

# DIABETES SELF MANAGEMENT EDUCATION SUPPORT PROGRAM

Chris Veenendall BSN, RN, CDE Nurse Educator 715-684-1278 Cheri Rott RDN, MS Dietitian 715-684-1279

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#### **DIABETES NUMBERS**

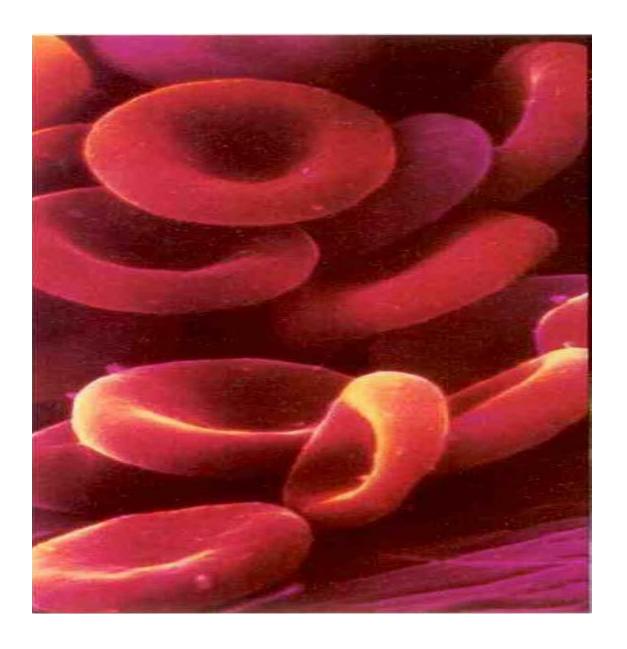
	Fasting	Meals (2 hrs after)
Normal	below 100	139 or less
Pre-diabetes	100-125 Impaired Fasting Glucose	140-199 Impaired Glucose Tolerance
Diabetes	126 +	200 +

To be diagnosed with diabetes, two tests are usually used

- fasting glucose test -nothing to eat for 8 hours before if 126 or above, this is diabetes
- Oral Glucose Tolerance Test (blood is drawn before you drink a high sugar drink. A value over 200 on this test is diabetes.

To be diagnosed with diabetes, these numbers must be elevated on **two** separate days.

• In 2010, the American Diabetes Association adopted the use of the A1c test for diagnosis of diabetes. The qualifying value was set at A1c of 6.5% or higher. Medicare does not recognize this test in diagnosis of diabetes.



The red blood cell lives for 90 days and accumulates all the sugar it is exposed to in the blood over these 90 days. It is a the sum total of what your body has been exposed to over the last 90 days, so is a valuable test for you and physician to get an A1c test done every 3 months to evaluate your treatment needs and evaluate how your management is going.

Cure . Care . Commitment

# What's Your Number?

... your Estimated Average Glucose (eAG) Number.



If you have diabetes you may know about A1C. A1C tells you the average level of glucose (sugar) in your blood over 2-3 months. It is reported as a percent (for example, 7%). Knowing your A1C tells you about your risk for complications of diabetes, problems caused by diabetes such as blindness, kidney disease, amputation, heart attack, and stroke.

Now we have a new way to report A1C called estimated average glucose, or eAG. eAG uses the same units that you see on a lab report or on your

An A1C of 7% — the goal for many people with diabetes — is the equivalent of an eAG of 154 mg/dl.

meter (for example, 154 mg/dl). Just like A1C, eAG lets you know the average level of glucose in your blood 24 hours a day, 7 days a week, for 2-3 months.

eAG can help you better understand your A1C level and helps you and your provider decide how to treat vour diabetes.

To learn more about how to take care of your diabetes, visit: diabetes.org 1-800-DIABETES

A1C to eAG Conversion Chart



SPONSORED BY



eAG:

My Results:

eAG:



# A new way to help you make the connection between daily and long-term glycemic control.

Introducing estimated Average Glucose (eAG), a new way to understand how well you're managing your diabetes

If you have diabetes you are probably familiar with the term A1C, the test that measures how well you are doing in managing your diabetes over time. The A1C represents the average glucose in your blood over a 3-4 month period.

We have long known that A1C measures average blood glucose over time, but there has never been a careful study to show exactly how a given A1C matches to an average glucose value. A major study has just been completed that tells us how to translate an A1C value into an average glucose value.

eAG is directly related to A1C, but uses the same values and units that you observe when you check your blood glucose with a meter or receive a fasting glucose value on a lab report. An A1C of 7% — the goal for most people with diabetes—is the equivalent of an eAG of 154 mg/dl.

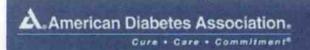
#### Why talk about eAG?

Though eAG and A1C represent the same thing—the average level of glucose in the body over time—for many patients, it may be helpful to begin thinking about their diabetes control in terms of eAG. First, it's simple: with eAG you use the same units you're familiar with from self-monitoring. Second, understanding the relationship between eAG and the values you get in self-monitoring may help you understand how your daily blood glucose checks relate to your long-term control.

#### How does eAG relate to my regular meter readings?

If you were to regularly check your blood glucose every morning, and before meals, you may find that your values are often in the low 100s, and the average value displayed on your meter might be around 125. But if your A1C during that same time period was 7.5, that would translate into an eAG of 169.

Why would the eAG number be so much higher than the "average" displayed by the meter?





#### estimated Average Glucose, eAG

The results of the A1C-Derived Average Glucose study (ADAG), published in Diabetes Care, have affirmed the existence of a line blood glucose levels.

In light of the study results, ADA is recommending the use of a new term in diabetes management, estimated average glucose, eAt A1C results to patients using the same units (mg/dl or mmol/l) that patients see routinely in blood glucose measurements.

