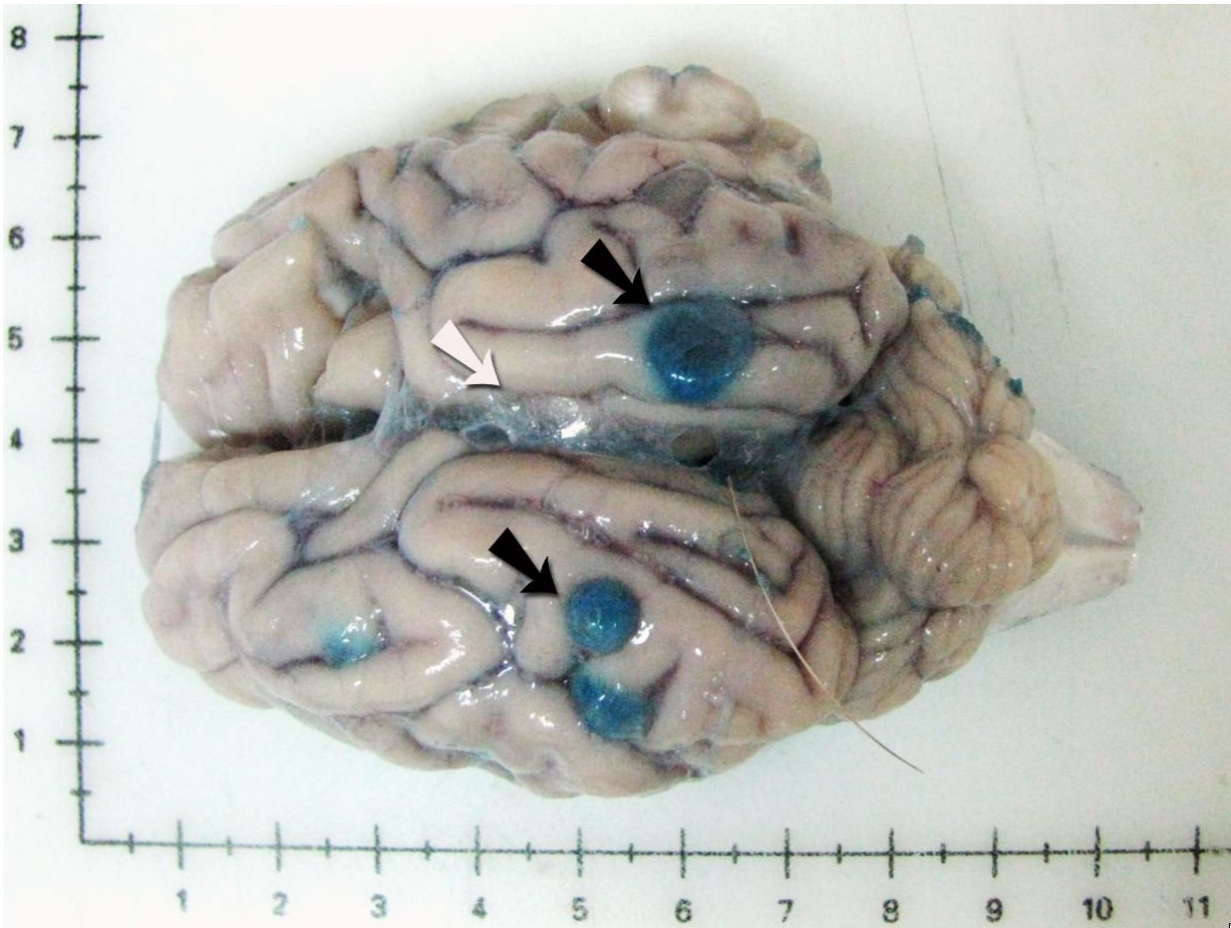


New treatment investigated for brain tapeworm infection

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Pig brain naturally infected with *Taenia solium* five days after praziquantel treatment and infused Evans blue prior to necropsy. The black arrows point to blue cysts with increased vascular permeability in surrounding tissues; a clear cyst with unaltered vascular permeability in surrounding tissues is indicated with a white arrow. Credit: Dr. Cristina Guerra-Giraldez, 2015

Treating neurocysticercosis (NCC), an infection of the brain with tapeworm larvae, often leads to inflammation and seizures when the parasites in the brain die. Now, researchers have reported in *PLOS Neglected Tropical Diseases*, using an animal model, that pretreatment with the anti-tumor necrosis factor drug etanercept (ETN) is a viable strategy to manage this post-treatment inflammation.

NCC, which is caused by the parasitic cestode *Taenia solium*, is a major cause of epilepsy in developing countries around the world. While treatments exist to kill the parasite, degenerating cysts in the brain provoke inflammatory responses that can cause a variety of neurological problems. Currently, physicians typically use corticosteroids to control this inflammatory response, but this comes at a high cost of severe side effects with long courses of treatment at high doses. Basic research has suggested that other antiinflammatory drugs including the tumor necrosis factor TNF- α blockers may suppress some pathways involved in the treatment-induced [inflammation](#).

In the new work, Siddhartha Mahanty of the National Institute of Allergy and Infectious Diseases, and colleagues from Universidad Peruana Cayetano Heredia in Lima, Peru, used pigs naturally infected with NCC to compare different treatment regimens. One group of pigs received the cysticidal drug praziquantel (PZQ) alone; another received PZQ preceded by two doses of the TNF- α blocker ETN; a third received PZQ as well as three doses of the steroid dexamethasone (DEX); and the last group was given no treatment.

Treatment with ETN before PZQ inhibited the levels of a number of genes known to regulate inflammation. The same degree of changes to gene levels were not seen in the other groups. The degree of inflammation was modestly decreased in both ETN and DEX pretreated animals. In addition, scores of cyst wall damage showed that pretreatment with ETN, unlike DEX, did not inhibit the damage to cysts

caused by PZQ.

"ETN, a licensed biologic, has been used for TNF- α blockade for over 20 years and has shown remarkable efficacy as an anti-inflammatory agent in rheumatoid arthritis and inflammatory bowel diseases; its safety profile is well known," the researchers say. "Our data demonstrate that TNF- α blockade induces potent suppression of post-[treatment](#) pericystic inflammation in a natural infection model of NCC."

More information: *PLOS Neglected Tropical Diseases*:
[journals.plos.org/plosntds/art ... journal.pntd.0006059](https://journals.plos.org/plosntds/article?id=10.1371/journal.pntd.0006059)

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