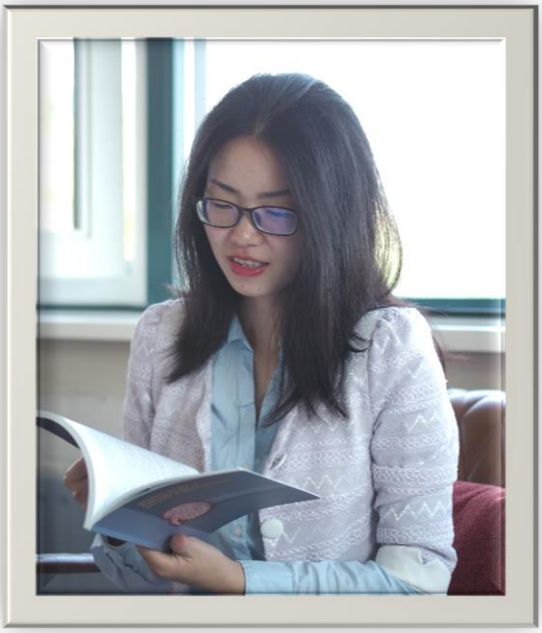
Functions of leptin in tuberculosis and diabetes: multi-omics studies across species

Ding, Y.

Curriculum Vitae



Yi Ding (Chinese: 丁义) was born in Chongqing, the People's Republic of China on 25th June, 1989. In September 2009 after graduating from high school, she started her bachelor studies at the College of Life Science, Hebei Normal University in Shijiazhuang, China. She did her internship in a middle school for six months and obtained a quantification certificate for high school teaching. In June 2013, she gained a bachelor diploma in biology. After that, she was admitted to the State key laboratory of biotherapy, Sichuan University in Chengdu, China. She was successful to get a scholarship at Sichuan University for her master studies. Her thesis project was about N-Acetylcysteine improvement of palmitate-induced myotube loss in skeletal muscle cells and high fat diet-induced muscle wasting in mice, under the supervision of Prof. dr. H. Xiao. In June 2016, she obtained a master diploma in cell biology. Subsequently, she worked in two biotechnical companies in China from July 2016 to August 2017. In September 2017, she was awarded a four-year grant from China Scholarship Council for PhD studies at the Institute of Biology, Leiden University, the Netherlands. The PhD projects focused on exploring the functions of leptin resulting from metabolic reprograming of tuberculosis and diabetes in mice and zebrafish, under the supervision of Prof. dr. H. P. Spaink and Prof. dr. A. Alia. The work of her PhD research is presented in this thesis. Currently from September 2021, she is working at the same institute as a post-doctoral fellow studying immune responses to *Mycobacterium avium* infection using zebrafish models.

List of publications

Ding Y, Haks MC, Forn-Cuní G, He J, Nowik N, Harms AC, Hankemeier T, Eeza MNH, Matysik J, Alia A, Spaink HP. Metabolomic and transcriptomic profiling of adult mice and larval zebrafish leptin mutants reveal a common pattern of changes in metabolites and signaling pathways. Cell Biosci. 2021 Jul 7;11(1):126.

He J*, **Ding Y***, Nowik N, Jager C, Eeza MNH, Alia A, Baelde HJ, Spaink HP. Leptin deficiency affects glucose homeostasis and results in adiposity in zebrafish. J Endocrinol. 2021 May;249(2):125-134.

Ding Y, Raterink RJ, Marín-Juez R, Veneman WJ, Egbers K, van den Eeden S, Haks MC, Joosten SA, Ottenhoff THM, Harms AC, Alia A, Hankemeier T, Spaink HP. Tuberculosis causes highly conserved metabolic changes in human patients, mycobacteria-infected mice and zebrafish larvae. Sci Rep. 2020 Jul 15;10(1):11635.

Ding Y, Haks MC, Ottenhoff THM, Harms AC, Hankemeier T, Eeza MNH, Matysik J, Alia A & Spaink HP. Leptin mutation and mycobacterial infection leads non-synergistically to the same metabolic syndrome.
Manuscript in preparation.

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