what foods to suggest they include more of if they have a poor magnesium intake! ☺

It also allows us to easily and intelligently answer specific questions that people ask. At a conference recently somebody asked if chia seeds were good magnesium sources. Another asked about sesame seeds. Without having to have the amounts memorized or having ever even looked them up, I could easily answer … "Yep … they are SEEDS!" **Being a baby plant means that the magnesium content per weight or per calorie is good.**

However, an additional comment of importance in this context would be "but those guys are really tiny so you would have to eat a whole lot if you were counting on them to meet your magnesium needs."

The Baby Plant rule also helps me easily answer questions that I regularly get like this: "Which is better for you: flax oil or flax seeds?" Answer: both have flax oil … a heart-healthy fat. But the flax SEED has lots of other goodies in there that the oil alone cannot contribute. So nutritionally, the flax seed is more "nutrient-dense" … but both are perfectly fine foods to use in one's diet or as a supplement.

Application … since doctors and others have no time to be asking this kind of thing (there are a lot of other critical issues to cover in their 15-20 minutes of contact time), one way to incorporate this approach is to have the receptionist give each patient checking in a bit of paper with screening questions like this on it:

About how often do you eat nuts and peanuts?
___ More than 5 times a week ___A couple of times a week ___Once a week or less

About how often do you eat peas or beans (like chili beans, baked beans, lentils, soy, refried beans.)?
___ More than 5 times a week ___A couple of times a week ___Once a week or less

This quickly identifies for the doctor/nurse/pharmacist/dietitian who might need some encouragement to eat more of these.

Then there could be a simple sheet given to all the patients that encourages intake of these foods for everyone. Most folks appreciate being told that nuts and peanuts are actually great foods for them … often they have been avoiding them because "they are high in calories" or "they have fat in them." The fat has the same calories as any other fat but they mostly monounsaturated and omega-3 fats. They don't increase risk of cancer or heart disease as some other fats appear to do. Because of this, I think of them as "**Happy Fat**: Dangerous to your butt, but not to your heart." ☺

Note that if **calories** ARE a major issue for a patient, the legumes, beans and whole grains have the magnesium and other goodies, but far less fat and fewer calories than nuts and peanuts have.

Also, note that if a person is actually **allergic** to these foods it is not appropriate to encourage them to eat them anyway … do I really have to say this? This is a situation in which a **supplemental magnesium** oxide or magnesium chloride is appropriate to provide adequate magnesium. Another situation for which supplemental magnesium is reasonable is if they simply hate those foods and are quite unlikely to eat them. You can still "encourage" them to eat these healthful foods, but don't count on them actually doing it. Get real!

Most "**multivitamins with minerals**" have between 10% and 25% of the RDA for magnesium, and this can be helpful if one's intake is just a little low, but it will not solve the problem if the dietary intake of magnesium is quite low … especially if the person has diabetes. Taking that multivitamin with minerals is still recommended for many reasons … but some additional magnesium would be in order.

[**Do not recommend magnesium citrate or sulfate** … these have poorly absorbed magnesium and they can cause loose stools. That's why they are used as laxatives and to clean out the colon before having a colonoscopy. They are a poor choice as nutritional supplements. ☹]

The **magnesium RDA** for women is 320 mg and 420 mg for men, and the RDAs are based on the needs of people who do not have diabetes or other health problems. **People with diabetes, however, often need magnesium intake levels above the RDA** for reasons explained later.

A low magnesium level in the blood (hypomagnesemia) is highly prevalent in outpatients with diabetes. High plasma triglycerides, waist circumference and albuminuria (all of importance in diabetes) are also independent correlates of hypomagnesemia.

The amount of magnesium in the blood is extremely important for heartbeat and other critical functions so the body works very hard to keep the amount in the blood in the normal range. The blood magnesium level is controlled by hormones and not by daily intake, so a low level of magnesium in the blood can be a marker of serious problems. It also means that **a normal blood magnesium level does not assure adequate magnesium levels in other parts of the body or in the diet**.

People with poorly controlled diabetes are at particular risk of actually losing magnesium. That is because when blood sugars are high enough that **sugar spills over into the urine**, it takes magnesium with it, resulting in an **additional loss of magnesium**.

This also occurs when one uses certain blood pressure medications like **thiazide diuretics,** like furosemide (Lasix.) Any diuretics known to result in potassium loss will also result in a magnesium loss, but often this is not recognized and corrected. More will be said about magnesium later, and I also have a separate handout available on line with much more detail on this critical nutrient.

Magnesium Summary

Magnesium insufficiency in the general public is quite common

Magnesium insufficiency is even more common among people with diabetes.

In spite of the recognized problems of in adequate magnesium intake from foods or supplements, intake is rarely evaluated by health professionals.

Laboratory assessment of serum magnesium levels does not reflect magnesium status in the tissues. The amount in the blood is controlled by the kidney so it is not very useful for assessing general adequacy. A low level is associated with serious health problems for a variety of reasons, but a normal level tells us very little about magnesium status in the tissues. **It is not an indicator of excellent magnesium status.**

Improving poor magnesium intake has the potential to help reduce the incidence of both obesity and type 2 diabetes. It also has roles in preventing hypertension (high blood pressure) and some neurologic problems such as leg cramps.

Improving poor magnesium intake has the potential to help decrease insulin resistance among people who already have diabetes. (At least, it gives us a more level playing field.)

Identifying folks whose diet is likely suboptimal is actually pretty darned easy, and giving them specific advice to improve it is also pretty darned easy using the "Baby Plants" rule as described above.

For more information on magnesium, please see: "Aunt Cathy's Guide to Nutrition: Magnesium" and "Aunt Cathy's Guide to Nutrition: My Current Top Five Easy Ways to Improve Your Family's Nutrition"

Vitamin K

Vitamin K adequacy is now recognized as important in diabetes because it has a role in both body fat and diabetes/glucose metabolism. Researchers have described a potential beneficial role for **p**hylloquinone (vitamin K in the form found in **p**lants) in glucose homeostasis. Higher phylloquinone intake was associated with greater insulin sensitivity and glycemic status. (Phylloquinone intake, insulin sensitivity, and glycemic status in men and women. Am J Clin Nutr. 2008 Jul;88(1):210-5.) Other forms of vitamin K (e.g. menaquinone and menatretenone) are also being studied with success in correcting complications of deficiency.

As **vitamin K inadequacy is now known to be much more common than was previously believed,** the role of vitamin K in diabetes and obesity will likely begin to be evaluated even more closely in the scientific community.

Assuring vitamin K adequacy has also been found to be of great importance for prevention of certain contributors to **cardiovascular disease**, such as calcification of the arteries, increased arterial inflammation and high blood pressure. Calcification of the **kidneys** is another consequence of vitamin K deficiency of particular importance in diabetes, as is **osteoporosis**, a condition known to be more prevalent among people with diabetes. **The role of vitamin K in these many conditions has only been identified in the years since 2005, so it is not yet well known, but the research is exploding on this topic.**

Other general health problems related to vitamin K deficiency include pre-eclampsia, varicose veins and cancer of the stomach and colon. **Clearly assuring adequacy is a very good idea for everyone, but it is especially important for people with diabetes.**

Vitamin K is fat soluble and this gives some folks the impression that it is therefore potentially quite toxic. **However, <u>vitamin K has been shown to be very safe</u>. In fact, no upper level of safety has been established for it because no one has ever been found to be harmed by it.**

A good way to think about its role in blood clotting is that it ALLOWS you to clot your blood if you get a biochemical message that you need to do some clotting to avoid excessive blood loss. Vitamin K does not MAKE you clot your blood, nor does it initiate the clotting of blood. It is not a hormone causing things to happen … it is just a tool along an assembly line that is needed for making clots when they are perceived to be needed because of injury and/or blood loss.

Re: Vitamin K interactions with the drug Coumadin/warfarin

The exception to the general non-toxicity of vitamin K is an interaction with a specific medication. Coumadin is an anticoagulant that works by interacting with vitamin K. For this reason, **consistency and adequacy of vitamin K are very important for the safety of using the drug.** People can actually use the drug Coumadin <u>more</u> safely when an adequate blood level of vitamin K is maintained because it prevents the extreme volatility that can be dangerous with this medication.

