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## Facts About Diabetic Eye Disease

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There are approximately 16 million Americans who have either Type I (juvenile onset) or Type II (adult onset) diabetes. All are at risk of developing sight-threatening eye diseases that are common complications of diabetes.

Although early detection and timely treatment can substantially reduce the risk of severe visual loss or blindness from diabetic eye disease, many people at risk are not having their eyes examined regularly to detect these problems before they impair vision. Increased awareness of the sight-saving benefits of annual eye examinations through dilated pupils is essential to reduce the significant social and personal costs of diabetic eye disease.

### What is diabetic eye disease?

Diabetic eye disease refers to a group of sight-threatening eye problems that people with diabetes may develop as a complication of the disease. They include:

- **Diabetic retinopathy.** This disease damages blood vessels in the retina, the light-sensitive tissue at the back of the eye that translates light into electrical impulses that the brain interprets as vision.
- **Cataract.** A cataract is opacity of the eye's crystalline lens that results in blurring of normal vision. People with diabetes are twice as likely to develop a cataract as someone who does not have the disease. In addition, cataracts tend to develop at an earlier age in people with diabetes, around late middle age.

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- **Glaucoma.** This disease occurs when increased fluid pressure in the eye leads to progressive optic nerve damage. People with diabetes are nearly twice as likely to develop glaucoma as other adults.

Cataract and glaucoma also affect many people who do not have diabetes.

### **What is the most common diabetic eye disease?**

Diabetic retinopathy. The NEI estimates that of the approximately 10.5 million Americans who have diagnosed diabetes, between 40-45 percent have some degree of diabetic retinopathy. Between 600,000-700,000 Americans have diabetic retinopathy severe enough to cause vision loss. As many as 24,000 people go blind from this disorder annually, making it a leading cause of blindness among working-age Americans.

### **What is the cost of diabetic retinopathy?**

It is estimated that a year of blindness costs the U.S. Government approximately \$13,607 annually per person in Social Security benefits, lost income tax revenue, and health care expenditures. If Americans at risk for developing diabetic eye disease were regularly screened and treated to preserve their sight, the net annual savings to the Government would be more than \$100 million.

### **What causes it?**

Diabetic retinopathy is a complex disease. Although scientists understand much about the disease's natural history, they are still unclear about its specific pathological causes. There is, however, a consensus that diabetic retinopathy probably does not stem from a single retinal change. Rather, the disease may be triggered by a combination of biochemical, metabolic, and hematologic abnormalities.

In people with diabetes, three metabolic and hematologic changes are suspected of being involved in the early stages of diabetic retinopathy:

- **Hyperglycemia.** A chronic increase in normal blood-glucose levels may gradually alter cell metabolism in the retinal blood vessels.
- **Blood platelet abnormalities.** Diabetes-related biochemical changes may make circulating blood platelets abnormally sticky.
- **Blood vessel narrowing.** Hematologic changes may cause the retinal blood vessels to constrict.

These abnormalities may cause certain cells to die inside the retinal blood vessels. This leads to altered blood flow, increased blood vessel permeability, and the growth of certain blood vessel components. As a result, tiny outcroppings--called microaneurysms--may bulge from the weak blood vessel walls. The microaneurysms, which resemble tiny blisters on the blood vessels, may leak blood onto the central retina, or macula, causing an early, sight-impairing swelling of this area called macular edema.

The disease enters its proliferative stage when new blood vessels begin to grow into the retina and optic disc to increase blood flow to these tissues. New blood vessels may form because of hormonal signals, i.e., growth hormone, sent to the eye. These new blood vessels are fragile and often leak blood and protein into the vitreous--the transparent gel that fills two-thirds of the inner eye--and retina, causing visual impairment.

As the disease progresses, the new blood vessels may also grow into the vitreous and cause it to detach gradually from the back of the eye. As the vitreous pulls away, it may detach the retina as well. As a result, severe visual loss or blindness will occur.

### **What are the symptoms of diabetic retinopathy?**

For many people with diabetic retinopathy, there are no early symptoms. There is no pain, no blurred vision, and no ocular inflammation. In fact, many people do not develop any visual impairment until the disease has advanced well into its proliferative stage. At this point, the vision that has been lost cannot be restored.

However, some people in the early and advanced stages of diabetic retinopathy may notice a change in their central and/or color vision. The loss of central vision results from macular edema, which can often be effectively treated.

### **How is diabetic eye disease detected?**

Because diabetic eye disease often has no early symptoms, it is detected during a comprehensive eye examination through dilated pupils. Dilation consists of the eye care professional's placing medicated drops into the eye to enlarge the pupil. By doing so, the practitioner can better examine the back of the eye for early signs of disease, such as microaneurysms, before noticeable vision loss occurs.

For example, if the eye care professional detects diabetic retinopathy early, he or she can then monitor the patient's condition and determine the best time to treat the problem, should it progress to that point. The National Eye Health Education Program--coordinated by the National Eye Institute, one of the Federal National Institutes of Health--recommends that people with diabetes undergo a comprehensive eye examination through dilated pupils at least once a year.

### **How is diabetic retinopathy treated?**

Laser surgery, also called photocoagulation, is now being used successfully to treat proliferative retinopathy. It is performed by aiming a narrow, high-energy beam of light through the pupil and onto the retina. The beam of light is used to make hundreds of small burns over the retinal surface that destroy the growing blood vessels.

Laser surgery is also used to treat macular edema. In this procedure, however, the laser is aimed directly onto leaking blood vessels in the macula. The beam of light then seals the blood vessels to stop their sight-impairing leakage.

Current treatment guidelines are so successful that even people with proliferative retinopathy have a 90 percent chance of maintaining their vision. Current treatment guidelines call for (1) regular eye examinations through dilated pupils, (2) timely laser surgery, and (3) when needed, vitrectomy, a surgical procedure that clears hemorrhaged blood that can cloud vision from inside the eye.

The Diabetes Control and Complications Trial (DCCT) showed that better control of blood sugar levels slows the onset and progression of retinopathy and lessens the need for laser surgery for severe retinopathy.

### **What research is being conducted?**

During the past 25 years, scientists have made great progress in managing and treating diabetic eye disease. Laser surgery, cataract surgery, and glaucoma medications and surgery have all been either developed or improved considerably during this period. But if this research progress is to continue, additional understanding is needed of the cellular and biochemical basis of each disease.

For example, NEI scientists have developed the first animal research model for advanced (proliferative) retinopathy. This model will allow researchers to study better the vascular changes associated with this disease. It will also allow them to initiate studies on new drugs that are designed to prevent and treat diabetic retinopathy.

Other NEI-funded scientists are studying several growth factors to determine whether they influence the development of weak new blood vessels that proliferate in advanced diabetic retinopathy. In other studies, NEI scientists have inoculated bacterial cells with the DNA sequence that codes for the enzyme aldose reductase, which has been demonstrated as being a major mechanism by which early retinal capillary cells break down in the formation of diabetic retinopathy. The inoculated cells are yielding abundant and active aldose reductase, which is valuable for use in the development of a safe and effective enzyme inhibitor.

A well-coordinated public health effort also requires accurate data on disease prevalence, progression, and associated factors. For this reason, the NEI is supporting a long-term epidemiologic study in southern Wisconsin on diabetic retinopathy.

As science moves forward in its study of diabetic eye disease, it is likely that new treatments will be a result of basic and clinical research. Improved treatment, coupled with heightened public awareness, should go far toward reducing diabetic eye disease as a future national health problem.