Causes of Diabetes

National Diabetes Information Clearinghouse



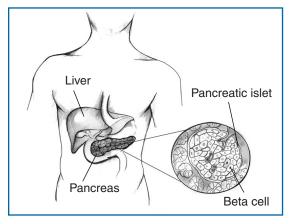


What is diabetes?

Diabetes is a complex group of diseases with a variety of causes. People with diabetes have high blood glucose, also called high blood sugar or hyperglycemia.

Diabetes is a disorder of metabolism—the way the body uses digested food for energy. The digestive tract breaks down carbohydrates—sugars and starches found in many foods—into glucose, a form of sugar that enters the bloodstream. With the help of the hormone insulin, cells throughout the body absorb glucose and use it for energy. Diabetes develops when the body doesn't make enough insulin or is not able to use insulin effectively, or both.

Insulin is made in the pancreas, an organ located behind the stomach. The pancreas contains clusters of cells called islets. Beta cells within the islets make insulin and release it into the blood.



Islets within the pancreas contain beta cells, which make insulin and release it into the blood.

If beta cells don't produce enough insulin, or the body doesn't respond to the insulin that is present, glucose builds up in the blood instead of being absorbed by cells in the body, leading to prediabetes or diabetes. Prediabetes is a condition in which blood glucose levels or A1C levels—which reflect average blood glucose levels—are higher than normal but not high enough to be diagnosed as diabetes. In diabetes, the body's cells are starved of energy despite high blood glucose levels.

Over time, high blood glucose damages nerves and blood vessels, leading to complications such as heart disease, stroke, kidney disease, blindness, dental disease, and amputations. Other complications of diabetes may include increased susceptibility to other diseases, loss of mobility with aging, depression, and pregnancy problems. No one is certain what starts the processes that cause diabetes, but scientists believe genes and environmental factors interact to cause diabetes in most cases.

The two main types of diabetes are type 1 diabetes and type 2 diabetes. A third type, gestational diabetes, develops only during pregnancy. Other types of diabetes are caused by defects in specific genes, diseases of the pancreas, certain drugs or chemicals, infections, and other conditions. Some people show signs of both type 1 and type 2 diabetes.

What causes type 1 diabetes?

Type 1 diabetes is caused by a lack of insulin due to the destruction of insulin-producing beta cells in the pancreas. In type 1 diabetes—an autoimmune disease—the body's immune system attacks and destroys the beta cells. Normally, the immune system protects the body from infection by identifying and destroying bacteria, viruses, and other potentially harmful foreign substances. But in autoimmune diseases, the immune system attacks the body's own cells. In type 1 diabetes, beta cell destruction may take place over several years, but symptoms of the disease usually develop over a short period of time.

Type 1 diabetes typically occurs in children and young adults, though it can appear at any age. In the past, type 1 diabetes was called juvenile diabetes or insulin-dependent diabetes mellitus.

Latent autoimmune diabetes in adults (LADA) may be a slowly developing kind of type 1 diabetes. Diagnosis usually occurs after age 30. In LADA, as in type 1 diabetes, the body's immune system destroys the beta cells. At the time of diagnosis, people with LADA may still produce their own insulin, but eventually most will need insulin shots or an insulin pump to control blood glucose

Genetic Susceptibility

Heredity plays an important part in determining who is likely to develop type 1 diabetes. Genes are passed down from biological parent to child. Genes carry instructions for making proteins that are

needed for the body's cells to function. Many genes, as well as interactions among genes, are thought to influence susceptibility to and protection from type 1 diabetes. The key genes may vary in different population groups. Variations in genes that affect more than 1 percent of a population group are called gene variants.

Certain gene variants that carry instructions for making proteins called human leukocyte antigens (HLAs) on white blood cells are linked to the risk of developing type 1 diabetes. The proteins produced by HLA genes help determine whether the immune system recognizes a cell as part of the body or as foreign material. Some combinations of HLA gene variants predict that a person will be at higher risk for type 1 diabetes, while other combinations are protective or have no effect on risk.

While HLA genes are the major risk genes for type 1 diabetes, many additional risk genes or gene regions have been found. Not only can these genes help identify people at risk for type 1 diabetes, but they also provide important clues to help scientists better understand how the disease develops and identify potential targets for therapy and prevention.