At present many people taking Coumadin are in fact deficient in vitamin K because of frequent misunderstandings and also due to the fact that the recognition of the body's many other roles for vitamin K is quite new. **Correcting the problem of actual vitamin K deficiency among users of this medication must be managed by the patient's health care provider.**

Note that many other anticoagulants do not operate via interaction with vitamin K, so assuring vitamin K adequacy is very easy and safe to achieve.

My paper for health care providers on this specific topic is available along with a more general one just on vitamin K and all the new findings about its roles in the body, recommended intake levels and sources.

For more information and details on the roles of vitamin K and ways to assure adequacy, please see my other handouts on line: "Aunt Cathy's Guide to Nutrition: Vitamin K -- New Issues in Cardiovascular Health, Osteoporosis, Cancer of the Liver and Colon, Diabetes and Varicose Veins." The other paper is called "Aunt Cathy's Guide to: Vitamin K -- Focus on the Vitamin K and Warfarin/Coumadin Anticoagulant Drugs Issue

Seven factors are contributing to the high prevalence of unrecognized vitamin K inadequacy:

1. Vitamin K is not included in many vitamin supplements because it was assumed that we could rely on intestinal bacteria to provide a substantial amount.

2. Our ability to rely on **intestinal bacteria** for vitamin K has been found to be overly optimistic, and we are more reliant on an outside source than we thought.

3. The best sources are **dark leafy green vegetables** and many folks rarely eat them.

4. Vitamin K status is **rarely evaluated** because it has always been assumed to be just fine based on the assumption that the intestinal bacteria made enough. At the moment, a blood test for vitamin K adequacy is not included in any of the major blood-test "panels" commonly obtained during a health evaluation. This situation may change as the problem of vitamin K inadequacy becomes better recognized, but for now it is very unlikely to be measured in anyone … even in people on the medication Coumadin, who are at great risk of vitamin K deficiency because of misunderstandings about the need for consistent vitamin K intake … not inadequate vitamin K intake. **Inducing a vitamin deficiency is never in the best interests of anyone.**

5. Currently established RDA-type **recommended levels of vitamin K intake have been shown to be insufficient** to maintain older individuals in the normal range of blood vitamin K. This has not yet been evaluated in other age groups, but it is likely that a similar result will be found.

6. People often **incorrectly believe that vitamin K is likely to be very toxic** because it is "fat soluble." It is actually extremely safe and no upper end of a safe intake has ever been found.

7. People using the **medication Coumadin** are the only people for whom vitamin K must be provided in a consistent and adequate) amount. Many of them are actually vitamin K deficient and the deficiency interferes with the safe use of the drug. At this time, because these issues are so new, the safe use of vitamin K with the drug Coumadin is very often poorly understood by the public and by many health care professionals.

Vitamin D

Vitamin D inadequacy is now known to be very common throughout the USA and around the world. The World Health Organization estimates that 40-50% of the world's population is vitamin D inadequate or insufficient. It is frequently unrecognized but it is now beginning to be checked much more often, and today a vitamin D assay is actually the most requested laboratory value in clinics and hospitals.

Over 200 tissues have receptors for vitamin D hormone, so inadequacy compromises a number of functions and increases risk of many health problems, including cardiovascular disease, cancer, autoimmune diseases and compromised immune function. New roles in diabetes are also being identified. Here are some highlights:

Type I Diabetes:

There is a growing body of good evidence that **inadequacy of vitamin D in early life may be one of the triggers that brings on Type I diabetes among genetically susceptible children.** For example, in a study in Finland, children who had experienced vitamin D –deficiency rickets as infants were four times more likely to develop Type I (insulin dependent) diabetes. The data is accumulating all around the globe, and it is consistent with the well known observation that **the northern tier of the US (and places of similar latitude around the world) is known as the "Rickets Belt", the "MS Belt" and the "Type I Diabetes Belt."**

Latitude is the most obvious factor in vitamin D inadequacy, but many other people are at risk because of skin color, clothing that covers a person up, diet, and several other important factors. **For many reasons, vitamin D inadequacy is a very large public health problem that has not yet captured the attention of most health professionals.** [I hope to be able to edit this last statement out in the near future.]

In terms of Type I diabetes in particular, the current state of the art is to recognize that assuring adequacy of vitamin D can be protective against Type I diabetes to at least some degree. There appear to be several polymorphisms of vitamin D receptors that can affect the relationship, and a variety of other factors. In any case, the documented high prevalence of vitamin D insufficiency in Americans and others, and in children with Type I diabetes in particular make it very reasonable to monitor vitamin D status in everyone and supplement if necessary to ASSURE adequacy instead of simply assuming adequacy.

Here are just three examples of the many references on this topic found at the end of this paper:

Vitamin D and increasing incidence of type 1 diabetes-evidence for an association?
Diabetes Obes Metab. 2010 Sep;12(9):737-43.
Significant vitamin D deficiency in youth with type 1 diabetes mellitus.
J Pediatr. 2009 Jan;154(1):132-4.
Vitamin D in diabetes mellitus-a new field of knowledge poised for D-velopment.
Diabetes Metab Res Rev. 2009 Jul;25(5):417-9.

Type II Diabetes:

Other studies suggest that inadequacy of vitamin D may contribute to Type II Diabetes as well as Type I, with additional roles in a wide variety of health problems. These include death from all causes, heart failure, cardiac arrest, cancer of the colon, prostate, pancreas and breast, MS, muscle/nerve pain that is often missed and diagnosed as fibromyalgia, rheumatoid arthritis and osteoarthritis, muscle weakness and falling (sarcopenia), fractures, osteoporosis, poor prenatal outcome, and much more.

In the spring of 2013 a meta-analysis of prospective studies was done by Harvard researchers that demonstrated a strong inverse relationship between vitamin D blood levels and incidence of type 2 diabetes. This is a very important report so I have pasted the abstract below:

Blood 25-Hydroxy Vitamin D Levels and Incident Type 2 Diabetes: A meta-analysis of prospective studies **[Harvard]** Diabetes Care **May 2013** vol. 36 no. 5 1422-1428

OBJECTIVE To quantitatively assess the strength and shape of the association between blood 25-hydroxy vitamin D [25(OH)D] levels and incident risk of type 2 diabetes. **RESEARCH DESIGN AND METHODS** A systematic search of the MEDLINE and Embase databases and a hand search of references from original reports were conducted up to 31 October 2012. Prospective observational studies that assessed the association between blood levels of 25(OH)D and risk of incident type 2 diabetes were included for meta-analysis. DerSimonian and Laird's random-effects model was used. A quadratic spline regression analysis was used to examine the shape of the association with a generalized least-squares trend test performed for the dose-response relation. **RESULTS** A total of 21 prospective studies involving 76,220 participants and 4,996 incident type 2 diabetes cases were included for meta-analysis. Comparing the highest to the lowest category of 25(OH)D levels, the summary relative risk for type 2 diabetes was 0.62 (95% CI 0.54–0.70). A spline regression model showed that higher 25(OH)D levels were monotonically associated with a lower diabetes risk. This inverse association did not differ by sex, duration of follow-up, study sample size, diabetes diagnostic criteria, or 25(OH)D assay method. **A linear trend analysis showed that each 10 nmol/L increment in 25(OH)D levels was associated with a 4% lower risk of type 2 diabetes** (95% CI 3–6; *P* for linear trend < 0.0001). **CONCLUSIONS Our meta-analysis showed an inverse and significant association between circulating 25(OH)D levels and risk of type 2 diabetes across a broad range of blood 25(OH)D levels in diverse populations.**

Assuring adequacy is easy, cheap and crucial to good health, and it is now recognized that **vitamin D inadequacy is very widespread in the US and around the world**. The World Health Organization (WHO) estimates that 40-50% of the world's population is at risk of inadequate vitamin D for a variety of reasons.

It is very clear that many people require levels of vitamin D intake well above the 200-400 iu RDA-ish recommendations. **As a rule-of-thumb, an intake of 2000 iu is a safe and reasonable maintenance dose.** Many people have been found in research studies to require that much to maintain healthy blood levels of this critical vitamin. **Additionally, <u>some</u> people have been shown to actually require levels as high as 5000-6000 iu/day just to MAINTAIN healthy levels.**

This illustrates the fact that people are not all the same and some people's requirements for particular nutrients are different from the presumed adequacy level. This is one important reason why a follow-up serum vitamin D level is a good idea a few months after having achieved a good blood level.