Calculate:	a1C to eAG	in	⊚ mg/dl
	eAG to A1C	m	O mmol/I
A1C value	7.9	_	Calculate

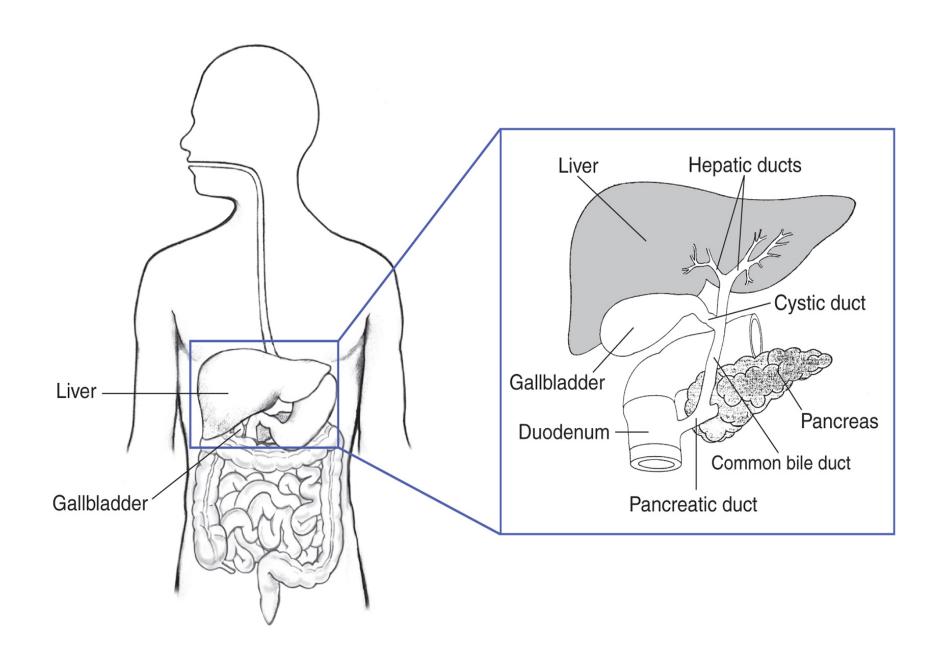
The relationship between A1C and eAG is described by the formula  $28.7 \times A1C - 46.7 = eAG$ .

A1C	eAG	
%	mg/dl	mmol/l
6	126	7.0
6.5	140	7.8
7	154	8.6
7.5	169	9.4
8	183	10.1
8.5	197	10.9
9	212	11.8
9.5	226	12.6
10	240	13.4

- Flyer describing the relationship between A1C and eAG.
- · eAG patient education print on demand piece.
- Questions and Answers regarding estimated Average Glucose (eAG)
- Translating the hemoglobin A1c assay into estimated average glucose values.
   David M. Nathan, Judith Kuenen, Rikke Borg, Hui Zheng, David Schoenfeld, and Robert J. Heine, for the A1c-Derived A Diabetes Care 2008
- Translating the hemoglobin A1C assay. Editorial.
   Richard Kahn and Vivian Fonseca. Diabetes Care 2008

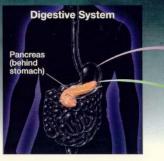
The liver makes stored sugar into glucose overnight to feed your body. Think of the heart, brain, lungs, kidneys, enzyme and hormone production the body makes overnight that need fuel from glucose to accomplish.

When you wake and put food into the stomach, it moves into the small intestine (duodenum) and connects to the pancreas/liver connection. The normal response is for the liver to stop making glucose, because your meal is a source of glucose. During the day, this food source of glucose goes into skeletal muscle to help us accomplish our work of the day.



#### The Progression of Type 2 Diabetes

Your body's inability to use its own insulin effectively is a major cause of Type 2 diabetes mellitus. As a result, glucose does not enter the body's cells and builds up in the bloodstream.



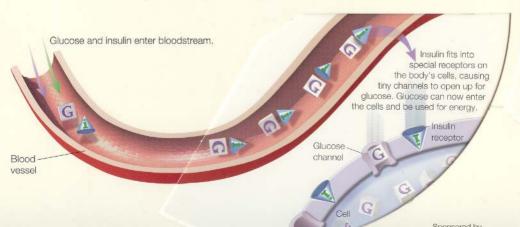


Your body breaks down all the food you eat into basic elements, including an important sugar called glucose. Glucose is your body's main source of energy.



Your pancreas produces a hormone called insulin, which enables glucose to enter the body's cells (muscle, fat and liver).

Normally the pancreas senses the amount of glucose in the bloodstream and releases the right amount of insulin. But in Type 2 diabetes, insulin resistance makes the body's cells less sensitive to insulin.



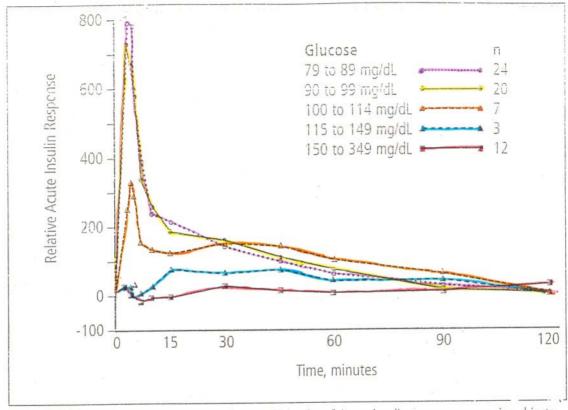
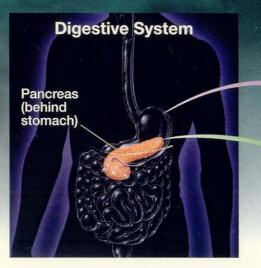


Figure 1. Acute insulin responses according to FBG values.<sup>7</sup> Acute insulin response occurs in subjects with FBG levels <115 mg/dL and are absent >115 mg/dL [Reproduced with permission from Brunzell et al. *J Clin Endocrinol Metab.* 1976;42:222-229].

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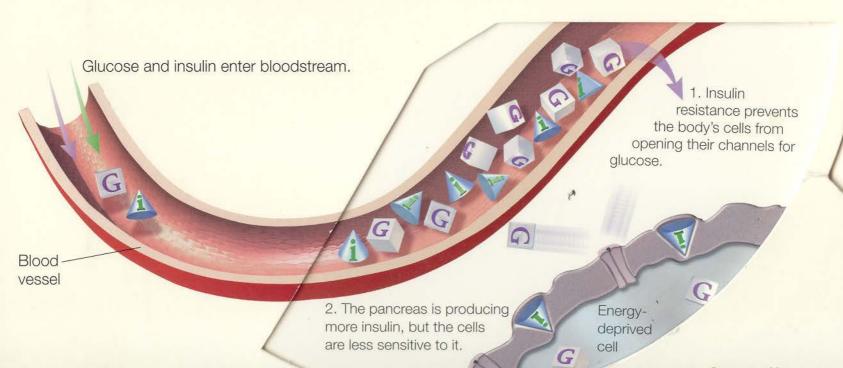
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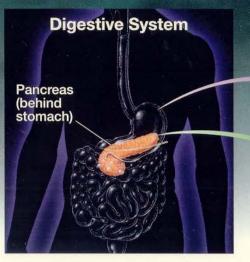
EARLY STAGE

Normally the pancreas senses the amount of glucose in the bloodstream and releases the right amount of insulin. But in Type 2 diabetes, insulin resistance makes the body's cells less sensitive to insulin.



