interference, it could greatly reduce the chances of harming future experiments.

Melanie Johnston-Hollitt, former director of the Murchison Widefield Array radio observatory in Western Australia, agrees. At Murchison, which is to be the Australian site of the giant Square Kilometre Array radio telescope, she helped to establish what is probably the world's largest radio-quiet zone, at more than 500 kilometres across.

Permits are required to carry electronic devices into the site, and "all equipment you take into that area goes through an additional electromagnetic testing process", to check for unwanted radio emissions, says Johnston-Hollitt, currently a radioastronomer at Curtin University in Perth, Australia. "I can tell you with confidence that you can do that with a cubesat," she says, referring to the tiny satellites that researchers fear could swarm around the Moon, creating a source of noise.

Even so, "to suppress interference to the level necessary to do precision radioastronomy is incredibly difficult", says astronomer Andrew Siemion, who leads the Breakthrough Listen search for extraterrestrial intelligence project at UC Berkeley. That work involves looking for signals across a broad range of radio waves – including the gigahertz frequencies at which satellites communicate.

Lunar economy

Astronomers face an uphill struggle. The same technological advances that promise to make the Moon more accessible for their experiments will also make the environment more crowded. More than 250 Moon missions are expected over the coming decade from the space agencies of the United States, Europe, Russia, South Korea, China, Japan, India, Canada and the United Arab Emirates – as well as a host of private companies. That will add up to a US$100-billion 'lunar economy', according to Northern Sky Research, a consulting firm in Cambridge, Massachusetts. There are also plans to install a lunar satellite navigation system, which could be a source of noise.

Alanna Krolikowski, a political scientist at Missouri University of Science and Technology in Rolla, thinks that researchers should push for international treaties to protect the Moon. "There is now widespread recognition that we need governance for this forthcoming lunar renaissance," she told last month's conference.

The Artemis Accords, an international agreement led by NASA, attempts to provide some guidance to help the agencies involved avoid disrupting each other's missions. But it is designed mainly to serve the needs of its signatory countries; a better way to regulate the Moon could be to have rules drafted by the United Nations Committee on the Peaceful Uses of Outer Space, Krolikowski said. "The window in which to do that is small – and shrinking."

Stray puppies play at the Chernobyl power plant in Ukraine.

SEAN GALLUP/GETTY

WHAT CHERNOBYL'S DOGS COULD TEACH US ABOUT RADIATION

Multi-year project in Ukraine aims to uncover the health effects of chronic radiation exposure.

By Freda Kreier

In the early hours of 26 April 1986, two explosions rocked the nuclear power plant near the Ukrainian city of Chernobyl, then part of the Soviet Union. The accident at reactor four spewed radioactive material into the air, leading the Soviet authorities to evacuate thousands of people from the area. Homes were left behind – and, in many cases, pets.

In the days after the accident, response crews sought out abandoned and stray dogs, with the goal of killing them to stop the spread of radioactivity. Yet some seem to have survived.

In the first genetic study of any large mammal in the area around Chernobyl, DNA collected from feral dogs living near the power plant today reveals that they are the descendants of dogs that were either present at the time of the accident or settled there shortly afterwards[1]. The study, published on 3 March in *Science Advances*, is the first step in a larger project aimed at determining how the dogs have adapted to survive in one of the most radioactive places on Earth. Researchers hope to use the knowledge gained to better understand the effects of long-term radiation exposure on human genetics and health.

"We have so much to learn from these animals," says Elaine Ostrander, a geneticist at the US National Institutes of Health in Bethesda, Maryland, and a co-author of the study. "This is a golden opportunity to see what happens when generations of large mammals live in a hostile environment."

The immediate impacts of the accident at Chernobyl were obvious: around 30 people who worked at the plant and firefighters who responded to the disaster died of radiation poisoning within a few months, according to the World Health Organization. And in the surrounding areas, pine trees withered and many insect species vanished, unable to survive in the radioactive soil.

What is less clear is how low levels of lingering radioactive material affect the plants and animals around Chernobyl today. A handful of studies have reported unusually high genetic mutation rates in barn swallows[2] and fruit flies[3] in the vicinity of the reactor, which is now entombed in a steel and concrete sarcophagus.

However, the health effects of low levels of radiation are still hotly debated. This matters because people risk exposure to low doses of radiation in all sorts of contexts, including through certain medical scans or while working

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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2. Ellegren, H., Lindgren, G., Primmer, C. & Møller, A. P. *Nature* **389**, 593–596 (1997).
3. Hancock, S. *et al. Environ. Res.* **172**, 333–337 (2019).

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.