[Also note that 2000 iu presumed maintenance level is not a therapeutic dose for rapidly correcting a very low blood level. That level is often 50,000 iu/wk for eight weeks (a little above 7000 iu/day.) [The "Top Five Recommendations" paper discusses these issues in greater detail.]

Because <u>both</u> diabetes and vitamin D deficiency independently increase risk of cardiovascular disease, it would be prudent to take the extra step to ASSURE that one's vitamin D level is adequate by getting an annual check of vitamin D stores especially during the winter.

Also as discussed in the "Top Five Recommendations" paper, the blood level associated with the best health is 35-40 ng/dL or higher, and not the 25 ng/dL that had earlier thought to reflect a health level. An important note: Some lab report print-outs do not yet reflect this changing interpretation of "adequacy"; they still say that 25 is "normal." So when your lab comes back, be sure to ask what your actual number of ng/dL is, and not be satisfied with a description that says "normal."

As discussed earlier, one's intake of **fruits and vegetables**, intake of **vitamin K, magnesium** and **chromium**, and one's serum **vitamin C** level all also appear to be factors in the likelihood of developing Type II diabetes. It makes sense that assuring adequacy of <u>all</u> of these (and not simply assuming it) is a very reasonable goal.

Gestational Diabetes:

Gestational diabetes ("pregnancy diabetes") develops in mid-pregnancy in some women. It appears to develop less often among women with more vitamin D and vitamin C in their blood. A recent study showed a positive correlation of 25(OH) vitamin D concentrations (the measurement of vitamin D stores) with insulin sensitivity; they suggested that "vitamin D deficiency could be a confirmative sign of insulin resistance."

In another study maternal 25(OH)D concentrations were found to be inversely related to fasting glucose. Women in another study identified in a clinic as having gestational diabetes may actually have had unrecognized Type II diabetes all along. Additionally, people who are significantly overweight need higher intakes than others to maintain a normal blood level of vitamin D.

A side note: with so many young women being overweight now, and because some will actually have unrecognized Type II diabetes, it will be important to begin checking for diabetes much earlier in pregnancy (preferably before conception) in order to minimize the birth defects that are associated with poorly controlled diabetes in very early pregnancy.

For much more on this issue, please see "Aunt Cathy"s Guide to Nutrition: Top 10 Issues in Nutrition for Pregnancy"

Adequacy of Iodine and Selenium

Iodine is important for normal metabolism. The mineral selenium also appears to assist with moving glucose into the cells. **Both selenium and iodine are centrally involved in the production of the hormone thyroxine** by the thyroid gland. Thyroxine is responsible for setting one's basal metabolic rate (BMR) … the amount of calories used to run one's body in a resting state. Poor production of thyroxine contributes to weight gain and fatigue.

Selenium is also a part of a very important antioxidant in the body called glutathione peroxidase. More will be said about selenium later. Risk of selenium and/or iodine inadequacy depends on the soil upon which one's food was grown. **Recently, a resurgence of inadequacy of iodine has been documented in America and around the world because of changes in food patterns. It is likely that the documented increase in iodine inadequacy is also contributing to weight problems and fatigue that interferes with exercising.**

A brief history: Iodine deficiency remains the number one cause of mental retardation in the world and unfortunately it is coming back here in the USA.

We thought we took care of it in the 1950s by iodizing salt, but since then many things changed.

- When iodizing salt was established in the US, our mommies stayed home all day and made everything from scratch using iodized salt. The world is not like that now.
- Then, we told people "Don't Eat Salt!"
- Then we have developed fads for using exotic sea salts (most are not iodized.)
- We make (and applaud) well-intentioned decisions to "not have a salt shaker on the table!" not realizing that that iodized salt might be the only iodine our family gets.
- We do eat a lot of sodium … usually a whole lot more than we should … but most folks are unaware that all that sodium in commercially made foods is **not iodized**. So they think they get plenty of iodine and aren't worried about it. Wrong!

- The newest factor to contribute to risk of inadequate iodizing is the (otherwise terrific) movement to eat locally grown food. It supports the local economy, reduces diesel fumes across America and the food is fresher. However, all across the northern tier there is inadequate iodine in the soil, so plants that grow there simply don't incorporate it. And plants do not look or taste any different if they have iodine in them or not.

  In this situation, people don't get enough iodine if most of their foods were raised locally. That is why the northern US used to be called "the Goiter belt." (Goiter is an enlargement of the thyroid gland… the gland gets bigger trying unsuccessfully to make up for not producing enough thyroxine because of inadequate iodine. ☹)

- Many multivitamins (including prenatal vitamins) do not yet contain iodine because we thought we got enough from the iodized salt alone. Check labels.

- Some places in the world with the same problem, like Australia, are mandating iodizing bread products instead of salt as a new way to get iodine into people. We are not there yet, but our old 1950s "iodize-the-salt" solution is no longer working.

- This big but largely unrecognized problem affects everyone, but I am discussing it especially here because avoiding the weight gain/exercise-intolerance/fatigue/BMR-lowering component of iodine deficiency is especially important for people with Type II diabetes or trying to avoid it. The other very critical issue is the deleterious effects of iodine deficiency on fetal development, so iodine status in pregnancy (whether diabetes-related or not) requires an even closer look.

For more information on the return of iodine deficiency as a major public health problem, please see:
"Aunt Cathy's Guide to Nutrition: My Current Top Five Easy Ways to Improve Your Family's Nutrition" and
"Aunt Cathy's Guide to Nutrition: New Attention to an Old Problem: Iodine Deficiency in Pregnancy and Lactation

Exercise

Exercise is not a nutrient, but it does play important roles in metabolism. Besides affecting lean body mass (and so BMR,) and the calories burned from activity itself, it also has the capacity of helping to move glucose into the cells, so it is a cornerstone of prevention and also of treatment of Type II diabetes. Exploring this topic further is outside of the scope of this nutrient-focused paper, however.

[One other CB note: People are getting the impression from ads from folks selling things that pushing protein intake way higher than what one would normally use will induce muscle development and reduce body fat. Sounds good, and I get asked about this all the time. The answer, though, is that while adequacy of protein is very important, any extra protein consumed beyond one's requirements is not stored as muscle. It is converted to calories. (Yes … calories.) The thing that induces muscle building is exercise … which explains why if I ate an entire meatloaf with ten times my daily protein requirement and then napped all day on the couch, I would still not achieve that svelte, lean-mean-fighting-machine physique. But I keep on trying … I mean, it has to be true if I saw it on TV or read about it on the internet, right? Pass the meatloaf! ☺]

Part Two: Minimizing secondary damage from poor diabetes control caused by higher than normal production of free radicals

"Free radicals" are normal waste products of metabolism. They can injure cell membranes all over the body if they are not "quenched." "Antioxidants" are the substances that quench free radicals. **A much greater production of free radicals than usual is well known and documented among people with diabetes**. This is also true in many other diseases with disturbed metabolism, such as inflammatory diseases like MS or arthritis, or conditions with irregular fuel metabolism, such as obtaining a high proportion of calories from alcohol.

In diabetes, the greatest production of free radicals occurs in the people with the least well-controlled blood sugar levels. Such excessive production of free radicals causes serious injury to cells and tissues and it is **an important contributor to the development of diabetes-related complications** like heart disease, blindness, poor wound healing, kidney injury and nerve damage.

The many references on this topic are at the end of the paper, but here is just one as a sample:

Dietary antioxidant capacity is inversely associated with diabetes biomarkers: The ATTICA study. Nutr Metab Cardiovasc Dis. 2010 Feb 18.

"CONCLUSIONS: Although more prospective studies are required, the data presented support the view that **dietary modification towards higher consumption of antioxidants should be implemented in public health strategies**, **in order to better control glycemic markers in individuals, and prevent the development of diabetes at the population level"**

Here are some important points from a number of different studies:

- **There is evidence of significant slowing of the development of diabetes complications by assuring a generous intake of antioxidants.** "Generous" in this context usually means substantially more than the usual RDA-type levels (i.e. RDA, RDI, AI, etc.). As noted earlier, the RDA-type guidelines (by definition) are designed to meet the needs of the "healthy" population, so also by definition, they have little to say about the specific needs of people with serious metabolic diseases.

