YOU Can Prevent and Cure Gestational Diabetes, and Lower Your Child’s Risk of Autism!

- Xanthurenic acid (XA)—a tryptophan metabolite—is high in serum in gestational diabetes
- Xanthurenic acid binds insulin, impeding its action
- Vitamin B₆ lowers xanthurenic acid levels to normal
- In two 1970s research studies, 86% and 100% of women with gestational diabetes normalized their blood sugar in two weeks by taking vitamin B₆
- Gestational diabetes increases autism risk for the unborn child; vitamin B₆ eliminates that extra risk

No, not kidding! If you’re a pregnant woman who never had any sort of diabetes before you became pregnant, and developed high blood sugar only after becoming pregnant (gestational diabetes), you can safely eliminate it all by yourself within two to three weeks. You might have the remedy at home already! If not, a trip to your favorite natural food store, compounding pharmacy, or maybe even an online order from the Tahoma Clinic Dispensary or other online source will equip you to eliminate gestational diabetes almost every time.

Of course, if you’re a man, you’ll never have this problem. However, your wife, sister, or daughter might, so keep this information in mind in case it’s ever needed.

One of many reasons gestational diabetes should be eliminated as rapidly as possible was discovered recently, and published in the Journal of the American Medical Association just last year. What is this reason? Autism!

Here’s what the researchers wrote:

Exposure to maternal gestational diabetes mellitus diagnosed by

26 weeks’ gestation was associated with increased risk of autism spectrum disorder in offspring.¹

Yes, that’s extra risk for the child of developing autism!

But even though blood and urine sugar is higher than normal in those with gestational diabetes, gestational diabetes is not type 2 or even type 1 diabetes mellitus! To make this point clear to everyone, gestational diabetes should be renamed “diabetes mellitus xanthurenica” to clearly identify its cause: excess serum xanthurenic acid. When this renaming occurs, even conventional medicine might quit treating gestational diabetes with a “diabetic diet” and insulin and might instead actually treat the cause!

Here’s what WebMD says is the cause of gestational diabetes:²

During pregnancy, the placenta . . . releases hormones that help your baby grow. Some of these
YOU Can Prevent and Cure Gestational Diabetes!

Continued from previous page

make it harder for your body to make or use insulin. This is called insulin resistance. . . . To keep your blood sugar levels steady, your pancreas has to make . . . as much as three times more [insulin] than usual. If it can’t make enough extra insulin, your blood sugar will rise and you’ll get gestational diabetes.

And here’s what the American Diabetes Association tells women:\textsuperscript{3}

Treatment for gestational diabetes always includes special meal plans and scheduled physical activity. It may also include daily blood glucose testing and insulin injections.

Let’s send a note to WebMD and the ADA: “Read the medical research!”

What actually causes gestational diabetes was well researched between the 1940s and 1975, when a report\textsuperscript{4} summarized the earlier research and then explained that gestational diabetes is caused by excessive amounts of xanthurenic acid, usually present in blood in very low levels. All this xanthurenic acid combines with insulin molecules and blocks its activity. The “xanthurenic acid-insulin complex” can’t activate insulin receptors nearly as well as insulin alone does, and blood sugar rises.

Back to the causes of diabetes mellitus type 2 and type 1. In type 2, the cause is overproduction of insulin in response to carbohydrates (and dairy, but an explanation for that at another time). As the March issue of Green Medicine explains, overproduced, chronically high insulin causes insulin resistance, which in turn leads to even more insulin secretion to overcome that resistance, which leads to even more insulin secretion.

This back-and-forth upward-trending interplay (more insulin, more resistance, even more insulin, even more resistance, and so on) goes on and on (unless “carbs”—and dairy—are significantly restricted) until the insulin resistance is so strong it can’t be completely overcome, no matter how much insulin there may be. Blood sugar then goes too high—and it’s diagnosed as type 2 diabetes. This known cause of type 2 diabetes is very different than the cause of diabetes mellitus xanthurenica!

What’s different during pregnancy? Among other things, it’s a combination of “genetic” causes together with those really-high-estrogen levels that women’s bodies make when pregnant—way, way more than when not pregnant.

The cause of type 1 diabetes is much simpler. For a variety of reasons, the insulin-producing cells (“islet cells”) become weak and die. When that happens, insulin levels go lower and lower, until there’s very little insulin, or even none—and that’s type 1 diabetes. Again, a very different cause from diabetes mellitus xanthurenica.