# The Progression of Type 2 Diabetes

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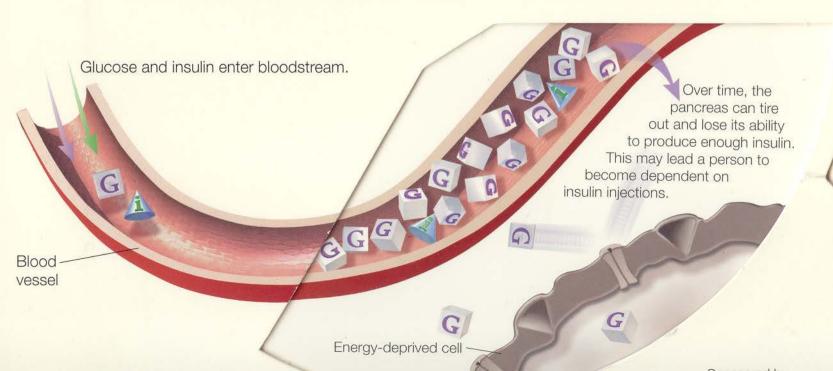
Your body breaks down all the food you eat into basic elements, including an important sugar called glucose. Glucose is your body's main source of energy.



Your pancreas produces a hormone called insulin, which enables glucose to enter the body's cells (muscle, fat and liver).

3. LATE STAGE

Normally the pancreas senses the amount of glucose in the bloodstream and releases the right amount of insulin. But in Type 2 diabetes, insulin resistance makes the body's cells less sensitive to insulin.



#### INSULIN RESISTANCE

related to and dependent on environment and genetics

Diabetes - too much sugar in the blood and not in the cells

**Cholesterol Problems** (not enough of the good or HDL, too much triglyceride, sticky LDL)

Cardiomyopathy (a big, but weaker heart)

**Hypertension** (high blood pressure)

Obesity- insulin is a growth factor- weight, skin tags, acanthosis nigricans

**Gout** - higher levels of uric acid seen in insulin resistance

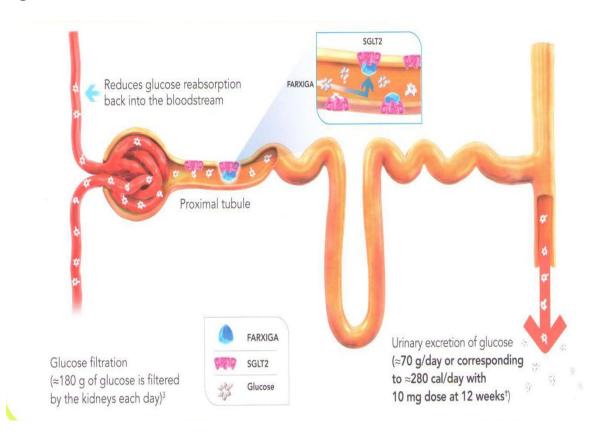
Polycystic Ovarian Syndrome (women who can't get pregnant- no regular periods)

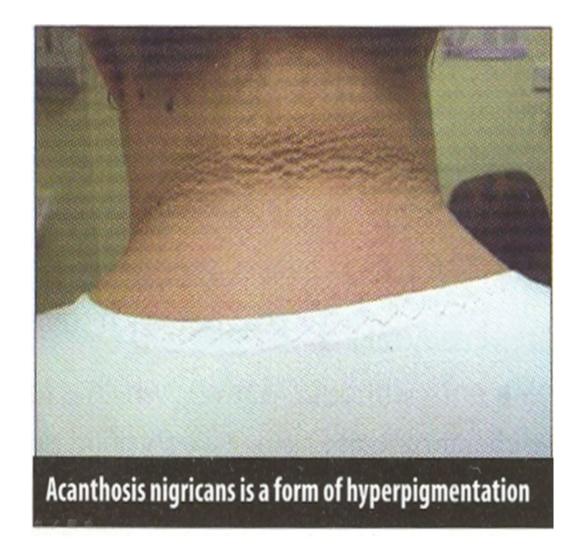
**Higher PAI-1** (makes blood clot easier, may be reason for stroke, heart attack)

Fatty Liver- liver stores excess sugar and converts to fat

**Depression-** mechanism rooted in circadian rhythm

Also in the development of diabetes, your kidney is making trouble for you much like the liver that can put out 3-4 times more glucose in a person with diabetes. In the kidney there are millions of little filters that blood goes through. The job is to take waste out of the blood stream and send it out as urine. But when there is diabetes present, right after it leaves the filter, there are little SGLT2- sites, that send sugar back to the circulation to help feed those poor starving cells! So some medications have been developed to counter this problem. Take an SGLT2-I medication first thing in the morning and drink 3 big extra glasses of water through the day. Beware of yeast overgrowth as all that sugar is now directed out with the urine.





This is usually seen in persons of color, or in very insulin resistant persons. The skin color changes and is a sign of insulin resistance.

More commonly in Caucasian's there are skin tags that signal insulin resistance but it can be seen in extreme insulin resistance cases of Caucasians also.

## guest editorial

hose looking for a common link among diabetes, high blood pressure, heart attack, and stroke believe they might have found it. The suspected culprit: inflammation.

Inflammation commonly occurs in response to a cut,



A Hot Topic: Inflammation

By Ronald P. Monsaert, MD. FACP. FACE Associate Editor

infection, an allergic reaction to poison ivy, and many other injuries to the body. But few had suspected that inflammatory processes might play a role in the development of atherosclerosis or diabetes. When we examine plaque deposits in clogged arteries, however, we see definite signs of inflamma-

tion, such as an accumulation of white blood cells in the plaque.

Recently, tests have been developed that measure hormones and other proteins circulating in the blood that tell us if an inflammatory process is going on. In response to these circulating substances, our liver makes a protein called C-reactive protein (CRP). CRP is elevated when a person has pneumonia, skin infections, or other similar conditions. But if we see higher levels of this protein in people who don't have an obvious infection, we now

suspect atherosclerosis (hardening of the arteries) is occurring. In fact, CRP has been shown to be a better risk marker for heart attack than cholesterol! Tests based on CRP may soon be used, along with cholesterol tests, to screen patients at risk for heart attack.

The good news here is that we can reduce these inflammatory processes. Eating a healthier diet, exercising more, reducing cholesterol, and quitting smoking are all ways to reduce CRP. Some drugs cut CRP levels as well, and new ones may soon be designed to do so even more efficiently.