Genetic testing can show what types of HLA genes a person carries and can reveal other genes linked to diabetes. However, most genetic testing is done in a research setting and is not yet available to individuals. Scientists are studying how the results of genetic testing can be used to improve type 1 diabetes prevention or treatment.

Autoimmune Destruction of Beta Cells

In type 1 diabetes, white blood cells called T cells attack and destroy beta cells. The process begins well before diabetes symptoms appear and continues after diagnosis. Often, type 1 diabetes is not diagnosed until most beta cells have already been destroyed. At this point, a person needs daily insulin treatment to survive. Finding ways to modify or stop this autoimmune process and preserve beta cell function is a major focus of current scientific research.

Recent research suggests insulin itself may be a key trigger of the immune attack on beta cells. The immune systems of people who are susceptible to developing type 1 diabetes respond to insulin as if it were a foreign substance, or antigen. To combat antigens, the body makes proteins called antibodies. Antibodies to insulin and other proteins produced by beta cells are found in people with type 1 diabetes. Researchers test for these antibodies to help identify people at increased risk of developing the disease. Testing the types and levels of antibodies in the blood can help determine whether a person has type 1 diabetes, LADA, or another type of diabetes.

Environmental Factors

Environmental factors, such as foods, viruses, and toxins, may play a role in the development of type 1 diabetes, but the exact nature of their role has not been determined. Some theories suggest that environmental factors trigger the autoimmune destruction of beta cells in people with a genetic susceptibility to diabetes. Other theories suggest that environmental factors play an ongoing role in diabetes, even after diagnosis.

Viruses and infections. A virus cannot cause diabetes on its own, but people are sometimes diagnosed with type 1 diabetes during or after a viral infection, suggesting a link between the two. Also, the onset of type 1 diabetes occurs more frequently during the winter when viral infections are more common. Viruses possibly associated with type 1 diabetes include coxsackievirus B, cytomegalovirus, adenovirus, rubella, and mumps. Scientists have described several ways these viruses may damage or destroy beta cells or possibly trigger an autoimmune response in susceptible people. For example, anti-islet antibodies have been found in patients with congenital rubella syndrome, and cytomegalovirus has been associated with significant beta cell damage and acute pancreatitis—inflammation of the pancreas. Scientists are trying to identify a virus that can cause type 1 diabetes so that a vaccine might be developed to prevent the disease.

Infant feeding practices. Some studies have suggested that dietary factors may raise or lower the risk of developing type 1 diabetes. For example, breastfed infants and infants receiving vitamin D supplements may have a reduced risk of developing type 1 diabetes, while early exposure to cow's milk and cereal proteins may increase risk. More research is needed to clarify how infant nutrition affects the risk for type 1 diabetes.

Read more in the Centers for Disease Control and Prevention's (CDC's) publication *National Diabetes Statistics Report, 2014* at *www.cdc.gov* for information about research studies related to type 1 diabetes.

What causes type 2 diabetes?

Type 2 diabetes—the most common form of diabetes—is caused by a combination of factors, including insulin resistance, a condition in which the body's muscle, fat, and liver cells do not use insulin effectively. Type 2 diabetes develops when the body can no longer produce enough insulin to compensate for the impaired ability to use insulin. Symptoms of type 2 diabetes may develop gradually and can be subtle; some people with type 2 diabetes remain undiagnosed for years.

Type 2 diabetes develops most often in middle-aged and older people who are also overweight or obese. The disease, once rare in youth, is becoming more common in overweight and obese children and adolescents. Scientists think genetic susceptibility and environmental factors are the most likely triggers of type 2 diabetes.

Genetic Susceptibility

Genes play a significant part in susceptibility to type 2 diabetes. Having certain genes or combinations of genes may increase or decrease a person's risk for developing the disease. The role of genes is suggested by the high rate of type 2 diabetes in families and identical twins and wide variations in diabetes prevalence by ethnicity. Type 2 diabetes occurs more frequently in African Americans, Alaska Natives, American Indians, Hispanics/Latinos, and some Asian Americans, Native Hawaiians, and Pacific Islander Americans than it does in non-Hispanic whites.

Recent studies have combined genetic data from large numbers of people, accelerating the pace of gene discovery. Though scientists have now identified many gene variants that increase susceptibility to type 2 diabetes, the majority have yet to be discovered. The known genes appear to affect insulin production rather than insulin resistance. Researchers are working to identify additional gene variants and to learn how they interact with one another and with environmental factors to cause diabetes.

