

Mice feel others' pain—literally

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showed increased activity in the ACC and INS. The authors found that inhibiting activity in the ACC reversed hyperalgesia in both primary and bystander [mice](#).

These results suggest a potential neural overlap between physically-induced and socially-transferred hyperalgesia.

More information: *eNeuro*,
[dx.doi.org/10.1523/ENEURO.0087-17.2017](https://doi.org/10.1523/ENEURO.0087-17.2017)

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Credit: Martha Sexton/public domain

Pain sensitivity associated with alcohol withdrawal may activate the same brain region in both drinking and non-drinking mice, finds a study published in *eNeuro*.

Monique Smith and colleagues previously showed that "bystander" mice housed with mice undergoing withdrawal from opioids or alcohol experience hyperalgesia, a heightened sensitivity to pain, just like the induced-withdrawal mice. In this study, the authors explored whether brain regions associated with pain and empathy for pain in humans—the somatosensory cortex, insula (INS), and [anterior cingulate cortex](#) (ACC)—might be involved in the social transfer of pain in mice.

Smith and colleagues compared the brain activity of "primary" mice with access to increasing concentrations of ethanol, bystander mice housed in the same room, and [control mice](#) housed in a separate room. The primary mice showed increased activity in the dorsal medial hypothalamus when access to alcohol was removed, which may indicate a role for this area in [alcohol withdrawal](#). In contrast, bystander mice

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