We have long known that people with diabetes run a significantly higher risk of heart disease than those without diabetes. We can rejoice that tight control of blood sugar obviously prevents eye, kidney, and nerve disease in people with diabetes, but these studies do not seem to show favorable impact for preventing heart disease. More recently the "statin" trials have shown a reduction in heart events and strokes; some studies indicate that patients with diabetes exhibit even greater improvements with these agents than do those who do not have diabetes. In a recently published study in the British journal The Lancet, these drugs were shown to benefit those with relatively normal cholesterol as much as they did those with higher cholesterol levels. These studies seem

to suggest that statins offer benefits beyond their ability to reduce cholesterol. Would you believe that these drugs reduce CRP by 30 percent? Maybe, in a few years, your doctor will use these and other drugs to not only lower your cholesterol but also to achieve a "safer level" (yet to be determined)

By the way, we've had an excellent anti-inflammatory drug on the market since the 1940s. We know that it prevents heart attack, headache, and muscle ache. It's recommended by the American Diabetes Asso ciation and by the American College of Cardiology. It's available over the counter, and it's cheap.

Give up? It's called aspirin! I wonder why many of those with diabetes who could benefit from an aspirin a day don't bother to take it. I also wonder why many of these same people buy over-the-counter herbs and other untested "remedies" hoping that they will prevent these problems. (Some of these products are mentioned in this issue of *Forecast*—see page 68. Oftentimes I see my patients spend lots of money on these products, and yet they appear reluctant to spend similar amounts on drugs that have been extensively tested and are known to have favorable outcomes.

But I shouldn't scratch my head about this too much-I might get an inflammation!

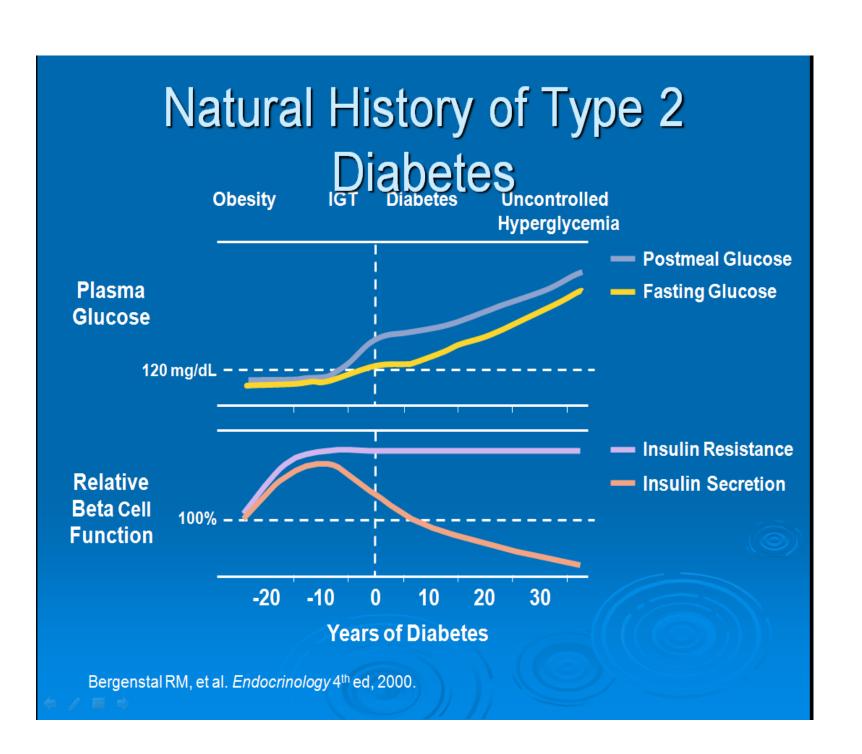
## INFLAMMATION IS THE PRECURSOR TO INSULIN RESISTANCE. SO PERHAPS IT ALL STARTS HERE WITH KNOWN CAUSES OF INFLAMMATION?

SMOKING – A DIRECT SOURCE OF INFLAMMATION.

In the 88,000 person Nurses Health Study, there was 42% more diabetes in the nurses who smoked than in those that did not.

- 2. ORAL CARE INFLAMMATION IN THE MOUTH. The dentist's office has had signs for years that report gingivitis or inflammation around the teeth can be sent in the blood stream to cause heart valve problems. It has been proposed that if dentists had A1c kits in their offices, they would find more diabetes than the clinics. Also a suspicion that gingivitis may play a part in Alzheimer formation!
- 3. **SLEEP APNEA LEFT UNTREATED.** This process causes problems from insulin resistance, congestive heart failure, hypertension (Blood Pressure problems), impotence, and blood that clots quicker leading to stroke and heart disease risk and diabetes.
- 4. **INSIDE THE BLOOD VESSELS.** The inflammation in your vessels with small dense LDL cholesterol that does not float like a beach ball in the vessel, but tends to erode the lining like a BB and set up shop for white cells to gather, foam cells to form and general inflammation in the area is established. Treatment: Statins
- 5. TUMMY FAT. "If you can't bend over and see your own genitals, you are obese!" Dr. Nancy Bohannon, endocrinologist, showed this principle at a conference with her tall, slender frame and there was shock at first, but she really gets her point across! If you can't tie your shoes without a grunt-what's in your way? We know that tummy fat is not quiet, but secretes a hormone called resistin that makes for the insulin resistance. Also perhaps tummy fat is talking to the brain saying "Feed me, Feed me!" If this can be proven, new medications have a chance to be developed to help.

WHO HAS THE LOWEST WHITE BLOOD CELL COUNT (measure of inflammation)?



To understand this slide, look first for the dotted line down the middle of the picture. This marks when diabetes is defined.

Across the bottom is the span of time this all occurs over- from 20 years before to nearly 40 years after diabetes is found.

Orange line: shows how insulin production goes down **naturally** in Type 2 diabetes

Purple line: shows insulin resistance rises even **before** you were diagnosed. That is why medications are started early after diagnosis, or even in Pre-diabetes. At the time of being told they have diabetes, some already have changes in their eyes or nerves.