Studies have shown that variants of the *TCF7L2* gene increase susceptibility to type 2 diabetes. For people who inherit two copies of the variants, the risk of developing type 2 diabetes is about 80 percent higher than for those who do not carry the gene variant. However, even in those with the variant, diet and physical activity leading to weight loss help delay diabetes, according to the Diabetes Prevention Program (DPP), a major clinical trial involving people at high risk.

¹Grant RW, Moore AF, and Florez JC. Genetic architecture of type 2 diabetes: recent progress and clinical implications. *Diabetes Care*. 2009;32(6):1107–1114.

Genes can also increase the risk of diabetes by increasing a person's tendency to become overweight or obese. One theory, known as the "thrifty gene" hypothesis, suggests certain genes increase the efficiency of metabolism to extract energy from food and store the energy for later use. This survival trait was advantageous for populations whose food supplies were scarce or unpredictable and could help keep people alive during famine. In modern times, however, when high-calorie foods are plentiful, such a trait can promote obesity and type 2 diabetes.

Obesity and Physical Inactivity

Physical inactivity and obesity are strongly associated with the development of type 2 diabetes. People who are genetically susceptible to type 2 diabetes are more vulnerable when these risk factors are present.

An imbalance between caloric intake and physical activity can lead to obesity, which causes insulin resistance and is common in people with type 2 diabetes. Central obesity, in which a person has excess abdominal fat, is a major risk factor not only for insulin resistance and type 2 diabetes but also for heart and blood vessel disease, also called cardiovascular disease (CVD). This excess "belly fat" produces hormones and other substances that can cause harmful, chronic effects in the body such as damage to blood vessels.

The DPP and other studies show that millions of people can lower their risk for type 2 diabetes by making lifestyle changes and losing weight. The DPP proved that people with prediabetes—at high risk of developing type 2 diabetes—could sharply lower their risk by losing weight through

regular physical activity and a diet low in fat and calories. In 2009, a follow-up study of DPP participants—the Diabetes Prevention Program Outcomes Study (DPPOS)—showed that the benefits of weight loss lasted for at least 10 years after the original study began.²

Read more about the DPP, funded under National Institutes of Health (NIH) clinical trial number NCT00004992, and the DPPOS, funded under NIH clinical trial number NCT00038727 in *Diabetes Prevention Program* at *www.diabetes.niddk. nih.gov.*

Insulin Resistance

Insulin resistance is a common condition in people who are overweight or obese, have excess abdominal fat, and are not physically active. Muscle, fat, and liver cells stop responding properly to insulin, forcing the pancreas to compensate by producing extra insulin. As long as beta cells are able to produce enough insulin, blood glucose levels stay in the normal range. But when insulin production falters because of beta cell dysfunction, glucose levels rise, leading to prediabetes or diabetes.

²Diabetes Prevention Program Research Group. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet.* 2009;374(9702):1677–1686.

Abnormal Glucose Production by the Liver

In some people with diabetes, an abnormal increase in glucose production by the liver also contributes to high blood glucose levels. Normally, the pancreas releases the hormone glucagon when blood glucose and insulin levels are low. Glucagon stimulates the liver to produce glucose and release it into the bloodstream. But when blood glucose and

insulin levels are high after a meal, glucagon levels drop, and the liver stores excess glucose for later, when it is needed. For reasons not completely understood, in many people with diabetes, glucagon levels stay higher than needed. High glucagon levels cause the liver to produce unneeded glucose, which contributes to high blood glucose levels. Metformin, the most commonly used drug to treat type 2 diabetes, reduces glucose production by the liver.

The Roles of Insulin and Glucagon in Normal Blood Glucose Regulation

A healthy person's body keeps blood glucose levels in a normal range through several complex mechanisms. Insulin and glucagon, two hormones made in the pancreas, help regulate blood glucose levels:

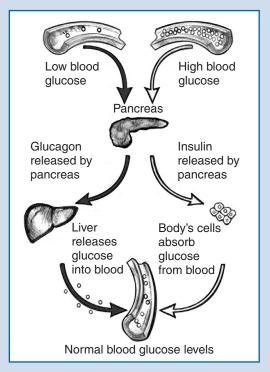
- Insulin, made by beta cells, lowers elevated blood glucose levels.
- Glucagon, made by alpha cells, raises low blood glucose levels.