- **Vitamin and minerals with roles as antioxidants that have been studied quite a lot have included vitamins C and E, and the minerals selenium** (part of the important antioxidant "glutathione peroxidase") **and zinc** (as part of "Zn-Cu superoxide dismutase.") Our old friend **vitamin E** (alpha-tocopherol and its cousins like gamma-tocopherol, "mixed tocopherols" and tocotrienols) are back in the news with new evidence of their safety, and a lot

of new research into its potential to protect against a variety of diabetes related complications and other problems.

- **The ideal level of antioxidant vitamins is unlikely to be around the RDA-type levels**. As just one example, the author of a recent study concluded that: "The results suggest that megadose **vitamin C** supplementation [1-3 grams/day] may have a beneficial effect in diabetes mellitus patients on both glycemic control and antioxidant status. Thus dietary measures to increase plasma vitamin C may be an important health strategy for reducing the complications of diabetes for patients." Also, many of these antioxidant substances like tocotrienols or phytochemical pigments do not have RDA levels established at all because they are not identified as essential substances like designated essential vitamins and minerals.

- **Higher vitamin C levels in the blood, and higher fruit and vegetable consumption were recently reported to be related to decreased incidence of type II diabetes** in a 12 year study of over 20,000 people. They found a strong inverse association between plasma vitamin C level and diabetes risk. The "odds ratio" of developing diabetes in the top quintile of plasma vitamin C was 0.38 in a model adjusted for demographic, lifestyle, and anthropometric variables. That means that they divided folks up into 5 groups based on their blood vitamin C level, and the group with the highest level was less likely to have developed diabetes. **For every 100 who developed it in the group with the lowest vitamin C level, there were only 38 who developed diabetes in the highest vitamin C group.**

- In a similarly adjusted model, **the odds ratio of diabetes in the top quintile** of **fruit and vegetable consumption** was 0.78. (That is, people in the top fourth of intake were found to be about ¼ less likely to have developed diabetes.) They concluded that higher plasma vitamin C level and, to a lesser degree, fruit and vegetable intake were associated with a substantially decreased risk of diabetes.

- It is also being shown that **combinations of antioxidants** may provide more benefit than a generous intake of any one antioxidant alone. As an example, in a study with rats with diabetes, the treatment with a combination of generous vitamins C and E had a significant positive effect on decreasing diabetic damage to **learning and memory**.

- Another unique antioxidant that has been studied extensively in diabetes is **alpha lipoic acid, (also called thioctic acid**,) a B-vitamin-like substance made in the body from the essential fatty acid linoleic acid. It also has a role in energy production, as it is required in two places in the TCA cycle involved in making ATP energy out of fuel. It is very safe, and the level that is generally agreed as being **most likely to bring about positive effects in diabetes research has been at least 600 mg/day.** A randomized, double-blind, placebo-controlled, multi-center trial showed that alpha lipoic acid at an oral dosage of **800 mg/day for 4 months** significantly improved cardiac autonomic neuropathy in patients with type 2 diabetes. (Treat Endocrinol. 2004;3(1):41-52.)

  Since that time there has been considerable amount of research exploring alpha lipoic acid in relation to several aspects of health for people with diabetes. Here is just a sampling from the last few months (as of 4/13) describing the use of alpha-lipoic acid in neuropathic pain, kidney and liver disease related to diabetes, converting fuel into energy (ATP), and protecting against

oxidative damage from excessive free radical production associated with diabetes:

> The protective effects of α-lipoic acid on **kidneys** in Type 2 diabetic goto-kakisaki rats via reducing oxidative stress. Int J Mol Sci. 2013 Mar 26;14(4):6746-56. **Painful diabetic neuropathy** management. Int J Evid Based Healthc. 2013 Mar;11(1):77-9. Nutritional supplements and their effect on **glucose control**. Adv Exp Med Biol. 2012;771: 381-95.Whither pathogenetic treatments for **diabetic polyneuropathy**? Diabetes Metab Res Rev. 2013 Feb 5. Decreased O-GlcNAcylation of the key proteins in kinase and redox signalling pathways is a novel mechanism of the beneficial effect of α-lipoic acid in **diabetic liver**. Br J Nutr. 2013 Jan 14:1-12Alpha-lipoic acid preserves the structural and functional integrity of **red blood cells** by adjusting the redox disturbance and decreasing O-GlcNAc modifications of antioxidant enzymes and heat shock proteins in diabetic rats. Eur J Nutr. 2012 Dec;51(8):975-86. The protective effect of α-Lipoic acid on **mitochondria in the kidney** of diabetic rats. Int J Clin Exp Med. 2013;6(2):90-7. Alpha-lipoic acid **upregulates antioxidant enzyme ge**ne expression and enzymatic activity in diabetic rat **kidney**s through an O-GlcNAc-dependent mechanism. Eur J Nutr. 2012 Oct 12.

This is one of the three supplement substances that were described earlier as being more expensive, and it is sometimes available on line or over the counter in pills of 50 mg or more. A prescription for a higher dose pill would facilitate things. One area in which alpha lipoic acid has been most effective (and most studied) is in peripheral neuropathy (nerve pain) research, but it now looks very promising in several areas of diabetes complication research.

- Another potent antioxidant with other roles in energy metabolism is "**ubiquinone" – Coenzyme Q-10.** It is very safe and helpful in a number of applications, but it is also more expensive and a prescription might be helpful for this reason. CoQ10 treatment significantly improved deranged carbohydrate and lipid metabolism of experimental chemically induced diabetes in rats. The mechanism of its beneficial effect appears to be its antioxidant property.

- It has shown benefit in a variety of conditions involving altered metabolism (like diabetes but others as well) and specifically useful in eye health and muscle health and in parkinsonism. It also appears to be protective against some muscle side-effects of statin drugs.

  There is evidence that using **combinations of CoQ10 with carnitine and DHA** (a particular omega-3 polyunsaturated fat discussed later,) or with **alpha-lipoic acid, carnitine and biotin,** for example, may elicit more positive change than any one alone.

  > A combination of nutriments improves mitochondrial biogenesis and function in skeletal muscle of type 2 diabetic \ Goto-Kakizaki rats. PLoS One. 2008 Jun 4;3(6):e2328.
  > Improvement of visual functions and fundus alterations in early age-related macular degeneration treated with a combination of acetyl-L-carnitine, n-3 fatty acids, and coenzyme Q10. Ophthalmologica. 2005 May-Jun;219(3):154-66.

- In early 2013 there has been promising information popping up about apossible **benefit of supplemental CoQ10 in diabetic neuropathy**. Here are three reports on that topic:

  > Diabetic Neuropathic Pain Development in Type 2 Diabetic Mouse Model and the Prophylactic andTherapeutic Effects of Coenzyme Q10. Neurobiol Dis. 2013 May 16.
  > Prophylactic and antinociceptive effects of coenzyme Q10 on diabetic neuropathic pain in a mouse model of type 1 diabetes. Anesthesiology. 2013 Apr;118(4):945-54.
  > Coenzyme Q10 prevents peripheral neuropathy and attenuates neuron loss in the db-/db- mouse, a type 2 diabetes model. Proc Natl Acad Sci U S A. 2013 Jan 8.

- **A generous intake of a variety of plant pigments (natural coloring agents) like carotene in carrots, lycopene in tomatoes, lutein in spinach, zeathanthin in corn, and anthocyanin, the blue/red color in blueberries, are being shown to be hugely beneficial in decreasing the complications rate in people who have diabetes.** They have been found to be VERY potent antioxidants – some (like Lycopene) have shown antioxidant protection at 200 times the antioxidant potential of vitamin E! These fruits and vegetables also tend to be "nutrient dense" f**o**ods ---lots of nutrients per calorie provided – and that is a definite benefit as well.

Research into the control of free radical production (or "quenching" them after they are formed,) has shown antioxidants to have promising roles in kidney health, circulation to the eye, lens and retinal health, circulation of blood to the extremities, wound healing, peripheral neuropathy, erectile dysfunction, the development of gestational diabetes, and birth defects.