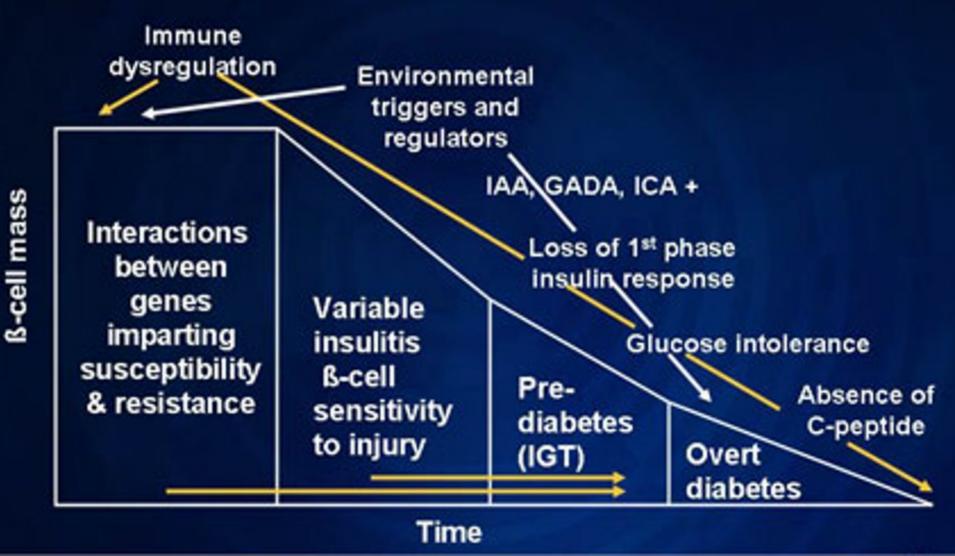
Yellow line: how high blood sugar is when you wake up and before eating.

Blue line: how high blood sugar is after eating.

Both the yellow and blue do not reflect treated/controlled diabetes. These lines can be changed by your habits and medications for diabetes.

Exercise reduces insulin resistance.

# Pathogenesis of Type 1 Diabetes



Atkinson MA, Eisenbarth GS. Lancet. 2001;358:221-229.



Comparison of type 1 and 2 diabetes			
Feature	Type 1 diabetes	Type 2 diabetes	
Onset	Sudden <sup>[4]</sup>	Gradual <sup>[4]</sup>	
Age at onset	Any age (mostly young) <sup>[4]</sup>	Mostly in adults	
Body habitus	Thin <sup>[4]</sup> or normal <sup>[5]</sup>	Often obese <sup>[4]</sup>	
Ketoacidosis	Common <sup>[4]</sup>	Rare <sup>[4]</sup>	
Autoantibodies	Usually present <sup>[4]</sup>	Absent <sup>[4]</sup>	
Endogenous insulin	Low or absent <sup>[4]</sup>	Normal, decreased or increased <sup>[4]</sup>	
Concordance in identical twins	50% <sup>[4]</sup>	90% <sup>[4]</sup>	
Prevalence	Less prevalent	More prevalent - 90 to 95% of U.S. diabetics <sup>[6]</sup>	



#### The Healthy Living Magazine

### **Understanding the Microbiome**

It's one of the hottest topics in science—but could it lead to a diabetes cure?

By Andrew Curry November 2015



Hiroshi Watanabe/Getty Images

Don't look now, but you're covered in bacteria—inside and out. The human body plays host to thousands of different species of microscopic organisms. All of the bacteria that live inside us are collectively known as the microbiome, and it's a world as diverse and complicated as any

rainforest. These organisms live in and on our hair and skin, and especially in our digestive system. Researchers estimate our bodies contain 100 trillion single-celled bacteria and just 10 trillion human cells.

All of these passengers aren't just along for the ride. We now know that some of the bacteria inside us perform necessary tasks. "The microbiome breaks down the food you eat and makes essential vitamins," says Stanford University geneticist Mike Snyder, PhD.

Other bacteria are unpleasant or harmful, producing toxins, eating away at our teeth, or making our armpits stink. Still others may be an evolutionary advantage gone wrong: By helping the body wring more calories out of food, for example, some species of gut bacteria may contribute to obesity and type 2 diabetes.

The idea that the trillions of bacteria inside our bodies could have a dramatic effect on our health is relatively new. In the past decade, research into this world within has become one of the hottest fields in science, driven by new gene sequencing technologies that let researchers quickly and inexpensively catalog and identify the thousands of different species living inside us.

As they learn more about how the microbiome works, some scientists have begun to wonder whether our bacteria are in a state of upheaval. Technological advances such as antibiotics, indoor plumbing, and processed food all have an effect on the bacteria inside us. Such triumphs of civilization have undoubtedly led to an overall improvement in human health: Around the world, people live longer than they did a century ago.

But by altering or upsetting a balance between our bodies, our diets, and the microbes established over millennia, these microbiome-altering factors may be promoting or even causing diseases virtually unknown to our hunter-gatherer ancestors, from type 2 diabetes to asthma, rheumatoid arthritis to irritable bowel syndrome.

"In general, there is an increasing awareness that the microbiome—which is essentially bacteria that live in our bodies—may have a role in the development of multiple diseases in human beings," says Nicolas Musi, MD, a diabetologist at the University of Texas Health Science Center in San Antonio. "There's an increasing interest in understanding how the microbiome and the human body interact."

Unlocking the secrets of the microbiome could contribute to a cure for diabetes. Researchers—including many funded by the American Diabetes Association (ADA)—are investigating the connections between the microbiome and diabetes.

We know more bacteria are good for you. The more diverse your microbiome, the less likely you are to have type 1.

—Ramnik Xavier, MD So far, the results are promising: Bacteria living in the digestive tract have been linked to obesity and inflammation, both contributors to type 2 diabetes. The interaction between the microbiome and the body's immune system might be a factor in triggering type 1 diabetes. Gestational diabetes and obesity during pregnancy may impact the

maternal microbiome and the bacteria that mothers pass on to their children during birth and breast-feeding, contributing to obesity in the offspring later in life.

There's lots more work to be done, particularly in terms of translating discoveries about the microbiome into practical, widely applicable treatments. One major possibility is probiotics, foods and dietary supplements that contain beneficial live bacteria. Scientists are already finding clues as to what doctors and patients might be doing in the future to maximize the microbiome.

#### Type 2

#### **Model Mice**

For her ADA-funded research into the cellular chemistry of people with type 2 diabetes, University of Wisconsin School of Medicine and Public Health diabetes researcher Michelle Kimple, PhD, uses specially bred mice, genetically altered so that they never feel full. The rodents eat so much they get fat; by the time they're 10 weeks old, they should develop the equivalent of type 2 diabetes. "They are extremely obese—they look like little marshmallows with legs," she says.

Not long ago, though, Kimple noticed something strange. The rodents raised in her lab were getting fat right on schedule. But the mice weren't developing type 2 diabetes. That, in turn, made them useless for the experiments she had planned.