When blood glucose levels rise after a meal, the pancreas releases insulin into the blood.

- Insulin helps muscle, fat, and liver cells absorb glucose from the bloodstream, lowering blood glucose levels.
- Insulin stimulates the liver and muscle tissue to store excess glucose. The stored form of glucose is called glycogen.
- Insulin also lowers blood glucose levels by reducing glucose production in the liver.

When blood glucose levels drop overnight or due to a skipped meal or heavy exercise, the pancreas releases glucagon into the blood.

- Glucagon signals the liver and muscle tissue to break down glycogen into glucose, which enters the bloodstream and raises blood glucose levels.
- If the body needs more glucose, glucagon stimulates the liver to make glucose from amino acids.



Insulin and glucagon help regulate blood glucose levels.

Metabolic Syndrome

Metabolic syndrome, also called insulin resistance syndrome, refers to a group of conditions common in people with insulin resistance, including

- higher than normal blood glucose levels
- increased waist size due to excess abdominal fat
- high blood pressure

 abnormal levels of cholesterol and triglycerides in the blood

People with metabolic syndrome have an increased risk of developing type 2 diabetes and CVD. Many studies have found that lifestyle changes, such as being physically active and losing excess weight, are the best ways to reverse metabolic syndrome, improve the body's response to insulin, and reduce risk for type 2 diabetes and CVD.

Cell Signaling and Regulation

Cells communicate through a complex network of molecular signaling pathways. For example, on cell surfaces, insulin receptor molecules capture, or bind, insulin molecules circulating in the bloodstream. This interaction between insulin and its receptor prompts the biochemical signals that enable the cells to absorb glucose from the blood and use it for energy.

Problems in cell signaling systems can set off a chain reaction that leads to diabetes or other diseases. Many studies have focused on how insulin signals cells to communicate and regulate action. Researchers have identified proteins and pathways that transmit the insulin signal and have mapped interactions between insulin and body tissues, including the way insulin helps the liver control blood glucose levels. Researchers have also found that key signals also come from fat cells, which produce substances that cause inflammation and insulin resistance.

This work holds the key to combating insulin resistance and diabetes. As scientists learn more about cell signaling systems involved in glucose regulation, they will have more opportunities to develop effective treatments.

Beta Cell Dysfunction

Scientists think beta cell dysfunction is a key contributor to type 2 diabetes. Beta cell impairment can cause inadequate or abnormal patterns of insulin release. Also, beta cells may be damaged by high blood glucose itself, a condition called glucose toxicity.

Scientists have not determined the causes of beta cell dysfunction in most cases. Single gene defects lead to specific forms of diabetes called maturity-onset diabetes of the young (MODY). The genes involved

regulate insulin production in the beta cells. Although these forms of diabetes are rare, they provide clues as to how beta cell function may be affected by key regulatory factors. Other gene variants are involved in determining the number and function of beta cells. But these variants account for only a small percentage of type 2 diabetes cases. Malnutrition early in life is also being investigated as a cause of beta cell dysfunction. The metabolic environment of the developing fetus may also create a predisposition for diabetes later in life.

Risk Factors for Type 2 Diabetes

People who develop type 2 diabetes are more likely to have the following characteristics:

- age 45 or older
- overweight or obese
- physically inactive
- parent or sibling with diabetes
- family background that is African American, Alaska Native, American Indian, Asian American, Hispanic/ Latino, or Pacific Islander American
- history of giving birth to a baby weighing more than 9 pounds
- history of gestational diabetes
- high blood pressure—140/90 or above—or being treated for high blood pressure
- high-density lipoprotein (HDL), or good, cholesterol below 35 milligrams per deciliter (mg/dL), or a triglyceride level above 250 mg/dL

- polycystic ovary syndrome, also called PCOS
- prediabetes—an A1C level of 5.7 to 6.4 percent; a fasting plasma glucose test result of 100–125 mg/dL, called impaired fasting glucose; or a 2-hour oral glucose tolerance test result of 140–199, called impaired glucose tolerance
- acanthosis nigricans, a condition associated with insulin resistance, characterized by a dark, velvety rash around the neck or armpits
- history of CVD

The American Diabetes Association (ADA) recommends that testing to detect prediabetes and type 2 diabetes be considered in adults who are overweight or obese and have one or more additional risk factors for diabetes. In adults without these risk factors, testing should begin at age 45.