It's time to seriously consider adding antioxidant protection to our standard protocols

For a review and recommendations for these vitamins, minerals and the plant pigment antioxidants, please see "Aunt Cathy's Guide to Nutrition: Eye Health" and others noted earlier.

Part Three: Reducing the severity of inflammation that has been shown to be associated with diabetes

Inflammation is now being recognized as a significant contributor to heart disease and to tissue damage in general. For example, inflammation inside the blood vessels is an important reason why cholesterol sticks to the arteries and builds up to block circulation. **Poor control of diabetes especially results in an increase in inflammation.**

Besides being a special problem in diabetes, this has been found to be true for many conditions that have an inflammatory component, such as rheumatoid arthritis, MS and inflammatory bowel disease. Inflammation is a normal part of the immune system, but excessive inflammation is injurious. Sometimes inappropriate messages can be sent to cause tissues to be inflamed more than they should. The inflammation results from any chronic disturbances in normal fuel metabolism, such as that seen with diabetes, autoimmune diseases and excessive alcohol consumption.

Two particular families of fats (called the omega-3 family and the omega-6 family) are used to make the inflammatory agents that are part of our immune system. The inflammatory agents are called prostaglandins. When prostaglandins are made from an omega-6 fat they are way more inflammatory than when they are made from an omega-3 fat.

Altering the ratio of omega-6 to omega-3 polyunsaturated fats has been shown to be an important step in achieving health for most Americans, and especially so in diabetes. Americans tend to eat about 10 grams of omega-6 fat for every gram of omega-3 fat. That is, we have about a 10-to-1 ratio of "sixes-to-threes."

The demonstrably heart-healthy "Mediterranean diet" provides about a 4-to-1 ratio, and most healthy people would clearly benefit from change in this direction. For people with diabetes, MS, and other hyper-inflammatory conditions, it has been suggested that a ratio of two-to-one may provide additional benefit.

One reason why our intake of omega-6 fats is so high is because many vegetable oils that we use a lot, like **corn oil, are almost all omega-6**. Soy oil has a little omega-3, canola has more, and flax oil has even more. However, the majority of our foods are not made with canola oil, much less flax oil.

What vegetable oils do they use in the Mediterranean Region? They use a lot of **olive oil and peanut oil** instead of other vegetable oils. Olive and peanut oils both are high in MONO-unsaturated fat … a type that is neither omega-3 nor omega-6 and which is **not** involved in making inflammatory prostaglandins. **If we were to replace a significant amount of our usual omega-6 dietary fat (like corn oil) with monounsaturated olive or peanut oil, though, it would make a very substantial change in the omega-6 to omega-3 ratio**.

These "heart healthy" monounsaturated and omega-3 fats are well represented in **nuts and peanuts.** This another good reason to eat these foods which are also such excellent sources of magnesium and chromium. **Nuts and peanuts are real winners for people with diabetes.**

The omega-3 and omega-6 vegetable oils can usually be converted in our bodies to the forms of fat used to make prostaglandins. These are the fats called EPA (the omega-3 one) and ARA (the omega-6 one.) We have always thought that if people ate the vegetable forms they could easily convert them to the EPA and ARA forms. **However, another important new discovery is that many people are unable to efficiently convert vegetable essential fats to these critical EPA and ARA fats that are used to make a variety of important substances.**

We can eat ARA omega-6 fat "ready-made" in meats, and "ready-made" EPA omega-3 fat in fish. However, if people who are unable to efficiently make these fats from vegetable oils eat meat and little fish, then ALL the fats they have available to make prostaglandins are the really inflammatory omega-6 type.

Additionally, we are supposed to make an important fat called **DHA** out of EPA. DHA is a primary fat of a healthy functional brain. Adequate DHA appears to be important in maintaining cognition, in retina health in the eye, and in mood. (This was mentioned above in the antioxidants –in-combination-with-stuff discussion.) **This means that some folks are much more dependent on their diet to assure adequacy of both of these key substances.** This defect in producing EPA, ARA and DHA fats from the forms in plants may be a significant contributor to inappropriate hyper-inflammation and so it is very important in diabetes especially.

This discovery is behind recommendations that **"ready-to-go" sources of these fats, like fish or fish oil supplements** have important health benefits for minimizing complications of diabetes, risk of cancer and heart disease, and in promoting a healthy outcome of a pregnancy. **The American Heart Association recommends eating fatty fish twice a week or taking a 1000 mg daily of fish oil (which contains ready-made EPA and DHA.)** Some folks are advised to take more … for example some people who have high triglycerides in their blood respond well to 2-4 times that amount. [Check with your doctor before using the higher doses, especially if you are taking aspirin or a medication to block blood clots.]

Omega-3 and Omega-6 Memory Tricks:

Here's an easy way to remember which family of polyunsaturated fat is which in the ratios of omega-6 and omega-3 fats, and also which family produces the most inflammatory prostaglandins and also the strongest blood-clotting messengers (thromboxanes):

Just remember this "six is always bigger than three."

The omega-**6** fat family produces the more inflammatory prostaglandins than omega-**3** fats do.

The omega-**6** family also produces stronger clot-promoting thromboxanes than omega-**3** fats do.

No matter whether the ratio of these fats in one's diet is expressed (that is, whether it is described as 4-to-1 or 1-to-4, the bigger of the two numbers will always represent the omega 6 fat!

(Thank heaven for these memory tricks or I couldn't keep this stuff straight!)

For more information and specific recommendations, please see:
"Aunt Cathy's Guide to Nutrition: Omega-3 Fats and Other Lipids."

Part Four: Helping to stabilize excessive fluctuations of blood sugar

Some individuals have a problem with widely fluctuating glucose levels in spite of following the diet, exercise and medication program very carefully. This used to be called **"brittle"** diabetes. In the past, these people were sometimes suspected of "cheating" on the diet, since health care professionals could not explain the phenomenon. Unfortunately, this accusatory response is still common in some settings.

Increasing knowledge of the effects of other **differences in types of carbohydrates consumed and factors affecting absorption** have led to some changes in our globally accepted recommendations. For example, the carbohydrate called sucrose (regular table sugar) is not looking automatically bad, and the carbohydrate in the form of high fructose corn syrup is looking a bit worse. The high-fructose corn syrup issue is a matter of considerable discussion just now and the jury is still out.

But clearly, some of the issues are related to whether one is taking in these forms of carbohydrate alone (as "empty calories" like in soft drinks) or whether they are eaten as a part of a meal that provides key nutrients involved in metabolizing the carbohydrate taken in.

The popular movement toward **whole grains/whole foods** has improved the **nutrient-to-carbohydrate ratio**, resulting in an improved magnesium and chromium intake, as described above. Increasing the intake of **soluble and insoluble fiber** in foods has positive effects, although we are quick to assume that it is the **fiber** in a "high fiber diet" that does all the good, when a high fiber diet clearly alters many other nutrition parameters as well. (If it were just the fiber providing the benefits, just taking some fiber supplements would do the job; but we only see the great results when people eat nutrient-dense foods that are naturally high in fiber.)

In the not too distant past, the prescribed diet for diabetes was pretty strict about whether the carbohydrate in a food was sucrose or glucose or lactose or fructose or starch, so people were advised to eat very specific amounts of very specific food groups at each meal. Swapping foods between different food groups was discouraged because it was thought that the carbohydrate would probably be used very differently in the body.

However, because we can now easily check people's blood sugar, we have learned that this distinction of carbohydrate type is much less important. We now commonly use a system of **counting grams of carbohydrate in a meal or snack** instead of the more rigid (and much less effective) "exchange system" for meal planning. This has been shown to make managing diabetes much simpler and people are much more successful using this approach than the older version.

Now we also no longer perceive **apple juice** and an **intact apple** to be nutritionally identical in regard to the diet for diabetes, even if the number of grams of carbohydrate are identical and they are both from the old "fruit group." Other substances in the apple, like pectin (a type of fiber) for example, make a difference in how it affects our blood sugar and also affects absorption of dietary cholesterol to some degree.

The same is true of enriched white bread and whole grain bread. Whole grain offers many important nutrients like magnesium, chromium, fiber and vitamin E that are not provided in white bread. That means that compared with nutrient-dense whole grain bread, white bread is more of an "empty calorie" food, and therefore less helpful in managing diabetes.