At first, Kimple thought she had been sold a faulty batch of mice. When genetic tests confirmed that the mice were as advertised, she set about looking for other explanations. Soon she focused on the conditions the mice lived in: Mice raised and fed in sterile conditions at a special breeding facility reliably developed type 2 diabetes. Those in her lab, where protective booties and handwashing are about the only steps taken to prevent contamination from outside, did not.

Her surprising conclusion: Something about the environment of her lab or the food the mice were eating was altering their microbiome, short-circuiting the process that led to diabetes. "Evidence strongly points to the gut microbiome as playing a role in the protection from developing diabetes," she says. "Something about the building is protecting these mice."

Kimple is now working with colleagues to investigate the bacteria in the digestive systems of the two groups of mice. She's already found some major differences, including species of bacteria that are all but missing in the sterile-raised mice. It wasn't what she expected when she began her work, but she's excited by the possibility that this lab mouse mystery might be a window into how diabetes and the microbiome are connected. "It's a surprisingly strong result," she says. "It's got the potential to inform on human disease just by trying to figure out what's going on in these mice."

#### Type 1

#### Type 1 Trigger

To understand how the microbiome might trigger type 1 diabetes, researchers are looking at the Nordic nation of Finland. One in 100 Finns have type 1. That's three times higher than the frequency in the United States (about 1 in 300 Americans have type 1) and nearly seven times higher than Finland's neighbor to the east, Russia.

University of Helsinki researcher Mikael Knip, MD, PhD, thinks the microbiome may be part of the explanation. To test the idea, Knip and colleagues collected a wide range of samples—from mothers' milk to stool and blood—from infants and toddlers in Finland, Russia, and Estonia and compared them.

The goal was to spot differences that could explain why Finnish kids are more likely to develop type 1 diabetes than their counterparts in Estonia and Russia. They found that children in Russia and Estonia were typically exposed to more bacteria earlier in life, via everything from infections to fermented food and breast milk, than children in Finland.

Ramnik Xavier, MD, a gastroenterologist and researcher at the Broad Institute of Harvard and MIT, had a more specific question. "We took 33 kids from Finland and asked ... could we identify microbial signals in kids who stayed healthy and kids who developed type 1 diabetes?" He used some of the Finnish stool samples Knip collected to look at the bacteria in the kids' digestive systems.

In a paper published in the journal *Cell Host & Microbe* in February, Xavier showed that the gut microbiome was indeed different in kids who went on to develop type 1. The changes showed up long before their symptoms: "A year before they developed diabetes, the complexity of their gut microbiome dropped," Xavier says. "We know more bacteria are good for you. The more diverse your microbiome, the less likely you are to have type 1."

Here's how it might work: In type 1, the body's immune system mistakes the cells that produce insulin for invaders and wipes them out. Perhaps, Finnish researcher Knip suggests, kids in Finland are too clean and healthy early in life. "With less early infection, the immune system has too little to do, so it starts looking for other targets," Knip says. "If you have a decreased microbial load, the immune system doesn't get the exposure it needs to develop healthy responses."

The next step is identifying what exactly is missing in the microbiome of kids who develop type 1. "If you can find the triggers of disease, you can shift, reprogram, or reset the microbiome," Xavier says, suggesting probiotic use might be one way to do so. That may stop the type 1 autoimmunity chain reaction before it ever starts.

#### Type 2

#### Little Bacteria, Big Data

In people whose genes put them at risk for type 2 diabetes, disturbances in the microbiome might be the factor that puts them over the edge, triggering the failure of pancreatic beta cells that produce insulin.

Snyder should know. In 2010, he was in the middle of a study that tracked thousands of different molecules in his blood, many of them products of his microbiome. Genetic testing had already shown he was at an increased risk for diabetes, but Snyder—a trim and muscular 50-something who showed up to an interview in July on a battered road bike, drenched in sweat—didn't take it too seriously.

Nearly a year into the study, though, he caught a cold from one of his sons. "My blood glucose shot through the roof after I got this infection," he says.

In the weeks that followed, Snyder and his team watched—almost in real time—as he developed type 2 diabetes. "My genome has me predisposed, and a virus triggered the disease," he theorizes. To bring his diabetes under control, Snyder changed his diet, started running, and doubled his biking. But he's had flare-ups following viral infections in the three years since the study was published.

The remarkable results led to an ongoing, multimillion-dollar study that applies the same scrutiny to the cells and microbiomes of 100 volunteers without diabetes, focusing on how infections affect what's going on in their bodies.

To illustrate, Snyder calls up a series of pie charts on his computer screen. The charts represent different species of gut bacteria living in his intestines before and after a cold. "Here's my stool—healthy, sick, 2½ days later," Snyder says, pointing to the computer. "When I got sick, you can see the whole microbiome changes. It had a system-wide effect."

Snyder's theory is that viral infections and other illnesses may upset the microbial balance inside us. "Whether it's overfeeding or a viral infection, the microbiome gets disturbed," he says. That, in turn, could trigger chemical changes that may tip the balance in people already prone to type 2 diabetes or other diseases.

The exhaustive look Snyder's lab takes at genes and molecules in their volunteers will probably never be practical for the average patient. But Snyder hopes the study's deep dive into patients' microbiomes can narrow down the possibilities for future research. "Once we find out which pathways to look for, we can switch to a lighter version," he says. In the future, doctors might have a new way to identify people at risk for type 2 diabetes and manage their microbiome using probiotics or dietary changes to prevent it from kicking in.

#### **Gestational Diabetes**

#### **Maternal Influence**

The trillions of bacteria in and on us arrive shortly after birth, colonizing our gut, skin, and mouth. Our mothers pass on their bacteria during birth and nourish the fledgling microbiome with special proteins during breast-feeding.

As a result, the first year or two of life have a big influence on what kinds of bacteria we're stuck with for the rest of our days. "We believe whatever comes first plays a big role in what gets

established and has permanence," says University of Colorado-Denver biologist Jed Friedman, PhD.

That, in turn, means the makeup of the maternal microbiome at the moment of birth is extremely important. Variety is key: Research shows that people with fewer species of bacteria are more likely to be obese and have a higher risk for type 2 diabetes. "With garden-variety obesity, the diversity shrinks and there's less room for healthy bacteria," Friedman says.

Yet Friedman's ADA-funded research shows that just before giving birth, the diversity of the mother's microbiome plummets. Friedman has experimented with mice, comparing animals given microbes from women in the first trimester of pregnancy to those given transplants of third-trimester microbes. "Mice given the microbiome from women in their third trimester became fatter and more insulin resistant than those given first-trimester microbes," he says.