What causes gestational diabetes?

Scientists believe gestational diabetes is caused by the hormonal changes and metabolic demands of pregnancy together with genetic and environmental factors.

Insulin Resistance and Beta Cell Dysfunction

Hormones produced by the placenta and other pregnancy-related factors contribute to insulin resistance, which occurs in all women during late pregnancy. Insulin resistance increases the amount of insulin needed to control blood glucose levels. If the pancreas can't produce enough insulin due to beta cell dysfunction, gestational diabetes occurs.

As with type 2 diabetes, excess weight is linked to gestational diabetes. Overweight or obese women are at particularly high risk for gestational diabetes because they start pregnancy with a higher need for insulin due to insulin resistance. Excessive weight gain during pregnancy may also increase risk.

Family History

Having a family history of diabetes is also a risk factor for gestational diabetes, suggesting that genes play a role in its development. Genetics may also explain why the disorder occurs more frequently in African Americans, American Indians, and Hispanics/Latinos. Many gene variants or combinations of variants may increase a woman's risk for developing gestational diabetes. Studies have found several gene variants associated with gestational diabetes, but these variants account for only a small fraction of women with gestational diabetes.

Future Risk of Type 2 Diabetes

Because a woman's hormones usually return to normal levels soon after giving birth, gestational diabetes disappears in most women after delivery. However, women who have gestational diabetes are more likely to develop gestational diabetes with future pregnancies and develop type 2 diabetes.³ Women with gestational diabetes should be tested for persistent diabetes 6 to 12 weeks after delivery and at least every 3 years thereafter.

Also, exposure to high glucose levels during gestation increases a child's risk for becoming overweight or obese and for developing type 2 diabetes later on. The result may be a cycle of diabetes affecting multiple generations in a family. For both mother and child, maintaining a healthy body weight and being physically active may help prevent type 2 diabetes.

³National diabetes statistics report, 2014. Centers for Disease Control and Prevention website. www.cdc.gov/diabeteS/pubs/statsreport14 htm. Updated June 13, 2014. Accessed June 16, 2014.

Other Types and Causes of Diabetes

Other types of diabetes have a variety of possible causes.

Genetic Mutations Affecting Beta Cells, Insulin, and Insulin Action

Some relatively uncommon forms of diabetes known as monogenic diabetes are caused by mutations, or changes, in a single gene. These mutations are usually inherited, but sometimes the gene mutation occurs spontaneously. Most of these gene mutations cause diabetes by reducing beta cells' ability to produce insulin.

The most common types of monogenic diabetes are neonatal diabetes mellitus (NDM) and MODY. NDM occurs in the first 6 months of life. MODY is usually found during adolescence or early adulthood but sometimes is not diagnosed until later in life. For more information about NDM and MODY, see the NDIC fact sheet Monogenic Forms of Diabetes at www.diabetes. niddk.nih.gov.

Other rare genetic mutations can cause diabetes by damaging the quality of insulin the body produces or by causing abnormalities in insulin receptors.

Other Genetic Diseases

Diabetes occurs in people with Down syndrome, Klinefelter syndrome, and Turner syndrome at higher rates than the general population. Scientists are investigating whether genes that may predispose people to genetic syndromes also predispose them to diabetes.

The genetic disorders cystic fibrosis and hemochromatosis are linked to diabetes. Cystic fibrosis produces abnormally thick mucus, which blocks the pancreas. The risk of diabetes increases with age in people with cystic fibrosis. Hemochromatosis causes the body to store too much iron. If the disorder is not treated, iron can build up in and damage the pancreas and other organs.

Damage to or Removal of the **Pancreas**

Pancreatitis, cancer, and trauma can all harm the pancreatic beta cells or impair insulin production, thus causing diabetes. If the damaged pancreas is removed, diabetes will occur due to the loss of the beta cells.