"Enriched" flour" means that the germ (the "baby plant") and the bran (fiber) of grains is removed and only four nutrients are replaced: vitamins 1, 2, and 3 and iron. That's all. They do not replace the magnesium, chromium, or other nutrients so it is clear that "whole grain" flour

is nutritionally very superior to "enriched." This is confusing to many people because the word "enriched suggests that this is the superior and more nutritious product. That's why I teach my patients to think of "enriched" as meaning "UNriched" … meaning that they took nutrients away and did not add them back.

Improved understanding of the fate of specific carbohydrate-containing foods as described above is lending some light to this puzzle, resulting in exploration of concepts such as **genetic differences** in people's metabolism, and the "**glycemic index**" of foods and meals. A quick definition: The Glycemic Index is a comparison of the effect on blood sugar from eating 50 grams of carbohydrate from a food compared with eating 50 grams of carbohydrate as plain glucose. ("Glyc- = glucose or sugar; "-emic" = in the blood.)

A food that is described as having a **"low glycemic index"** is one that will cause a **smaller rise in blood sugar** in response to eating it than would be produced by plain glucose. The lower blood sugar response can help decrease injury to the circulatory system caused by frequent high peaks of blood sugar. In general, and for several reasons, more complex "nutritionally-dense" foods will usually have a lower glycemic index than "empty calorie" foods like regular soda. However, the glycemic index is just one more tool to use to manage blood sugar in diabetes. A complete evaluation of this research is too large to include here.

Carnitine: A player in energy metabolism with particular importance in diabetes (and also a potent antioxidant)

One other important piece of the puzzle of large fluctuations in blood sugar is less well known among health professionals, so I will address it here in some detail. It has been found that relative inadequacy of a substance called "carnitine" exists in some people with diabetes and in others.

Additionally, there is increasing evidence that supplemental carnitine can be of significant help in prevention or improvement of a number of complications of diabetes. For example, in a recent study **the average serum free-L-carnitine levels in patients with diabetes who had complications was almost 25% lower than in the patients with no diabetes-related complications.** On the basis of the study results, the researchers suggested that "carnitine supplementation in diabetic patients, especially in patients with diabetes complications, might be useful."

Relative carnitine inadequacy has also been found to be a factor in a number of other health conditions, with special importance in kidney disease, high triglycerides, obesity, poor wound-healing and poor eye health … all of which are risk factors related to diabetes.

Normally, one makes an adequate amount of carnitine in the liver, the brain and the kidney from methionine and lysine (two essential amino acids,) and additionally carnitine may be obtained

from meat. (That's why it is called carnitine – it comes from "carne" which means meat in Latin and Spanish. Memory device: think of "chili-con-carnitine.") **Individual differences in requirements, diet, genetic carnitine production, and the use of certain medications (like valproic acid – "Depekote") for seizure control) can result in a relative inadequacy.**

Carnitine is a key component of a cellular transporter called "carnitine palmitoyl transferase" which allows fatty acids to cross the mitochondrial membrane to be used as fuel to make ATP.

Because inadequate carnitine impairs one's ability to move fatty acids into the mitochondrial membrane to be used for fuel, **symptoms can include lethargy, significant weight gain and obesity, poor exercise endurance, poor muscle tone, heart muscle damage (cardiomyopathy,) greatly fluctuating blood sugar, excessive appetite, elevated triglyceride levels in the blood, and if applicable, "breakthrough seizures." and increased risk of liver toxicity from use of certain medications.**

The key feature that affects blood sugar is the fact that there are times when we normally switch to burning primarily fat as a fuel source in order to spare glucose for the brain – sort of like having "dual heat" in your home: gas and wood. The brain does not burn long chains of fat like other tissues can do. It can use only tiny bits of fuel like glucose molecules. **But if fat is unable to be burned by other tissues because of carnitine inadequacy, a person will have to burn glucose that he/she really can't afford to burn.** [It is like living in North Dakota in the winter and running out of fuel. You have to burn something to stay alive so you start burning the furniture!]

This unusual need to burn carbohydrate because fat cannot be used can result in an extremely low blood sugar. This, in turn, can trigger release of glucose as glycogen from the liver, causing blood sugar to rebound up high. At night when we are fasting, we usually switch over to burning mostly fat. But poor carnitine status can make that effort fail. As a result, people can experience a low blood sugar in the night and it may not even wake them up. While they sleep, a rush of "rescue" glucose from the glycogen stores in the liver will result in people waking up with surprisingly high blood sugars.

Even more seriously, if no glycogen is available to provide the glucose to correct it, the low blood sugar can be injurious, and even life-threatening. This very serious kind of low blood sugar is especially a problem in people who use insulin as part of their diabetes management.

People using insulin who experience unexpected low blood sugars or unstable blood sugars should be sure to check out the possibility that inadequate carnitine is contributing to the problem. In some studies people with Type-1 diabetes have been found to have low carnitine levels in spite of our usual ability to simply make enough ourselves. It is a bit like insulin in that way … people usually simply make enough ourselves and they do not need to take insulin. But as you are aware, some folks DO need to take it even if others do not. It may be the same with carnitine.

And, it may also turn out to be similar in another way as well. Many autoimmune diseases

involve the decreased production of a key substance that one's body used to make and then quits making. In Type I diabetes, it is insulin. In pernicious anemia it is carnitine. In parkinsonism it is dopamine. Failed production of thyroxine hormone by the thyroid gland is another. This is just my speculation here, but it occurs to me that carnitine production may be similarly affected in genetically susceptible people, and they simply begin to make less.

What started me to think about it was the following eye-opening 1989 report of 54 children with Type I diabetes who were screened for this and about 1/4 were found to be carnitine deficient and about half had other evidence of carnitine inadequacy.

Now, how many children in any group would one expect to have inadequacy or deficiency of a substance one easily makes oneself? The answer to that should be zero . But then why was this a very common problem found in these children with diabetes? Maybe the ability to readily make adequate carnitine is also something that can be lost as a consequence of autoimmune disease. Just thinkin' …. This has not been studied yet as far as I know. Here's the abstract of the report … I am including it here in its entirety because it is such an important finding but it has received amazingly little attention in spite of my going on and on about this all the time.

Relative carnitine insufficiency in children with type I diabetes mellitus.

Winter SC, Simon M, Zorn EM. Am J Dis Child. **1989** Nov;143(11):1337-9.

Department of Pediatrics, University of California, San Francisco.

- Recognizing the similarity of type I diabetes mellitus to inborn errors of metabolism that have responded to carnitine therapy, we initiated a study of **54 children with type I diabetes mellitus.**
- Examining a fasting blood sample for levels of carnitine, glucose, and glycosylated hemoglobin A1c, and a urine sample for levels of ketones and glucose, we found **13 children were deficient of free carnitine (less than 20 mumol/L) and 30 had elevated acyl carnitine levels (greater than 11 mumol/L)**.
- Statistical tests confirmed a significant difference between the diabetic population and normal population for reduced free carnitine, elevated acyl carnitine, and an elevated ratio of acyl carnitine to free carnitine.
- Also, a significant correlation was found between the levels of urine glucose and ketones and the level of acyl carnitine.
- **Our data indicate that carnitine deficiency and relative insufficiency may be an overlooked component in the management of diabetes.**

Interestingly, since we now know that carnitine lab values underestimate the number of people with carnitine problems (because they do not reflect all the tissues likely to be affected,) the number of children in the study above found to have inadequate or deficient carnitine likely is an underestimate of the prevalence of this problem as well.

Since that report was published, I have worked with several children with Type I diabetes whose blood sugar was very hard to manage in spite of the family doing everything right in terms of insulin shots, carbohydrate consumption and exercise. They had frequent episodes of very low glucose. A trial on carnitine worked amazingly well for doing away with the troubling low blood sugar episodes in these children.

Bottom line: if a person experiences this kind of yo-yo blood sugar pattern with no identifiable cause, a trial on carnitine would be a good idea. Getting a blood level is not as helpful (even though they did it in the study), because if it is low, then one would prescribe some carnitine. However, even if it is "normal," the blood levels do not necessarily reflect adequacy in the other tissues like the heart muscle. Carnitine deficiency is well known to result in severe cardiomyopathy. **So, if there are suggestive symptoms at all, one would want to do a trial anyway**.

