

Mind

We may finally know which brain cells cause motion sickness

Researchers have identified neurons in mice that influence whether the animals experience motion sickness, which could lead to new ways of preventing the condition in humans


By [Grace Wade](#)


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🚩 Motion sickness may be driven by neurons in the vestibular system

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Certain neurons in mice appear to govern the animals' experience of [motion sickness](#)  [/article/mg24732961-000-prone-to-motion-sickness-your-sex-diet-and-shoe-size-who-to-blame/](#). If the same is true for humans, it could lead to new ways of treating the condition.

Motion sickness  /definition/motion-sickness/ occurs when your eyes perceive movement differently than your vestibular system, which detects how your body moves through space – whether that is forwards, backwards or side-to-side – and is made up of structures in your inner ear. When your brain receives conflicting information, as it can during a car or boat ride, you may experience queasiness, dizziness or a sense of being off-balance.


However, the exact neurological underpinnings of motion sickness are unclear. To learn more, **Albert Quintana**  <http://www.quintanalab.org/quintanalab-members> at the Autonomous University of Barcelona in Spain and his colleagues examined brain cells in the vestibular system of mice, focusing on a particular set of neurons that were previously shown to play a role in **balance** 
<https://pubmed.ncbi.nlm.nih.gov/34171443/>.

Using a technique to switch cells **on and off with light**  /definition/optogenetics/, the researchers activated these neurons in seven mice. They assessed whether the rodents ate less and moved around less in their cages, indicators of motion-sickness symptoms. After half an hour, these mice travelled a third as far as five other mice that didn't have the neurons activated – a distance comparable to that of mice who were spun inside a rotation device. The animals also ate less food after the cells were activated, which the authors suggest could be a result of nausea.

The researchers then genetically analysed the neurons. They found that the cells with a receptor already known to play a role in nausea were especially critical for inducing motion sickness symptoms in the mice. They gave 10 mice a medication called devazepide to block the receptor, and spun them inside a rotation device. Half an hour later, these animals walked three times the distance of mice given a placebo before being spun. This suggests that devazepide could be used to prevent motion sickness, says Quintana.

Currently available motion sickness medications can cause drowsiness, which limits people's ability to do certain activities. Devazepide doesn't cause drowsiness, though, says Quintana. It is also already approved for treating certain gastrointestinal conditions in humans. "So, it is a drug that potentially can go to market [for motion sickness] quite easily," says Quintana.

But we don't yet know if the same brain pathway dictates motion sickness in humans, he

 "It would be interesting to quantify and see if these cells are also causally involved [in other symptoms of motion sickness]," says **Kathleen Cullen** 

<https://www.bme.jhu.edu/people/faculty/kathleen-cullen/> at Johns Hopkins University in Maryland. It is probably the case that multiple neurons and brain circuits help govern the condition, she says.

