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Study finds that hot and cold senses interact

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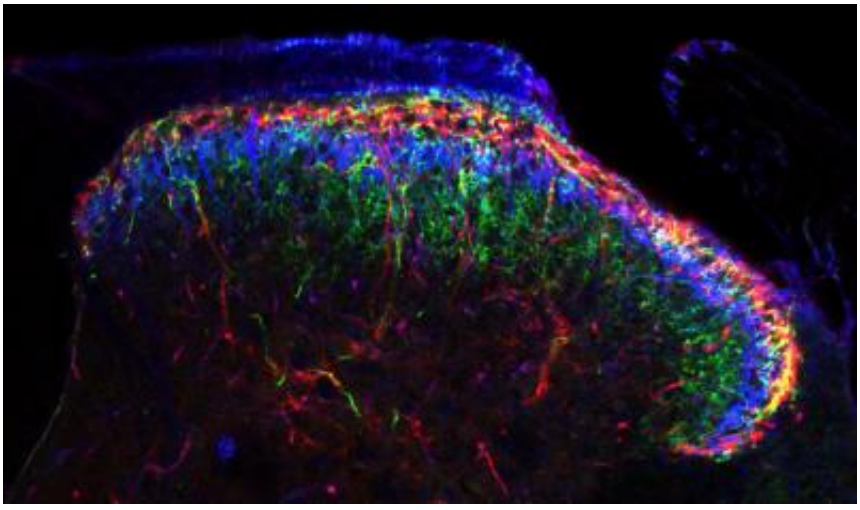

The neural circuit that senses heat and itch is labeled in red. Neural circuits that process other sensory stimuli are labeled in blue and green. The image is from the spinal cord. Credit: Zylka Lab, UNC School of Medicine.

A study from the University of North Carolina School of Medicine offers new insights into how the nervous system processes hot and cold temperatures. The research led by neuroscientist Mark J. Zylka, PhD, associate professor of cell biology and physiology, found an interaction between the neural circuits that detect hot and cold stimuli: cold perception is enhanced when nerve circuitry for heat is inactivated.

"This discovery has implications for how we perceive hot and cold temperatures and for why people with certain forms of chronic pain, such as neuropathic pain, or pain arising as direct consequence of a

nervous system injury or disease, experience heightened responses to cold temperatures," says Zylka, a member of the UNC Neuroscience Center.

The study also has implications for why a promising new class of pain relief drugs known as TRPV1 antagonists (they block a neuron receptor protein) cause many patients to shiver and "feel cold" prior to the onset of hyperthermia, an abnormally elevated body temperature. Enhanced cold followed by hyperthermia is a major side effect that has limited the use of these drugs in patients with chronic pain associated with multiple sclerosis, cancer, and osteoarthritis.

Zylka's research sheds new light on how the neural circuits that regulate temperature sensation bring about these responses, and could suggest ways of reducing such side-effects associated with TRPV1 antagonists and related drugs.

The research was selected by the journal *Neuron* as cover story for the April 10, 2013 print edition and was available in the April 4, 2013 advanced online edition.

This new study used cutting edge cell ablation technology to delete the nerve circuit that encodes heat and some forms of itch while preserving the circuitry that sense cold temperatures. This manipulation results in animals that were practically "blind" to heat, meaning they could no longer detect hot temperatures, Zylka explains. "Just like removing heat from a room makes us feel cold (such as with an air conditioner), removing the circuit that animals use to sense heat made them hypersensitive to cold. Physiological studies indicated that these distinct circuits regulate one another in the spinal cord."

TRPV1 is a receptor for heat and is found in the primary sensory nerve circuit that Zylka studied. TRPV1 antagonists make patients temporarily

blind to heat, which Zylka speculates is analogous to what happened when his lab deleted the animals' circuit that detects heat: cold hypersensitivity.

Zylka emphasizes that future studies will be needed to confirm that TRPV1 antagonists affect cold responses in a manner similar to what his lab found with nerve circuit deletion.

Provided by University of North Carolina Health Care