For health professionals who want to do a trial for a patient who has symptoms as described, the usual dose in children is 50-100 mg/kg/day divided into 3 doses (just because of the potential GI effects of high osmolality,) with a maximum usually 3000 mg/day. There is no reason not to start at the 100 mg/kg level in this situation, as the working presumption of the trial is that tissues may be depleted because the child has symptoms suggesting it.

Starting too low and stopping too soon may mask a true effect. When I do a trial, we monitor selected symptoms for change, and usually the trial would be continued for at least a month even in the absence of symptom relief, in order to be sure that any deficiency would have been corrected to the point of detection of symptom change.

The other group of people with diabetes for whom a trial on carnitine is a very reasonable step is those who are extremely obese.

They may also have very poor exercise tolerance and a variety of other symptoms, and we have a tendency to assume that the "lazy couch-potato lifestyle" is a choice that causes their obesity. While that is always a possibility and often quite contributory, **remember that having inadequate carnitine can cause one to be both very fat AND be a "couch potato"** … which is one reason why simply telling some folks that they should get off the couch and exercise more is not always helpful.

Some folks simply can't fuel their leg muscles to do endurance-type exercise if they can't get the preferred fuel of muscles (fat) into the mitochondria to burn it. I have had many patients who fit this description. Rather than blame them for being lazy and obese, a bit of compassion is in order … and a trial on carnitine to see if it helps. I have had MANY patients for whom it did amazing things to allow them to burn fat for fuel, exercise longer, feel stronger, lose weight and not feel starved all the time. Life changing in many cases. No effect at all in others.

Carnitine is the other supplemental substance that could requires a prescription for a trial and which can sometimes be expensive. Expense is quite variable, however. Insurance will sometimes pay for the prescription kind but not pay for over-the-counter products. Both prescription and over-the-counter products are available, and more carnitine supplements are becoming available over-the-counter for lower prices, especially on-line.

When considering the cost of a trial on carnitine, it is useful to consider the cost of carnitine supplementation against the cost of failing to identify and correct a carnitine insufficiency problem: Consider the risk to your patient in damage from blood sugar vacillation, impairment of energy production, compromised endurance, heart muscle injury, hypertriglyceridemia, excessive drive to eat, the cost and consequences of bariatric surgery that might have been avoidable, and the social consequences of being obese in America.

I am especially suspicious of a carnitine metabolism problem when a person has had bariatric surgery but who somehow manages to gain the weight back and may even undergo a second surgery. These folks often suffer considerable derision ("You'd think she would have more self respect than to let herself gain all that weight back again!") I think we should be less quick to be insulting and more quick to explore the very real possibility of a carnitine-related metabolic problem. Just sayin'.

Please see my more detailed paper on this topic:
"Aunt Cathy's Guide to Nutrition: A Short Carnitine Discussion That Might Be Helpful."

Part Five: Minimizing risk of developing other threats to health and well-being, including some conditions that are known to be exacerbated by diabetes.

There are a number of conditions for which people with diabetes are at increased risk. These include **elevated homocysteine levels, depression, cardiovascular disease (including stroke), leg cramps, and general neuropathy.**

Some are related to **medications,** such as the effect of metformin (Glucophage) on vitamin B12 level. Other common problems that can cause trouble for people with diabetes (or anyone) are these: Proton pump inhibitors for **GERD** may also contribute to poor absorption of vitamin B12 from food, and seriously obese people with diabetes may have had (or plan to have) **gastric bypass (bariatric) surgery**, which can also compromise vitamin B12 absorption and absorption of many nutrients. Old age alone can also impair absorption of vitamin B12 from food sources because of a common loss of stomach acid with aging. These situations will need a closer look than usual among people with diabetes.

At present**, follow-up of micronutrient status in people who have had bariatric surgery**

has not been consistent or studied adequately. Often the only outcomes evaluated are losing weight and keeping it off, and any benefits seen in terms of cholesterol, blood pressure and diabetes. While positive changes in these health conditions are extremely welcome**, the important potential problem of relative micronutrient inadequacy will not show up until a few years after the surgery**. Some are surprising and quite severe, such as permanent neurologic damage from copper deficiency … a condition rarely seen but now showing up among some of the post-bariatric surgery populations.

For many patients, risks associated with potential multiple micronutrient deficiencies are not assessed or followed at all. Some programs just note in the chart that the patient was told to take a multivitamin with minerals. There is huge variability among these products in terms of actual content (from clearly inadequate products with very minimal numbers of micronutrients to more balanced products, but none is truly "complete" even for the healthy population.) The forms and the cost are other variables. But my observation is that one of the most common mistakes in failing to prevent micronutrient deficiencies in this context is the simple assumption that "normal" RDA/RDI/AI levels of nutrients are at all likely to be sufficient for this population. There are so many reasons why they will not do the job.

An additional concern is a lack of long term monitoring of these issues for bariatric surgery patients. Interestingly, many people discontinue whatever supplementation regimen they were prescribed after a few months or years, and apparently this is rarely recognized prior to discovering a serious problem.

Overweight people anticipating bariatric surgery are often presumed by the public and also by health professionals as being "well nourished." However, their size is really only a reliable indication of generous amounts of calories stored, not micronutrient or even protein adequacy. **Many studies have shown that the population undergoing bariatric surgery are often in very poor micronutrient status even before the surgery is performed, so impaired absorption of nutrients after surgery is even more serious.** Recovery from the surgery itself can also be compromised.

High triglycerides are known to be a risk factor for **stroke** in people with diabetes especially. Three of the nutrition factors described earlier are associated with correcting high triglycerides: assuring an adequate intake of chromium as **chromium** picolinate, maintaining a **ratio of omega-6 to omega-3 fatty acids** in the neighborhood of 4:1, and correcting relative **carnitine inadequacy**. All are potentially quite beneficial. High triglycerides are often seen in **renal** failure as well … another diabetes-related problem. This may also have a carnitine connection, as the kidney is one of the three places in the body where we do make carnitine.

Inadequate **vitamin B6 and magnesium** appear to be involved in diabetic **neuropathy**, and also in **heart disease and leg cramps.** Vitamins **B1,B6, B12 and folic acid** are critical for preventing **elevated homocysteine**, a major contributor to stroke (or at least a marker for increased risk of stroke,) and possibly to alzheimers. Folic acid inadequacy is associated with **cancer** of the breast, prostate and colon. It is also a contributor to **depression** and inadequacy also makes therapeutic interventions for depression (such as SSRIs) work much less well. Chronic **antibiotic use and alcohol abuse** both impair folic acid absorption.

Certain groups of people have a genetic problem that requires special attention to folic acid. For example, among some people of Irish heritage, the MTHFR gene has been discovered that contributes to higher rates of certain birth defects, depression and alcoholism related to problems associated with folic acid metabolism.

Happily this particular problem is significantly less of a concern since 1998 when folic acid in a well-utilized form was added to grain products in America. In the five years after starting this supplementation compared with the 5 years before supplementation, neural tube birth defects were cut in half, and stroke occurrence was cut by 10% across the USA. Wow! This illustrates that there is greater variety in the needs of individuals for nutrients in general than was assumed in the past, and it is clear that the assumed "adequate" intake of a nutrient for public health estimates is not the same as an individual's optimal intake.

(Please see "Aunt Cathy's Guide to Nutrition: Folic Acid" and "Aunt Cathy's Guide to Nutrition: Vitamin B12" for details, references and recommendations.)

Some studies with **vitamin B6 have used levels of 50 to 100 mg to achieve a desired effect in people with diabetic neuropathy.** This is much higher than the RDA-type level of about 2 mg, but not anywhere near a level that might be a problem. This is especially true if one is hypothesizing that their vitamin B6 requirements are in fact higher than the requirements of other people. Vitamin B6 (pyridoxine) is a cofactor with magnesium in a large number of metabolic pathways, including all amino acid metabolism, all energy metabolism, and all nerve function. They work together so often in nature that some attention is now being paid to evaluating the effectiveness of using either of these nutrients in combination with each other to bring about a particular metabolic effect.

Niacin (vitamin B3), riboflavin (vitamin B2) and thiamine (vitamin B1) and biotin are all being studied. Biotin is less familiar to most people but it is looking to be extremely interesting in a variety of metabolic conditions including diabetes. For example, biotin has a key role in gluconeogenesis … making new glucose out of amino acids.

