2-to-3-yr POST-DOCTORAL POSITION
NEUROIMMUNOLOGY & PARASITOLOGY

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Team 'Eukaryotic Pathogens: Inflammation, T cell immunity and Chemoresistance'
Toulouse Institute for Infectious and Inflammatory Diseases (INFINITy)

Background

Neuroinflammation is a hallmark of many brain diseases, including Alzheimer's disease (AD). Neuroinfllammation can be triggered by persistent brain infections such as the one caused by the *Toxoplasma gondii* (*T. gondii*) parasite. We and others have shown that latent *T. gondii* infection, which affects ~30% of the global population, has been linked to various neurological disorders (1) and can accelerate AD trajectory (in prep). Current anti-parasitic drugs can eliminate the disease's active tachyzoite stage but are largely ineffective against cyst-forming bradyzoites in neurons, which maintain chronic infection. Neuronal MHC I presentation (2) and brain-resident CD8+ T cells are crucial for controlling *T. gondii* parasites in the brain (3), but the ability of CD8+ T cells to recognize and target bradyzoite-infected neurons is limited due to reduced MHC I antigen presentation.

Our recent work (Gutierrez-Loli et al. in prep) revealed that bradyzoite-infected neurons fail to upregulate the antigen presentation machinery upon IFN-γ stimulation, suggesting an immune evasion strategy. Evidence suggests that such immune evasion is driven by parasite-secreted factors inducing epigenetic silencing of MHC genes, a mechanism reminiscent of tumor immune escape.

The goal of the ANR project DEFEND is to investigate and neutralize the mechanisms allowing *T. gondii* latent stages to escape CD8+ T cell immune surveillance. We will address 3 specific aims:

(i) identify the parasite effectors that prevent bradyzoite-infected neurons to present antigens to CD8+ T cells via MHC I molecules and unravel their mechanisms of action ;

(ii) investigate the impact of epigenetic modulators on *T. gondii* chronic brain parasite load ;

(iii) evaluate the therapeutic potential of epigenetic modulators to mitigate *T. gondii*-mediated neurological dysfunction, in particular the acceleration of Alzheimer's disease.

The post-doctoral researcher will work on one or a combination of these projects, using controlled experimental settings that enable us to study *T. gondii* infection *in vivo* and *in vitro*.

Context of the laboratory and position details

Our team is hosted at Toulouse Institute for Infectious and Inflammatory Diseases (INFINITy), a research Centre affiliated with Inserm, CNRS and University of Toulouse. Infinity provides a stimulating environment to perform state-of-the-art research in immunology and infectious diseases, with cutting-edge on-site technological facilities. Research teams at Infinity host international trainees, ranging from undergraduate students to postdocs. Seminars and lab meetings are held in English. Infinity is located in Toulouse, a dynamic and attractive city in the Southwest of France, ~1-hr away from Paris by plane.

The post-doctoral fellow will work in the context of an ANR-sponsored project (PRC DEFEND 2025-2029). Net salary will be adjusted depending on experience, starting at 2330 € / mo, including health benefits. The position is expected to start between **Nov 2025 and May 2026**.

Candidate profile

The postdoctoral fellow should be **highly self-motivated**, at ease with team work and able to independently organize his/her workload. Prior knowledge in neuroimmunology and/or infectious diseases, as well as research experience in transcriptomics and/or flow cytometry and/or mouse behavioral studies, would be beneficial, but is not mandatory. International candidates are strongly encouraged to apply. Applicants are invited to email a CV, a short statement of research interests and future goals + the names & contact information of 2 references to **N. Blanchard :** nicolas.blanchard@inserm.fr. Deadline: Sep 30th 2025

On-site interviews of short-listed candidates may be organized.

References

1. Belloy M, Schmitt BAM, Marty FH, Paut C, Bassot E, Aida A, et al. Toxoplasma gondii infection and chronic IL-1 elevation drive hippocampal DNA double-strand break signaling, leading to cognitive deficits. **Nature neuroscience. 2025**. Epub 2025/08/22.
2. Salvioni A, Belloy M, Lebourg A, Bassot E, Cantaloube-Ferrieu V, Vasseur V, et al. Robust Control of a Brain-Persisting Parasite through MHC I Presentation by Infected Neurons. **Cell reports. 2019**;27(11):3254-68 e8. Epub 2019/06/13.
3. Porte R, Belloy M, Audibert A, Bassot E, Aida A, Alis M, et al. Protective function and differentiation cues of brain-resident CD8+ T cells during surveillance of latent Toxoplasma gondii infection. **Proc Natl Acad Sci U S A. 2024**;121(24):e2403054121. Epub 2024/06/05.