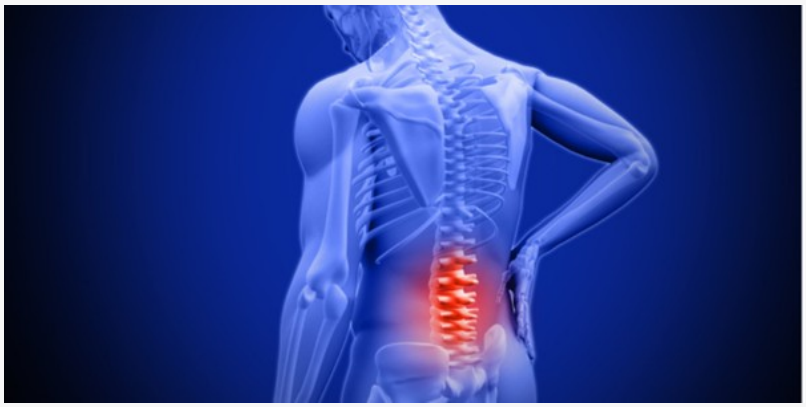
Blocking Pain at the Source: Hormone Therapy Rewires Nerve Signals in Aging Spines

Researchers reveal that hormone treatment reduces abnormal nerve invasion and improves chronic back pain



Damage to the vertebral endplates can promote abnormal growth of pain-sensing nerves, increasing pain sensitivity. Parathyroid hormone (PTH) stimulates bone cells to release signals that repel these invading nerves and help ease chronic spinal pain.

CHINA, February 4, 2026 /EINPresswire.com/ -- Low back pain (LBP) is a crucial risk factor for future health decline, affecting quality of life of individuals. Now, researchers have discovered that a hormone treatment can ease chronic LBP in mice by preventing harmful nerve growth inside damaged spinal tissue. The study shows that parathyroid hormone triggers bone cells to release a protein that repels pain-sensing nerves. These findings offer new clues for developing treatments for age-related and injury-related back pain.

Low back pain (LBP) is one of the most common health problems worldwide, affecting people of all ages and placing a heavy burden on healthcare systems. Many patients experience persistent discomfort that interferes with work, sleep, and daily activities. Yet in most cases, doctors cannot identify a clear structural cause, making long-term treatment difficult.

A new study published in Volume 14 of the journal Bone Research on January 22, 2026, suggests that a hormone treatment may help relieve chronic back pain by limiting abnormal nerve growth inside damaged spinal tissue. This study conducted by a team of researchers led by Dr. Janet L. Crane from the Center for Musculoskeletal Research, Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, United States, offers new insight into how bone cells influence pain signals in degenerating spines.

“During spinal degeneration, pain-sensing nerves grow into regions where they normally do not exist. Our findings show that parathyroid hormone can reverse this process by activating natural

signals that push these nerves away," says Dr. Crane.

Parathyroid hormone (PTH) is produced by the parathyroid glands and helps regulate calcium levels and bone remodeling. Synthetic forms of PTH are already used to treat osteoporosis. Previous studies have suggested that these treatments may also reduce bone-related pain, but the biological reason behind this effect remained unclear. To investigate further, the researchers studied three mouse models that mimic common causes of spinal degeneration: natural aging, mechanical instability caused by surgery, and genetic susceptibility. These models allowed the team to analyze how degeneration affects both bone structure and nerve growth. The mice received daily injections of PTH for periods ranging from two weeks to two months, while control animals received inactive solutions. The researchers then studied the animals' spinal tissue using high-resolution imaging and tested their sensitivity to pressure, heat, and physical activity.

After one to two months of treatment, mice that received PTH showed clear improvements in the structure of their vertebral endplates—the thin layers that separate spinal discs from vertebrae. These endplates became less porous and more stable. At the same time, treated mice tolerated pressure better, withdrew more slowly from heat, and were more active than untreated animals.

The team also analyzed nerve fibers inside the spine. In degenerated tissue, pain-sensing nerves often grow into abnormal locations, increasing sensitivity and discomfort. The researchers found that PTH treatment significantly reduced the number of these fibers, as measured by markers such as PGP9.5 and CGRP.

Further experiments revealed how this process works. PTH stimulated osteoblasts—cells responsible for building bone—to produce a protein called Slit3. Slit3 acts as a guidance signal that repels growing nerve fibers, preventing them from invading sensitive areas.

Laboratory tests confirmed that Slit3 directly blocked nerve growth. When nerve cells were exposed to Slit3 in culture, their extensions became shorter and less invasive. In contrast, when the researchers genetically removed Slit3 from osteoblasts in mice, PTH no longer reduced nerve growth or relieved pain. The team also identified a regulatory protein called FoxA2 that helps activate Slit3 production in response to PTH. This finding provides further insight into how hormone signals are translated into changes in nerve behavior.

Although the study was conducted in animal models, the results may help explain why some patients receiving PTH-based treatments for osteoporosis report reduced back pain. The researchers emphasize the need for further human studies before these findings can be applied in medical practice.

"Our study suggests that PTH treatment of LBP during spinal degeneration may reduce aberrant innervation, laying the foundation for future clinical trials exploring the efficacy of PTH as a disease-modifying and pain-relief treatment for spinal degeneration," concludes Dr. Crane.

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Reference
Title of original paper: PTH induced osteoblast Slit3 to decrease aberrant sensory innervation in degenerated vertebral endplates to relieve low back pain in mice
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About Dr. Janet L. Crane from Johns Hopkins University School of Medicine
Dr. Janet L. Crane is an Associate Professor of Pediatrics at the Johns Hopkins University School of Medicine, United States where she serves as the Director of the Pediatric Bone Health Program. She has a joint appointment in the Center for Musculoskeletal Research in the Department of Orthopedic Surgery. She earned her bachelor's degree in nutritional science from the University of Missouri and completed her medical degree at the University of Maryland–Baltimore. Her research is focused on metabolic bone diseases and skeletal fragility. Dr. Crane has published extensively on bone remodeling, metabolic bone disorders, and skeletal pain mechanisms.

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