Some examples of some newer reports exploring the role of biotin in diabetes, including biotin combined with other nutrients in diabetes research:

Effects of biotin supplementation in the diet on insulin secretion, islet gene expression, glucose homeostasis and beta-cell proportion. J Nutr Biochem. 2013 Jan;24(1):169-77.

Anti-diabetic activity of chromium picolinate and biotin in rats with type 2 diabetes induced by high-fat and streptozotocin. Br J Nutr. 2012 Dec 5:1-9.

Effects of biotin deficiency on pancreatic islet morphology, insulin sensitivity and glucose homeostasis. J Nutr Biochem. 2012 Apr;23(4):392-9.

Pharmacological concentrations of **biotin** reduce serum **triglycerides** and the expression of lipogenic genes. Eur J Pharmacol. 2010 Oct 10;644(1-3):263-8. A combination of nutriments improves mitochondrial

biogenesis and function in skeletal muscle of type 2 diabetic Goto-Kakizaki rats. PLoS One. 2008 Jun 4;3(6):e2328. [CB note: They used biotin, alpha-lipoic acid, carnitine and nicotinamide (vitamin B3)] **Their conclusion:** All of these effects of mitochondrial nutrients are **comparable to that of the antidiabetic drug, pioglitazone**. In addition, the treatment with nutrients, unlike pioglitazone, **did not cause body weight gain**.

Chromium picolinate and biotin combination reduces atherogenic index of plasma in patients with type 2 diabetes mellitus: a placebo-controlled, double-blinded, randomized clinical trial. Am J Med Sci. 2007 Mar;333(3):145-53.

B vitamins are generally among the least toxic vitamins – B6 is the only one documented to cause problems with high amounts, and that was only in the most sensitive people, and never at levels below 200 mg/day. Most people who experienced any tingling in the forearms (the symptom in question) were taking over 500 mg chronically. Some people find it safe, cheaper and convenient to take a "B-**100** Complex" tablet along with their general multivitamin with minerals, instead of trying to tinker with a lot of individual B vitamins.

As always, check with your physician about applying any of these ideas to your own health circumstances. But it may be helpful to share this handout with him/her with the section marked that you are asking about. Providing it before your visit to discuss it will give your physician a chance to review the research studies I have listed.

Much of this is quite new, and there is a lot of research out there to try to keep up with. They have a boatload of other diabetes-related research to study … I keep up just on some nutrition issues. Additionally, there is a lot of wacky stuff out there that health professionals are aware of. It can be overwhelming, so there can be a tendency to "Throw the Baby out with the Bathwater." The purpose of this review is to help sort out the real "babies" in that bathwater that are very safe and that can be very helpful for improving the health of patients.

It has recently been found that serious chronic conditions like **celiac disease, hemochromatosis and certain thyroid problems are much more prevalent in the general population** than has been previously thought. Health professionals would do well to keep these conditions in mind especially among their patients with diabetes. **There are some links between hemochromatosis and diabetes, between hemochromatosis and celiac disease, and between diabetes and celiac disease**. For example, according to a recent report, "higher iron stores (reflected by a significantly elevated ferritin concentration and a lower ratio of transferrin receptors to ferritin) are associated with an increased risk of type 2 diabetes in healthy women independent of known diabetes risk factors."

Thyroid health problems may be related to **diabetes**, but as described earlier, there is also a newly recognized recurrence of **iodine deficiency** in the US resulting in goiter (enlarged thyroid), weight gain and loss of energy. Additionally, the American Medical Association recommends that all women over 40 have their thyroid function checked annually. There are **age-related changes** that can make women's (especially) thyroid hormone gradually become inadequate.

Celiac disease, an autoimmune disease that is triggered by exposure to gluten in wheat, rye and barley is genetically more common among people with Type I diabetes. At our clinic, we

screen children with Type I diabetes for hidden celiac disease with a "Tissue Transglutaminase" blood test. We also check children with **Down syndrome**, as they have an increased risk of both celiac disease and Type I diabetes. They have also been found to produce way more free radicals than other people so they need more antioxidants than usual. They also sometimes benefit from carnitine supplementation, which can be helpful in particular with low muscle tone.

In addition to the gastrointestinal problems that are the most commonly recognized symptoms of celiac disease (which can certainly affect absorption of many nutrients,) it is now recognized that there are other less well recognized neurologic and dermatologic manifestations of celiac disease, and they could be complicating the recognition and treatment of neurologic and skin disorders thought to be primarily related to diabetes. Celiac is also by nature inflammatory and it also contributes to increased production of free radicals. **Crohn's disease** (Inflammatory Bowel Disease / IBD) now appears to be another inflammatory autoimmune "cousin" to watch out for.

The increased risk of **developing "pernicious anemia" (an auto-immune type of very injurious kind of anemia) is also a threat in people with Type I diabetes**. It may not be recognized because the neurologic symptoms of B-12 deficiency are often mistakenly attributed to diabetes-related neurologic complications. The development of pernicious anemia was recognized in an adult friend who has Type I diabetes, and she suffered terrible neurologic pain, some of which continues to be a problem even after the vitamin B12 deficiency was resolved by administering it as a shot.

This is a big deal because it is so harmful if unrecognized and uncorrected, so I am going to re-cap this here a bit. As noted earlier, in addition to developing the autoimmune diseases called pernicious anemia that blocks intestinal absorption of vitamin B12, there are **<u>other</u> factors that can interfere with vitamin B12 deficiency as well with serious consequences unless adjustments are made. These include:**

* certain gastrointestinal diseases or intestinal surgery (e.g. gastrectomy, bariatric surgery, inflammatory bowel disease)

* age-related loss of stomach acid,

* the use of medications that block acid production called PPIs – "proton pump inhibitors" for GERD (Gastro-Esophageal Reflux) and

* the use of the diabetes drug metformin (Glucophage.)

For more information, please see "Aunt Cathy's Guide to Nutrition: Hemochromatosis" "Aunt Cathy's Guide to Nutrition: Vitamin B12 " and "Aunt Cathy's Guide to Nutrition: Other Nutrition Issues in Celiac Disease."

7. Discovering the emerging miscellaneous effects of a variety of phytochemicals on the diabetic state.

Around the world and in the US quite a number of plants and their various "phyto-chemicals" ("plant chemicals") are being investigated for their ability to influence health in diabetes and in general. The phytochemical pigmanets (naturally occurring coloring agents in plants) were described earlier as being very important as potent antioxidants that protect against free radical damage. These are important for everyone's health, but especially so for people with diabetes.

However, there are many other (non-pigment) plant chemicals that are being invesitigated as being of possible benefit in diabetes in a variety of ways. This is a huge area of research and it is far outside of the scope of this paper. In general, the properties being looked at are more of a pharmaceutical nature than a nutrition nature, even though some of the plants have a food connection. The food-related substances include certain herbs and spices like cinnamon, curcumin, ginger, ginseng and other substances in fruits and vegetables like pomagrate, stevia, and many, many more. In most cases the research is in early days in terms of drawing any conclusions about form, efficacy, and safety.

But if you would like to get a taste of the kinds of studies being undertaken, simply log on to Public Medline (www.pubmed.gov) and pick the box at the right that says "for health professionals." Then check the "Medline" box. You will see a box at the top of the screen in which you should enter search terms such as: "diabetes plant" or "diabetes herb" or "diabetes phytochemical."

You will find reports like this: "Coffee consumption and risk of type 2 diabetes mellitus: an 11-year prospective study of 28,812 postmenopausal women" in the journal Arch Intern Med. 2006 Jun 26;166(12): 1311-6. The conclusion of this study was that "Coffee intake, especially decaffeinated coffee, was inversely associated with risk of type 2 diabetes mellitus in this cohort of postmenopausal women." That is, the coffee-drinkers had LOWER risk. More recent studies have supported this finding. It is notable because assumptions have long been that something we like so much has GOT to be really bad for you. Personally, I am not too surprised that coffee may have some health benefits … Hey! It's a BEAN! ☺

Some References (…these are just the ones from 2010 or earlier … there are lots more that are newer, but I am leaving these here from when I wrote the very first versions of this paper so you can see that there is a TON of research and it is piling up more and more every day … and that I am not making this stuff up!)

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