Endocrine Diseases

Endocrine diseases affect organs that produce hormones. Cushing's syndrome and acromegaly are examples of hormonal disorders that can cause prediabetes and diabetes by inducing insulin resistance. Cushing's syndrome is marked by excessive production of cortisol—sometimes called the "stress hormone." Acromegaly occurs when the body produces too much growth hormone. Glucagonoma, a rare tumor of the pancreas, can also cause diabetes. The tumor causes the body to produce too much glucagon. Hyperthyroidism, a disorder that occurs when the thyroid gland produces too much thyroid hormone, can also cause elevated blood glucose levels.

Autoimmune Disorders

Rare disorders characterized by antibodies that disrupt insulin action can lead to diabetes. This kind of diabetes is often associated with other autoimmune disorders such as lupus ervthematosus. Another rare autoimmune disorder called stiff-man syndrome is associated with antibodies that attack the beta cells, similar to type 1 diabetes.

Medications and Chemical Toxins

Some medications, such as nicotinic acid and certain types of diuretics, anti-seizure drugs, psychiatric drugs, and drugs to treat human immunodeficiency virus (HIV), can impair beta cells or disrupt insulin action. Pentamidine, a drug prescribed to treat a type of pneumonia, can increase the risk of pancreatitis, beta cell damage, and diabetes. Also, glucocorticoids—steroid hormones that are chemically similar to naturally produced cortisol—may impair insulin action. Glucocorticoids are used to treat inflammatory illnesses such as rheumatoid arthritis, asthma, lupus, and ulcerative colitis.

Many chemical toxins can damage or destroy beta cells in animals, but only a few have been linked to diabetes in humans. For example, dioxin—a contaminant of the herbicide Agent Orange, used during the Vietnam War—may be linked to the development of type 2 diabetes. In 2000, based on a report from the Institute of Medicine, the U.S. Department of Veterans Affairs (VA) added diabetes to the list of conditions for which Vietnam veterans are eligible for disability compensation. Also, a chemical in a rat poison no longer in use has been shown to cause diabetes if ingested. Some studies suggest a high intake of nitrogen-containing chemicals such as nitrates and nitrites might increase the risk of diabetes. Arsenic has also been studied for possible links to diabetes.

Lipodystrophy

Lipodystrophy is a condition in which fat tissue is lost or redistributed in the body. The condition is associated with insulin resistance and type 2 diabetes.

Points to Remember

- Diabetes is a complex group of diseases with a variety of causes. Scientists believe genes and environmental factors interact to cause diabetes in most cases.
- People with diabetes have high blood glucose, also called high blood sugar or hyperglycemia. Diabetes develops when the body doesn't make enough insulin or is not able to use insulin effectively, or both.
- Insulin is a hormone made by beta cells in the pancreas. Insulin helps cells throughout the body absorb and use glucose for energy. If the body does not produce enough insulin or cannot use insulin effectively, glucose builds up in the blood instead of being absorbed by cells in the body, and the body is starved of energy.
- Prediabetes is a condition in which blood glucose levels or A1C levels are higher than normal but not high enough to be diagnosed as diabetes. People with prediabetes can substantially reduce their risk of developing diabetes by losing weight and increasing physical activity.
- The two main types of diabetes are type 1 diabetes and type 2 diabetes. Gestational diabetes is a third form of diabetes that develops only during pregnancy.

- Type 1 diabetes is caused by a lack of insulin due to the destruction of insulin-producing beta cells. In type 1 diabetes—an autoimmune disease the body's immune system attacks and destroys the beta cells.
- Type 2 diabetes—the most common form of diabetes—is caused by a combination of factors, including insulin resistance, a condition in which the body's muscle, fat, and liver cells do not use insulin effectively. Type 2 diabetes develops when the body can no longer produce enough insulin to compensate for the impaired ability to use insulin.
- Scientists believe gestational diabetes is caused by the hormonal changes and metabolic demands of pregnancy together with genetic and environmental factors. Risk factors for gestational diabetes include being overweight and having a family history of diabetes.
- Monogenic forms of diabetes are relatively uncommon and are caused by mutations in single genes that limit insulin production, quality, or action in the body.
- Other types of diabetes are caused by diseases and injuries that damage the pancreas; certain chemical toxins and medications; infections; and other conditions.

Hope through Research

The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) conducts and supports research into many kinds of disorders, including diabetes. Researchers throughout the United States and the world are working to better understand, prevent, and treat this disease. Finding ways to prevent or delay diabetes in people at risk is a key goal of this research. Scientists are identifying genes that may be involved in type 1 or type 2 diabetes and studying how they work. Studies are clarifying the complicated mechanisms that damage beta cells in diabetes. A great deal of work is also focused on preventing the different types of diabetes and improving treatment.