In evolutionary terms, this is no coincidence: Mothers tend to give babies the mix of bacteria that maximizes weight gain. "They're transmitting the microbiome that will allow the infant to extract as much energy as possible from food," Friedman says. "In theory, it makes sense." Evolutionarily speaking, you'd want to give babies the best shot at growing big and fast in a world where food might be hard to come by. But in modern times, where calories are overabundant, a fat-maximizing microbiome at birth can set kids up for weight problems later in life.

For babies born to obese mothers or mothers with gestational diabetes, the problem is worse. Gestational diabetes and obesity impact the maternal microbiome even more dramatically. Those differences are passed on to babies. "They're fatter at birth, and they're at high risk for extra weight gain," Friedman says. "What the microbes are doing is telling the body to store more fat."

The fix, for now, is familiar: Women should aim to reach a healthy weight before getting pregnant or work to healthfully manage weight gain during pregnancy. Women with gestational diabetes should control it as much as they're able. And, experts say, breast-feeding is recommended for as long as possible after birth.

#### Type 2

#### **Bacterial Byproducts**

Not all of the bacteria living inside us are helpful. Some produce a harmful substance known as endotoxin. Normally, it stays in the gut, with a small amount leaking into the bloodstream through the intestinal walls.

When levels of endotoxin in the body rise during an infection, they prompt the immune system to respond. The resulting clash makes us sick. "It's responsible for the normal signs and symptoms of bacterial infection," University of Texas researcher Musi says, including fever, chills, a racing heartbeat, and—in large amounts—even organ failure.

Could type 2 diabetes be connected to endotoxins? It would make sense: People with diabetes, as well as people who are obese, often have persistent inflammation in their bodies. It's as though they're constantly a little bit sick, with their immune systems working overtime.

Perhaps, Musi says, they're reacting to unusually high levels of bacterial byproducts in the blood. "People with type 2 diabetes have two to three times the endotoxin level in their bloodstream that a lean, healthy person would have," he says.

But why? Again, the answer may lie in the microbiome. "One idea is that eating more fat can favor the growth of certain bacteria proven to produce more endotoxin," he explains. With funding from the ADA, Musi is studying the effects of a fatty diet on gut bacteria—and on the intestinal walls, which could be damaged by fat and allow more endotoxin to leak through.

To test his theory, Musi has healthy volunteers eat a diet with approximately twice the usual fat content (but the same number of calories their previous diet included). After a month, he looks at the levels of endotoxin in their blood. When the study is complete, he should know if diet contributes to higher levels of endotoxins. If so, it would be a clue as to whether the microbiome drives the onset of type 2 diabetes.

It would also provide a simple way to reduce type 2 diabetes risk. "Ideally, nutritional changes would be the best treatment," Musi says. "Promoting a diet with a lower content of fat is a natural."

#### Bacteria, Defined

Bacteria are one of the first life forms to have developed on Earth. Single-celled and microscopic, they are also one of the most common and widespread organisms on the planet.

#### **Probiotics, Defined**

Probiotics are live bacteria, eaten in pill form or in food, in high enough concentrations that, the theory is, the body's microbiome is affected in a positive way. Yogurt with specially added bacteria is one common example.

#### **Mapping the Course**

The American Diabetes Association and the JDRF recently published a paper in the journal *Diabetes* that summarized expert opinion about specific areas of microbiome research that have the most promise for catalyzing the development of new treatments for diabetes.

Source: Diabetes, published online Sept. 29, 2015

American Diabetes Association, 1701 N. Beauregard St., Alexandria, VA 22311



Monitoring blood sugar gives you a peek within and much like these tiny bits of water on a stalk of grass you see the whole picture if you look close.

Diabetes in control is defined as blood sugar 80-130mg/dl when waking in the morning. Two hours after eating the goal is to be below 180mg/dl. Special populations will see targets tighter than this, or even far looser if there is heart disease or young children for example.

Remember your glucose is constantly changing. After you eat there is an expected rise. But, if your after-meal glucose is over 180, you probably feel quite tired, need a nap, etc.

If your insurance is Medicare, only one test strip per day is allowed if you are on pills. Three strips per day if you take insulin. The doctor can override this number for special reasons- like having low blood sugars, no awareness when going low, seeking improved control with more testing, etc. Since April 2013 this has

become more difficult, but if you want to test more, it is your provider that can help you get more strips.

The smaller lancets, 30, 32, 33 gauge, make for more comfortable testing. Leave your lancet device on the lowest setting. Use the side of the finger, along the nail, poke, wait 5 seconds after, then squeeze from your palm to the finger tip. Some meters need very little blood. If you can't succeed after several attempts, then you can try a deeper setting- but give the shallow poke the benefit of the doubt. There is no reason for pain with glucose testing. Start with a softer finger, like the little finger.

Your meter device can tell if you are getting ill (numbers higher than usual), how foods affect your glucose, when you drank alcohol and the next morning you are higher than usual, when you are low and not safe to drive a car, for a few examples. It can show you how effective exercise is. This ability is at your fingertips- no need to wait for the doctor to tell you how you are doing, if you know the goals and have the machine!!!

With one strip per day, please test at different times of the day. Using it once per day as you rise is a very false sense of security. Your A1C is the product of every moment for the last 3 months. If your fasting values are under 130, but your A1C is over 8% (controlled diabetes is under 7%) that tells you there is a big problem after your meals that you never looked at before, but could have.

When first diagnosed, you are working at improving the numbers. It may take several months, but control should be yours by 3 months from diagnosis. Work with your provider by bringing your log book and discussing your concern for numbers above the goal.

Once established with diabetes, you get a flavor for what it takes to keep you in control. There will be times that are not perfect. No one is perfect. These are just numbers. Knowing when you are in or out of control is important though.

What would you say about: Guy A's log book?

Before breakfast	Before lunch	Before supper	Before bed
162	89	105	140
174	102	113	158
126	125	80	162

What is the goal for fasting glucose, for before meals (HINT: they are the same 80-130)

What causes high sugars in the morning?

Liver output of glucose due to hormones raising glucose to wake you happens from 3AM on , alcohol the evening before, rarely a high fat supper

Rechecked and A1C still elevated.

Added Metformin and now asked to add a few glucose checks after eating finds:

132/ 252	78/ 189	105/ 280	146
128/ <mark>264</mark>	95/ <b>175</b>	112/ 300	160
110/ 280	88/ 182	89/178	135

What is the goal for 2 hours after you eat with diabetes? (under 180) How did these post-meal numbers look?