But doesn’t everyone’s body chemistry make xanthurenic acid? (It’s a metabolite of tryptophan.) Indeed, 100% of us have this body chemistry. So why don’t we all have gestational diabetes even if we’re not pregnant or even women? The reason is that levels of xanthurenic acid are relatively low in most of us (unless we’re deficient in a certain B vitamin to be named later), so not very much of the “xanthurenic acid-insulin complex” is formed.
YOU Can Prevent and Cure Gestational Diabetes!

Continued from previous page

What’s different during pregnancy? Among other things, it’s a combination of “genetic” causes together with those really-high-estrogen levels that women’s bodies make when pregnant—way, way more than when not pregnant. But why does all that extra estrogen cause only a minority of women’s bodies to make lots more xanthurenic acid and develop gestational diabetes, when most women’s bodies don’t do that?

Women who develop gestational diabetes have “weakness” in the enzymes that metabolize tryptophan into serotonin and melatonin, but no weakness in the enzymes that metabolize tryptophan into xanthurenic acid.

That’s the “genetic” part: women who develop gestational diabetes have “weakness” in the enzymes that metabolize tryptophan into serotonin and melatonin, but no weakness in the enzymes that metabolize tryptophan into xanthurenic acid. Without the pregnancy levels of estrogen “putting pressure” on these weak enzymes, they can perform as they do in most women—metabolizing much less of their tryptophan into xanthurenic acid, and much more of it into many other molecules we’ve all heard about, including serotonin and melatonin.

With the high levels of estrogen during pregnancy, the weak enzymes falter and metabolize much more tryptophan than usual into xanthurenic acid and much less into serotonin, melatonin, and related molecules. If there’s much more xanthurenic acid, there’s much more “insulin-xanthurenic acid complex” formed, and greater impairment of insulin activity. With enough insulin impaired, diabetes—“gestational”—is the result.

But a woman can’t stop being pregnant (for many months, anyway), and she definitely can’t change her genetics, so she can’t really rid herself of gestational diabetes, returning to normal blood sugar levels (while reducing her baby’s risk of autism, too) within two to three weeks. Or can she?

Yes, she can! To understand how, here is a refresher on what many of us learned—or should have learned—in high school and college chemistry about how enzymes change one molecule into another. The key is that enzymes never work alone. They’re always aided by co-factors that are almost always “essential” (necessary to life) vitamins and minerals! Without those co-factors, the enzymes can’t function, and ultimately we die. That’s why they’re defined as “essential” nutrients!

“Weak” enzyme function can frequently be strengthened by adding in more co-factors! A key co-factor for the enzymes that metabolize tryptophan into serotonin and melatonin is vitamin B₆. Here are the results that women with gestational diabetes achieved by taking extra vitamin B₆ to strengthen their genetically “weak” enzymes. In 1975, fourteen pregnant women were diagnosed with gestational diabetes by the standard glucose tolerance test. All the women took 100 milligrams of vitamin B₆ (as pyridoxine) daily for two weeks. Repeat testing found that twelve of the fourteen (86%) no longer had the problem! It’s 2016, yet despite all this forty- to seventy-year-old basic science and clinical research demonstrating the cause and cure of gestational diabetes, it’s still not being applied!

And one last fact: textbooks of laboratory medicine in the 1940s told us that higher-than-usual xanthurenic acid in urine is diagnostic for vitamin B₆ deficiency! It’s 2016, yet despite all this forty- to seventy-year-old basic science and clinical research demonstrating the cause and cure of gestational diabetes, it’s still not being applied!

But you—yes, you, if you want to prevent gestational diabetes or cure yourself of it—can apply this extensive science. You can safely prevent or cure gestational diabetes yourself, and at the same time reduce your child’s risk of autism!
To Eliminate Gestational Diabetes, Use Pyridoxal Phosphate, Not Pyridoxine

Don’t use the “pyridoxine” form of vitamin B₆. That’s actually the “inactive” form of vitamin B₆, which actually does not activate the receptors for this vitamin. Most—but not all—humans can “activate” pyridoxine, but we have no way (without testing) to know if you are in the pyridoxine-activating group or not. (It’s quite possible that the 14% whose gestational diabetes didn’t disappear in the 1975 research summarized above were “poor activators” of pyridoxine.)

To make sure the pyridoxine actually does its job, it’s best to use the “active” form, pyridoxal-5-phosphate (usually shortened to P5P), fortunately available nearly everywhere supplements are sold, usually in a 50 milligram size. Don’t stop using your “pregnancy multivitamin/mineral supplement” as it contains the rest of the B-complex vitamins which “back up” the pyridoxal-5-phosphate.

