at nuclear power plants, says David Brenner, a radiation biophysicist at Columbia University in New York City. "It's really difficult to figure out the effects" of this type of exposure, he adds, "but pretty important that we do so".

This was a motivating factor for co-author Timothy Mousseau, an evolutionary ecologist at the University of South Carolina in Columbia. In 2017, Mousseau joined a volunteer mission to provide veterinary care to the hundreds of stray dogs living in the exclusion zone, a 2,600-square-kilometre area around the power plant to which Ukrainian officials restrict access for safety reasons.

Over the course of three years of trips to the area, Mousseau and his colleagues collected blood samples from some 300 dogs living at the power plant and around the mostly deserted city of Chernobyl.

DNA analysis revealed that the canines were not newcomers to the area. By comparing the animals' genetic profiles to those of other free-roaming dogs in Eastern Europe, the team found that the canines at the power plant — some of which are related to shepherd breeds — have been isolated from other dog populations for decades. And the researchers learnt that, despite Soviet concerns during the 1980s that the dogs would migrate and spread radioactive material, most of these animals hadn't moved far: those living closest to the plant are genetically distinct from their kin living just a few kilometres away.

A radioactive legacy

The dogs' continued presence in the area shows that they have been able to survive and breed, even while living near the reactor, "which is remarkable", says Ostrander. The 1986 accident deposited the deadly radioactive isotope caesium-137 at levels 10–400 times higher near the power plant than in the city of Chernobyl, just 15 kilometres away.

But teasing out which genetic changes in the dogs are caused by radiation and which are caused by other factors — such as inbreeding or non-radioactive pollutants — won't be easy, Brenner cautions. The team acknowledges these challenges, but the researchers argue that their detailed knowledge of these dogs' ancestry and the levels of radiation they were historically exposed to "provides an ideal focus group for our future studies".

The ongoing war in Ukraine hasn't stopped the group's research. But with fewer tourists visiting and leaving food scraps, the dogs are struggling to get by. So the researchers are working with a non-governmental organization to provide food, safeguarding the survival of Chernobyl's dogs — and their radioactive legacy — in the lean times ahead.

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HOW THE BRAIN SENSES A FLU INFECTION — AND ORDERS REST

Scientists trace the throat neurons that detect signs of infection and relay this information to the brain.

By Liam Drew

A case of influenza can make even the toughest people take to bed and lose their appetites. Now, scientists have identified neurons in mice that notify the brain of a flu infection, triggering decreases in movement and hunger (N.-R. Bin *et al. Nature* https://doi.org/jz7d; 2023).

Similar neurons connecting to other parts of the body might notify the brain of other infections, too, the authors say. The work was published on 8 March in *Nature*.

"This study flips previous thinking on its head," says Ishmail Abdus-Saboor, a sensory biologist at Columbia University in New York City who was not involved in the research. "This is paradigm-shifting in terms of how we think about sickness behaviour."

Brain surveillance

Before this research, "it was not clear how the brain becomes aware that there's an infection in the body", says study co-author Stephen Liberles, a neuroscientist at Harvard Medical School in Boston, Massachusetts. Many scientists thought that messenger molecules from infected tissue move through the bloodstream to the brain, diffusing into it to activate regions that trigger sickness behaviours.

The infection alert travels along neurons on "a dedicated highway to the brain".

Among the top candidates for these messenger molecules were signalling chemicals called prostaglandins, which are made in infected tissues. Aspirin and ibuprofen block prostaglandin production — and also suppress sickness behaviours, hinting that prostaglandins are key to triggering such behaviours.

The authors showed that a specific prostaglandin receptor, called EP3, is responsible for generating sickness behaviours. EP3 is found on neurons throughout the body, including in the brain. To test its function, the researchers deleted the brain's EP3 receptors in mice and infected the animals with flu virus. The mice still changed their behaviour — indicating that the brain is not getting infection dispatches from blood-borne prostaglandins.

Instead, the authors found that the key agents are a specific EP3-containing population of neurons located in the animal's neck. These neurons have branches that stretch from the mouse equivalent of the tonsils to the brainstem. This geography makes sense: the tonsil area "serves as the interface between the outside air and what goes in the airway", says study co-author Na-Ryum Bin, a neurobiologist also at Harvard. The area is rich in immune cells that churn out prostaglandins when they encounter pathogens.

The results tell a narrative of illness: flu viruses enter the airway and infect throat cells, triggering prostaglandin production, and these previously unappreciated neurons respond. The infection alert then travels along the neurons' branches on "a dedicated highway to the brain", Abdus-Saboor says.

A behavioural paradox

Neural pathways do something blood-borne signals cannot: they tell the brain the infection's exact location. The authors note that many other types of neuron have receptors for prostaglandins and other immune-related signals. They suggest that further dedicated pathways could exist, such as those for detecting gut infections, triggering nausea.

The study also revealed a paradox. Scientists assume that there is an evolutionary advantage to sickness behaviour. But when the team blocked those behaviours, such as food avoidance, mice were less likely to die of the flu. Liberles speculates that this behaviour-modifying system evolved because it is beneficial in most cases of infection — even if it isn't in all. Alternatively, behaviours such as immobility might be advantageous by reducing the spread of pathogens between individuals.

The new results don't tell the full story. The infection-sensing tonsil neurons trigger sickness behaviour only during a flu infection's first stage, which affects the upper airway and lasts roughly a week. As the virus moves into the lower respiratory tract over the course of the illness, another nerve pathway takes over the job of driving sickness behaviours. "If we could find a way to block that second pathway, that, in combination, could have tremendous clinical impact," Liberles says.