



Universiteit
Leiden
The Netherlands

Toll-like receptor signaling in the innate immune system of zebrafish larvae

Yang, S.

Citation

Yang, S. (2016, December 20). *Toll-like receptor signaling in the innate immune system of zebrafish larvae*. Retrieved from <https://hdl.handle.net/1887/45057>

Version: Not Applicable (or Unknown)

License: [Licence agreement concerning inclusion of doctoral thesis in the Institutional Repository of the University of Leiden](#)

Downloaded from: <https://hdl.handle.net/1887/45057>

Note: To cite this publication please use the final published version (if applicable).

Cover Page



Universiteit Leiden



The handle <http://hdl.handle.net/1887/45057> holds various files of this Leiden University dissertation.

Author: Yang, S.

Title: Toll-like receptor signaling in the innate immune system of zebrafish larvae

Issue Date: 2016-12-20

Propositions

Accompanying the PhD thesis:

Toll-like receptor signaling in the innate immune system of zebrafish larvae

- 1 TLR signaling pathways are more important than previously expected in maintaining homeostasis of developing organisms. (This thesis)
- 2 TLR2 and TLR5 ligands induce distinct innate immunity expression signatures in zebrafish embryos. (This thesis).
- 3 TLR2 has a protective role in zebrafish larval defense against systemic *M. marinum* infection. (This thesis)
- 4 Tlr2 signaling contributes to granuloma formation at the very early steps of the infection process that are not dependent on T-cell activity. (This thesis)
- 5 A better understanding of the interaction between TLRs and their co-receptors is necessary in order to understand the similarities and differences of intracellular signaling induced by different TLRs.
- 6 There should be more attention to the negative control of TLR signaling by multiple mechanisms including dissociation of adaptor complexes, degradation of signal proteins, and transcriptional regulation. (T. Kondo, 2012. *Trends in Immunology*)
- 7 Zebrafish larvae can be used to study the reason why in mice a role of Tlr2 could only be shown at high doses of mycobacterial infection, whereas at a low dose no effect was observed of the mutation. (N. Reiling, 2002. *Journal of Immunology* and M. B. Drennan, 2004. *American Journal of Pathology*)
- 8 Generating a mutation closely after the ATG start codon is not a good idea.
- 9 Rescue mechanisms of gene defects in vertebrates are often not understood and present a big challenge in mutation construction and gene function analysis.
- 10 The statement “No pain, no gain”, is as modern today as it was 2000 years ago. (Zhang Heng, this thesis).