

## INVITATION PUBLIC DEFENSE

Unravelling the mechanisms involved in the induction of the intestinal IL-17A response against the protozoan parasite *Giardia*

Charlotte Van Crombrugge  
12<sup>th</sup> February 2026 16.00h

## PROMOTORS

Prof. dr. P. Geldhof  
Faculty of Veterinary Medicine, UGent

Dr. L. Seys  
Faculty of Veterinary Medicine, UGent

## Curriculum Vitae

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Charlotte Van Crombrugge was born on July 8 1997 in Ghent. In 2015, she graduated from Sint-Lievenscollege in Ghent, where she completed an education in Greek–Mathematics. She subsequently enrolled in the study Veterinary Medicine at Ghent University.

During her bachelor studies, she participated in the competitive *Honours Programme in Life Sciences*, through which she gained her first in-depth exposure to scientific research. As part of this program, she joined the Laboratory of Parasitology at the Faculty of Veterinary Medicine, where she conducted her first independent research on the intestinal parasite *Giardia*.

During her master's thesis, Charlotte continued her research on *Giardia*, for which she received the award for Best Master's Thesis. In 2021, she obtained the degree of Master in Veterinary Medicine with the highest distinction. Immediately after graduation, she began her PhD at the Laboratory of Parasitology, funded by the Research Foundation Flanders (FWO).

Charlotte Van Crombrugge is author or co-author of multiple scientific publications and has been an invited speaker at both national and international conferences.

## Where?

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The defense will take place on:  
Thursday 12<sup>th</sup> February 2026 at 16.00h

Raadzaal  
Dean's building  
Faculty of Veterinary Medicine  
Ghent University, Campus Merelbeke  
Salisburylaan 133, Merelbeke

A reception will be held in classroom 1.1 (old library) on the 1<sup>st</sup> floor of the Dean's building (entrance 2).

## How to attend?

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If you would like to attend, please register before February 5<sup>th</sup> 2026 by e-mail to [charlotte.vancrombrugge@UGent.be](mailto:charlotte.vancrombrugge@UGent.be)

## Members of the Jury

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Prof. dr. S. Croubels  
Chairperson of the Jury

Dr. S. Rausch  
Institut für Immunologie, FU Berlin

Prof. dr. H. Favoreel  
Faculty of Veterinary Medicine, UGent

Prof. dr. L. Vereecke  
Faculty of Medicine and Health Sciences, UGent

Prof. dr. B. Dewals  
Faculté de Médecine Vétérinaire, ULiège

## Thesis Summary

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*Giardia duodenalis* is a globally prevalent intestinal protozoan parasite with a broad host range, capable of infecting both humans and animals. Giardiasis is associated with diarrhea, malabsorption and growth retardation, and can cause production losses in livestock. In recent years, interleukin-17A (IL-17A) has been identified as a central player in the protective immune response against *Giardia*. Following infection, IL-17A is strongly induced in the small intestine and activates various effector mechanisms, including antimicrobial peptide production, complement activation, and IgA production, all of which contribute to the elimination of the parasite from the intestine. Despite the importance of IL-17A, the anatomical origin of this response and the involved cellular sources remain incompletely characterized.

The aim of this thesis was therefore to determine where in the intestinal tract IL-17A is produced, by which cell types, and which gut-associated lymphoid structures are involved in the induction of this response.

Chapter 3 describes the cellular sources of IL-17A in *Giardia muris* infected C57BL/6 mice. The induction of IL-17A mRNA was largely restricted to the small intestine and was absent in the mesenteric lymph nodes. In T-cell knockout mice, IL-17A expression was strongly impaired, indicating that T cells are the main source of IL-17A during infection. Flow cytometric analysis showed that IL-17A production occurs predominantly in the lamina propria of the small intestine, with CD4<sup>+</sup> Th cells forming the dominant IL-17A-producing population. A limited contribution to IL-17A production was observed from macrophages, while other innate cell types showed no infection-induced IL-17A production.

Chapter 4 addresses the anatomical localization of the IL-17A response and the role of antigen-presenting cells located in the Peyer's patches. *Giardia* trophozoites were predominantly present in the proximal and middle regions of the small intestine, where the IL-17A response initially started after infection. IL-17A expression was increased only in Peyer's patches located in the proximal part of the small intestine. Conventional dendritic cells type 2 in the proximal Peyer's patches were observed migrating from the follicle-associated epithelium to the interfollicular region. However, blockade of lymphocyte migration from Peyer's patches affected neither the induction of IL-17A in the small intestine nor the elimination of *Giardia*, indicating that Peyer's patches are not the sole source of anti-giardial IL-17A induction.

Chapter 5 integrates these findings into an updated model of IL-17A-mediated immunity against *Giardia*, in which the proximal small intestine and Peyer's patches are identified as important sites of Th17 activity, with Th17 cells as the main source of anti-giardial IL-17A.