The NIDDK conducts research in its own laboratories and supports a great deal of basic and clinical research in medical centers and hospitals throughout the United States. Several other components of the NIH also conduct and support research related to diabetes. Other Government agencies that

sponsor diabetes programs are the Centers for Disease Control and Prevention, the Indian Health Service, the Health Resources and Services Administration, the VA, and the U.S. Department of Defense. In addition, many organizations outside the Government support diabetes research and education activities. These organizations include the ADA, the Juvenile Diabetes Research Foundation International. the American Association of Diabetes Educators, the American Association of Clinical Endocrinologists, and The Endocrine Society.

Clinical trials are research studies involving people. Clinical trials look at safe and effective new ways to prevent, detect, or treat disease. Researchers also use clinical trials to look at other aspects of care, such as improving the quality of life for people with chronic illnesses. To learn more about clinical trials, why they matter, and how to participate, visit the NIH Clinical Research Trials and You website at www.nih.gov/ health/clinicaltrials. For information about current studies, visit www.ClinicalTrials.gov.

For More Information

American Association of Diabetes Educators

200 West Madison Street, Suite 800

Chicago, IL 60606 Phone: 1–800–338–3633

Internet: www.diabeteseducator.org

American Diabetes Association

1701 North Beauregard Street

Alexandria, VA 22311

Phone: 1-800-DIABETES

(1-800-342-2383)

Email: askADA@diabetes.org Internet: www.diabetes.org

Immune Tolerance Network

Office of the Director Administrative Operations Benaroya Research Institute

1201 Ninth Avenue Seattle, WA 98101-2795 Phone: 206–342–6562

Fax: 206-342-6588

Email: director@immunetolerance.org Internet: www.immunetolerance.org

JDRF

26 Broadway, 14th Floor New York, NY 10004

Phone: 1–800–533–CURE (1–800–533–2873)

Fax: 212-785-9595 Email: info@jdrf.org Internet: www.jdrf.org

Acknowledgments

Publications produced by the Clearinghouse are carefully reviewed by both NIDDK scientists and outside experts. This publication was reviewed by Alan Shuldiner, M.D., University of Maryland.

You may also find additional information about this topic by visiting MedlinePlus at www.medlineplus.gov.

This publication may contain information about medications and, when taken as prescribed, the conditions they treat. When prepared, this publication included the most current information available. For updates or for questions about any medications, contact the U.S. Food and Drug Administration toll-free at 1-888-INFO-FDA (1-888-463-6332) or visit www.fda.gov. Consult your health care provider for more information.

National Diabetes Education Program

1 Diabetes Way Bethesda, MD 20814-9692 Phone: 1–888–693–NDEP (1-888-693-6337)TTY: 1-866-569-1162

Fax: 703–738–4929

Email: ndep@mail.nih.gov Internet: www.ndep.nih.gov

www.yourdiabetesinfo.org

The National Diabetes Education Program is a federally funded program sponsored by the U.S. Department of Health and Human Services' National Institutes of Health and the Centers for Disease Control and Prevention and includes over 200 partners at the federal, state, and local levels, working together to reduce the morbidity and mortality associated with diabetes.

National Diabetes Information Clearinghouse

1 Information Way Bethesda, MD 20892-3560 Phone: 1–800–860–8747 TTY: 1-866-569-1162

Fax: 703-738-4929

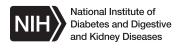
Email: ndic@info.niddk.nih.gov Internet: www.diabetes.niddk.nih.gov

The National Diabetes Information Clearinghouse (NDIC) is a service of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). The NIDDK is part of the National Institutes of Health of the U.S. Department of Health and Human Services. Established in 1978, the Clearinghouse provides information about diabetes to people with diabetes and their families, health care professionals, and the public. The NDIC answers inquiries, develops and distributes publications, and works closely with professional and patient organizations and Government agencies to coordinate resources about diabetes.

This publication is not copyrighted. The Clearinghouse encourages users of this publication to duplicate and distribute as many copies as desired.

This publication is available at www.diabetes.niddk.nih.gov.





NIH Publication No. 14-5164 June 2014