Check With Your “Natural Medicine” Doctor…

1. If you have any doubts at all about doing this!
2. Towards the anticipated delivery date. Vitamin B₆ in both forms can inhibit the production of prolactin,¹² the hormone necessary for normal lactation and nursing. Work with a physician skilled and knowledgeable in natural and nutritional medicine to help you determine (possibly while checking your own blood sugar) a P5P “tapering schedule” so you can nurse your child normally. This physician will also be able to tell you about botanicals used for centuries by to improve lactation should they be needed.

A special thank you to author Adelle Davis, whose description of the research findings in reference #4 has helped me to help women reverse gestational diabetes for more than thirty years! ●

Endnotes

8. Vandelli, I. “The use of vitamin B₆ (pyridoxine) for suppressing the elimination of xanthurenic acid in pregnant and non-pregnant women following the oral intake of a measured quantity of tryptophan.” Acta Vitamin. (Milano) 1951;5:55.
The Almost-Forgotten Work of Dr. Arthur Alexander Knapp

- Vitamin D and eye health
- Myopia (nearsightedness), keratoconus, cataract, optic nerve atrophy, retinitis pigmentosa—all treatable with vitamin D
- Do children who need glasses really need vitamin D?

Last month’s Green Medicine reported a “breakthrough” publication1 from a major American university (in collaboration with a group from a Chinese university) about ultraviolet blood irradiation, a decades-old, effective, and safe natural medicine treatment to eliminate infections. The report was titled, in part, “The Cure that Time Forgot.”

There are literally hundreds of “cures that time forgot”! More accurately, there are literally hundreds of cures that “time” has ignored, or in many instances repressed, all because there wasn’t enough money to be made using safe, effective natural treatments—no $1,000 per pill, no $1.6 billion dollars to complete the FDA approval process.

But enough about making big, big money from ill health, and onto the remarkable (and almost entirely forgotten) work done and published in the 1930s and 1940s by Arthur Alexander Knapp, MD, an ophthalmologist and director of the Eye Service at Sing Sing prison hospital, who did research at the College of Physicians and Surgeons and Columbia University and at the New York Eye and Ear Infirmary.

Dr. Knapp observed that many eye disorders are a sign of a latent nutritional deficiency, and can be among the first signs of a systemic problem caused by nutrient inadequacy. He believed that understanding eye problems would help clarify or determine the overall diagnosis.

Dr. Knapp’s observations were made in both animals and humans. Some of his earliest work involved feeding animals a diet deficient in one or more nutrients and comparing the diseases that resulted to those of animals fed a normal diet. Animals fed diets low in calcium and vitamin D developed several eye problems very similar to those seen in humans.

With increasing nutritional deficiencies, the animals’ eye disorders became progressively worse. He reported that myopia (nearsightedness), keratoconus (cone-shaped deformation of the cornea—the frontal surface—of the eye), cataracts, optic nerve atrophy, retinitis pigmentosa (abnormal pigmentation of the retina which can progress to blindness), and allergic conjunctivitis were all related to, and treatable in varying degrees with, vitamin D and calcium.2

Animals fed diets low in calcium and vitamin D developed several eye problems very similar to those seen in humans.

His research in animals was followed by its application in humans.

To prove that vitamin D and calcium really made improvements in myopia and keratoconus, Dr. Knapp actually had plaster casts made of the shape of the cornea and the visible part of the eyes of human sufferers from myopia and keratoconus, before and after their treatments. (I don’t think I’d want plaster anywhere near my eyes, but Dr. Knapp was able to accomplish this with no harm done to the individuals involved!) The change in the shape of the plaster casts proved that the shape of the eyes had actually changed with calcium and vitamin D treatment. He often recommended as much as 50,000 IU vitamin D daily during treatment! (These are high doses, so don’t “do it yourself.” If you have any of these problems, always work with a physician skilled and knowledgeable in nutritional and natural medicine.)

In a 1938 study, Dr. Knapp reported that treatment with vitamin D and calcium helped reduce myopia in close to 50% of the patients.3

In individuals with a more severe form of myopia, termed “rapidly progressive,” over 50% of this group had either no further worsening in their myopia, or a significant decrease in the “rapid progression.” One-third actually had improvement in vision.

Although Dr. Knapp reported no research or clinical work with children, his research suggests that parents who are told their child needs glasses because of myopia (nearsightedness) should have the child tested for vitamin D. If the test result is found to be below the “tropical optimal” (60-100 ng/ml), supplemental vitamin D might slow, stop, or even reverse the child’s myopia!

