



# Your Questions Answered

## NEUROPATHY/FIBROMYALGIA

**Q** I have peripheral neuropathy, but people often confuse it with fibromyalgia. What is the simplest way to explain the difference?



**DR. RICHARD  
A. LEWIS  
RESPONDS:**

**A** Peripheral neuropathy is a collection of disorders in which peripheral nerve fibers, which carry signals to and from the central nervous system (the brain and spinal cord), become damaged. Diabetes is the most common cause. Typically, nerve fibers in the hands and/or feet are affected. Symptoms include pins and needles, numbness, tingling, and weakness.

People with fibromyalgia may experience the same symptoms. However, these symptoms tend to come and go in fibromyalgia. In peripheral neuropathy, they are usually constant.

Fibromyalgia can cause other symptoms that people with peripheral neuropathy do not experience, including pain in soft tissue areas such as muscles and joints.

When a piece of the nerve (a biopsy) is taken from someone with peripheral neuropathy and tested, abnormalities in the nerve fibers can be seen. But in someone who has fibromyalgia—even if the person has numbness and tingling—no abnormalities are typically found.

Fibromyalgia is believed to result primarily from pain-processing problems in the central nervous system. As far as we know, the peripheral nerves are not damaged in any way. People with fibromyalgia have many tender points on the body, often in the shoulders, neck, and low back. But when a piece of the nerve from these areas is tested, no abnormalities are typically found. People with fibromyalgia who have pain in their shoulders and backs often describe burning, tingling, and shooting pains in their arms



The symptoms of peripheral neuropathy are usually constant, while those of fibromyalgia tend to come and go.

and legs, which may sound like peripheral neuropathy. However, the results of nerve conduction tests—in which a series of electrical impulses are given to the nerve—are usually normal.

When someone comes to me with tingling, numbness, and burning, I need to determine whether these are symptoms of peripheral neuropathy or something else. If I don't find anything abnormal from a biopsy or a nerve conduction test and the person also has tender points, then fibromyalgia may be the cause.

The treatments for fibromyalgia that have been approved by the U.S. Food and Drug Administration—including pregabalin (Lyrica), duloxetine (Cymbalta), and milnacipran (Savella)—can also be effective in treating the pain of peripheral neuropathy. The reason is that these medicines affect pain processing in the central nervous system, and while peripheral neuropathy is caused by damage to nerves outside of the brain and spinal cord, that pain still must be processed by the central nervous system.

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**DO YOU HAVE A QUESTION TO ASK THE EXPERTS?**

Send it to [neurologynow@lwwny.com](mailto:neurologynow@lwwny.com)

## HUNTINGTON'S DISEASE

**Q** How has the discovery of the gene for Huntington's disease changed treatment options and research?



**DR. ANNIE  
KILLORAN  
RESPONDS:**

**A** The drugs we use to treat Huntington's disease (HD) are not new, but they can help manage some symptoms. Unwanted movements can be managed with neuroleptic drugs such as haloperidol. Unfortunately, these drugs can't slow the underlying disease process. This can only be done by "neuroprotective" drugs, which inhibit cell death and deter disease progression. These drugs don't exist yet for HD, but research has advanced since discovering the HD gene in 1993.

By identifying the gene that causes HD, scientists have been able to study genetically engineered animal models of the disease, which helps us understand the disease process and

identify targets for new drugs. We have learned the defective HD gene produces an abnormal protein called huntingtin that damages the brain. Now, we are testing drugs that block this protein or stop it from being produced in the first place. We need to do these tests in animal models of HD before we can do clinical trials in humans.

The discovery of the HD gene has also helped to make genetic testing widely available. Each child of an HD parent has a 50-50 chance of inheriting the HD gene. Adults at risk for HD can learn of their gene status years before they develop any signs of the disease. Many people feel this can help them plan better for the future.

By participating in studies, people who test positive for the gene mutation but who don't have signs of the disease can help researchers find biomarkers—such as those we might measure in the blood—that indicate the presence of a disease. We hope to use these biomarkers to tell us how well a neuroprotective treatment is working, long before the patient starts showing any of signs of HD.

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Annie Killoran, M.D., is a neurologist and AAN member with a Master's in Pathology and Molecular Medicine who is completing a fellowship in Experimental Therapeutics.

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