What could be done? Daily exercise increased/ established Recheck portion size and meal plan

If these two are both in place, it may mean a medication that effectively helps you with the high numbers that come after you eat a regular meal plan meal.

Name a few things that affect your blood sugar: eating, exercise, stress, illness, forgetting doses of medications, omission of medication, season change or time of the year, alcohol, food left on your hands before testing, new

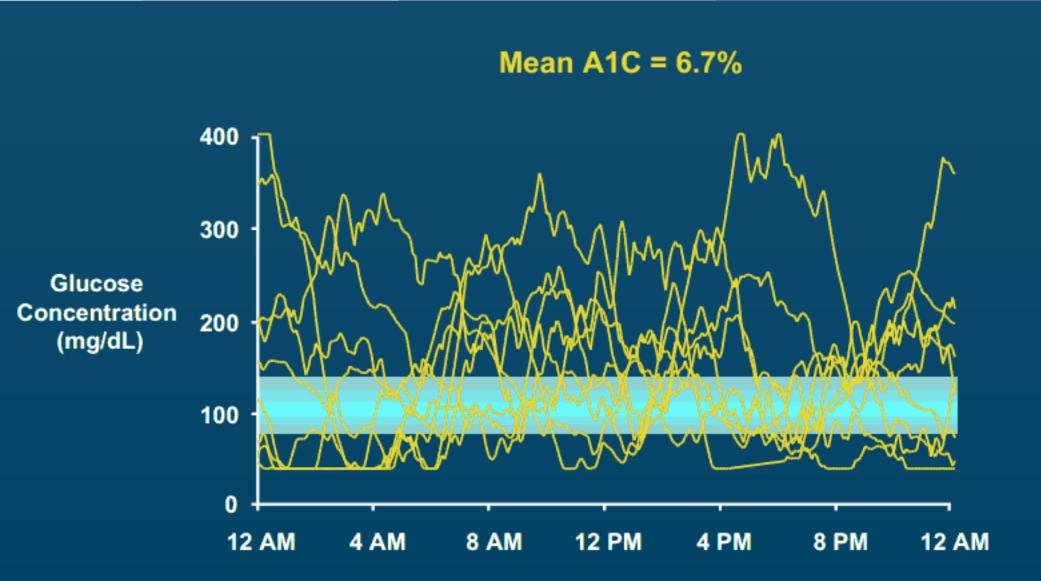
medications (esp steroids, some antibiotics and some anti psychotics). Lack of sleep or season changes? What has your testing shown you?

A new meter is usually due every two years as the technology advances. That change is usually made it the strip technology, and of course you will need a new meter that fits that strip.... But most insurances cover monitoring costs, especially Medicare, Medicaid and private insurers. The accuracy of the meter you use is a concern also. Ask the pharmacist for the most accurate one your insurance covers.



This is a continuous tracing of a person's glucose from Tuesday to Friday. He ate regularly at 5:30AM, 11:30AM and 6 PM, can you see what is happening? His baseline numbers range from below 70 to just over 100, but after eating, glucose rises to over 300mg/dl and stays up for hours afterward. Because he only tested before meals, he never saw this. The A1c was 8.2% and he was hurt because he felt he was a very good manager of his diabetes. The A1c test, to raise his awareness, the Continuous glucose Tracing to document what was happening, helped him stay away from complications. Remember glucose variability after meals is connected to heart disease.

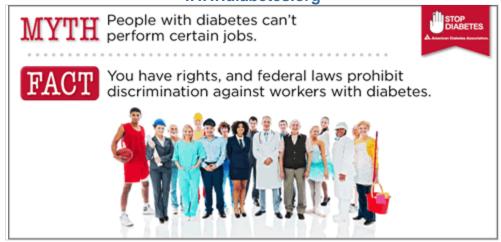
# Glucose Fluctuations Are Not Adequately Measured by A1C



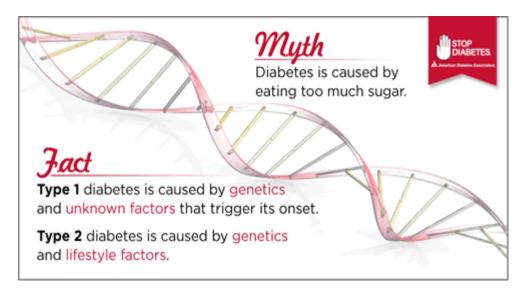
Type 1 diabetes, N = 9
24-h CGMS glucose sensor data
Data on file, Amylin Pharmaceuticals, Inc.

#### DIABETES MYTHS FROM THE AMERICAN DIABETES ASSOCIATION

#### www.diabetes.org







MYTH: Diabetes is not a serious disease.



**FACT:** Diabetes is a growing epidemic with a devastating physical, emotional and financial toll on our country.

It kills more Americans each year than AIDS and breast cancer

# COMBINED.

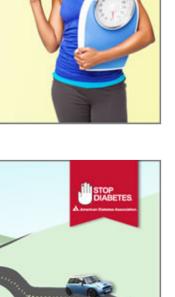
# MYTH

You have to lose a lot of weight for your diabetes to improve.

## FACT

Losing just 7% of your body weight can offer significant health benefits - about 15 pounds if you weigh 200.



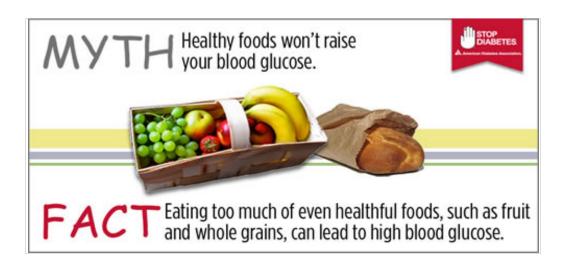


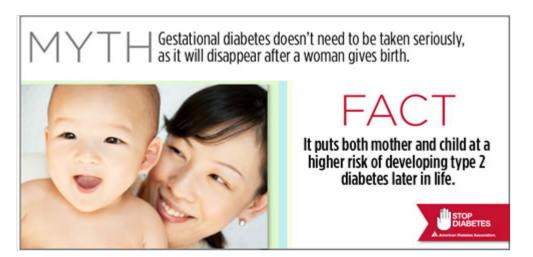
#### **MYTH**

People who use insulin are unsafe drivers.

#### **FACT**

The vast majority of drivers who use insulin can safely operate motor vehicles.















**Not always.** Some people cannot feel or recognize the symptoms of low blood glucose, or **hypoglycemia**, which can be dangerous.