Back to Dr. Knapp. One of his greatest successes was in treatment of keratoconus (remember, that’s a cone-shaped deformation of the cornea—the frontal surface—of the eye) with vitamin D and calcium. He reported in 1938 and 1939 that every individual treated had a favorable reduction in the “height” of the abnormally cone-shaped cornea4 (remember those plaster casts!), and that there were significant improvements in vision and in other objective criteria.4–6

Dr. Knapp’s work with experimental animals helped him to explain the improvements he found in humans. In

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experimental animals with both vitamin D and calcium deficiency, weaknesses developed in the walls of the eye, including the outer surfaces of both the cornea and the entire rest of the eyeball. This weakness was clearly observable under the microscope, and in Dr. Knapp’s technical terms, the “fibrous tunic” of these tissues was defective and weakened.

The defect worsened progressively with increasing vitamin D deficiency. Because of this weakening of the walls of the cornea and eyeball, the corneas and entire eyes literally enlarged—as proven by those before-and-after plaster casts, as well as measurement with a toolmaker’s microscope, accurate to 1/10,000th of an inch. Keratoconus and myopia were the logical and invariable results of these enlargements.

Even though Dr. Knapp reported improvements in retinitis pigmentosa in the 1940s, his work is still not being applied by mainstream medicine. Here’s an excerpt from his 1978 summary report “Blindness: Forty Years of Original Research”:

Patients with retinitis pigmentosa were treated with sufficient vitamin D and calcium. Without exception, every patient improved. Most of those treated were of the advanced type with very contracted [visual] fields and often poor central vision.⁷

He also observed that after improvement of retinitis pigmentosa with vitamin D and calcium treatment, “optic nerve pallor” (abnormal paleness of the optic nerve likely due to lack of blood flow) disappeared, and significantly increased blood flow could be observed.

In 1942, Dr. Knapp reported a study of 47 patients with allergic conjunctivitis of the eyes caused by allergy associated with myopia and keratoconus. The symptoms included itching, sensitivity to light, discharge, and tearing. Of the 43 who received treatment with vitamin D and calcium, 42 improved significantly⁸.

In the 1940s, Dr. Knapp was a captain in the Naval Reserve of these United States, assigned to the South Pacific. He worked with marines and sailors who had eaten only canned food for up to eighteen months while in service. Many had decreases in their vision over that time, documented by comparing the results of eye testing in these patients when they were recruited for service to results of eye testing when the men first reported symptoms at the base hospital.

As the visual declines were very similar to those Dr. Knapp observed in the 1930s in nutrient-deficient animals, he recommended calcium and high-dose vitamin D treatment, which improved their vision in many cases.⁹

Being an ophthalmologist, Dr. Knapp treated other eye complaints in these servicemen, including inflamed eyes accompanied by itching and photophobia (unusual sensitivity to light), and “vernal catarrh”—a stringy type of discharge from the eye caused by allergy. Some of the servicemen who complained of eye disorders were found to have “hazy fundi oculi,” which he described as an abnormality affecting the ocular nerve. Some of the men with “pallor of the optic nerve” had enlarged blind spots.

Treatment was simple: nutrient-dense diets full of fresh fruits, vegetables, and meats, along with multiple vitamin/mineral supplements. Most men recovered rapidly with this diet, many within just a few days. No other health issues were noted in any of the men except the eye disorders, and all otherwise appeared healthy.¹⁰

Back to the 1930s again, when Dr. Knapp conducted many of his animal studies with Dr. S. N. Blackberg. Some of these studies focused on the effects of a diet so low in vitamin D that rickets (bone deformation) resulted. They studied the development of eye disorders in these animals.

After only six weeks on the very low vitamin D diet, the dogs had many areas of deterioration in their eyes at about the same time as they developed an early stage of rickets. These eye problems became more and more pronounced during the next two months. Six months after the beginning of the very low vitamin D diet, the dogs developed cataracts.¹⁰

After twenty-nine weeks, one of the animals experienced conditions so severe that eye ulcerations developed and the dog died. In a control group of animals fed either a kennel diet or supplemented with vitamin D, none of the symptoms of rickets or eye disorders occurred.

In a later publication, Drs. Blackberg and Knapp theorized that the degenerative tissue changes they observed in animals in advanced stages of vitamin D deficiency in this study—including thinning, dimpling, and “cornification” (a callus-like thickening) of the surface cells of the cornea—may be responsible for weakening the cornea (as well as the rest of the eye) and contributing to pathological changes similar to those observed in humans.¹¹

Very fortunately, even if the importance of vitamin D to eye health—as demonstrated seventy to eighty years ago by Dr. Arthur Alexander Knapp and his colleagues—is another preventive and treatment measure that “time forgot,” you and I can do the preventive part ourselves! With the help of a physician skilled and knowledgeable in natural medicine, we can determine what amount of vitamin D is optimal for each one of us.

Of course it’s harder to treat ourselves for myopia, keratoconus, or any of the other eye problems noted above. Check with a physician skilled and knowledgeable in natural medicine about that, and

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maybe bring a copy of this article (with the references) along with you! ●

Endnotes

6. Ibid.

Who’ll Stop the (Glyphosate) Rain?

• Glyphosate (Roundup) found in rain; not disclosed for seven years!
• Glyphosate concentrations found in urine damages testicles

Many of us were told in school that the fall of the Roman Empire was accelerated by the plumbing of their cities, which was made of lead. Urban Romans were drinking lead in their water every day, but were said to be unaware of lead's hazards!

We’re doing much the same thing to ourselves in the 20th and 21st centuries. Or perhaps even though most of us are well aware of the risks of glyphosate (aka Monsanto’s Roundup), it’s still being sprayed all around our country and others by Big Agribusiness in collusion with government agencies. It’s old news that more than one Big Agribusiness official has gone back and forth from that position to a regulating position with los federales. But enough of that... .

In 2016, men’s testosterone levels at all ages are significantly lower than our grandfathers’ levels at the same age. Fertility problems have been steadily increasing, especially in the last two or three decades. Sperm counts are generally lower in the average man in the 21st century than in the 1950s.

It’s also old news by now that many of the herbicides and pesticides used in farming (except organic farming) are—for humans—“environmental estrogens.” These environmental estrogens are part of the reason for lower testosterone levels and lower sperm counts in each generation (or perhaps we should call it each degeneration) of American men since the 1950s.

Even though Big Agribusiness and the agencies supposedly in the business of “protecting public health” don’t seem to give a d---, each of us has been able to at least partially protect ourselves and our families by “going organic.” Enough of us have been done this that sales of organic foods are increasing at a tremendous rate.

Unfortunately, the GMO industry depends heavily on a chemical that is directly toxic to men’s testicles, a toxicity that public health authorities continue to ignore. Not only is it found in food—from which we can protect ourselves—but it’s literally falling from the sky everywhere! No kidding: a report published in 2014 tells us that glyphosate—yes, that’s Roundup—together with glyphosate’s primary “breakdown product” was found in 75% or more of rain and air samples! Even worse, these samples were taken in 2007, but the results weren’t published until 2014!

If Glyphosate (Roundup) Rain Isn’t Enough... .

The abstract below, taken verbatim [with “translations” in brackets] from research published in 2012, makes it perfectly clear...
that glyphosate, a principal active ingredient in the chemical spray Roundup, is toxic to testicle cells—even killing them—and significantly lowers testosterone synthesis:

Roundup is being used increasingly . . . on genetically modified plants grown for food and feed that contain its residues. Here we tested glyphosate and its formulation on mature rat fresh testicular cells from 1 to 10,000 parts per million . . . the range [found] in some human urine and in [the] environment. . . . We show that from 1 to 48 hours of Roundup exposure Leydig cells [the testicular cells which make testosterone] are damaged. Within 24–48 h this [Roundup] formulation is also toxic on the other cells, mainly by necrosis [cell death], by contrast to glyphosate alone which is essentially toxic on Sertoli cells [testicular cells which make sperm]. Later, it also induces apoptosis [cell suicide] at higher doses in germ cells and in Sertoli germ cells co-cultures. At lower . . . concentrations of Roundup and glyphosate (1 part per million), the main endocrine disruption is a testosterone decrease by 35%. The pesticide has thus an endocrine impact [lower testosterone levels and sperm counts] at very low environmental doses, but . . . a high contamination appears to provoke an acute rat testicular toxicity [cell death].

Remember, men! Even if you’re eating 100% organic and not spraying your lawn with Roundup, you can’t avoid breathing and drinking the stuff! It’s time to demand that public health authorities actually start to protect us from glyphosate!

GMO corn chips, anyone? ●

Endnotes

About Dr. Jonathan V. Wright

Dr. Wright established Tahoma Clinic in 1973 in Washington State to offer nutritional and other natural therapies for common health conditions instead of patented medications. A long-time researcher, author, speaker, and clinician, he has educated physicians in his techniques since 1983. Dubbed the “Father of Bio-Identical Hormones” by his peers, Dr. Wright was the first physician in the United States to prescribe comprehensive hormone replacement therapy (in the early 1980s) with hormones identical to those found in nature. This therapy (shortened to “BHRT”) is now used nationwide by millions.

A well-respected author, he has authored 13 books (with two texts achieving best-selling status), numerous medical articles, served as a columnist from 1976 to 2000, and since 1994 has written a popular monthly newsletter on natural health topics.

—ANH-USA