A warm welcome! New Group Leader Dr. Guoliang Cui

HI-TRON Mainz welcomes Dr. Guoliang Cui. Dr. Cui joins the HI-TRON Mainz with a focus on T-Cell metabolisms. His research focuses on CD8⁺ T cell immunometabolic checkpoints in cancer and infection using human samples and mouse models (inducible and transplanted melanoma, LCMV infection, influenza infection and listeria infection) to identify the key metabolic pathways that support or suppress protective CD8⁺ T cell function.
We welcomed him with three questions right at the start.

1) Why do you want to work at HI-TRON Mainz and what attracts you to the Mainz site?

HI-TRON does not only offer an excellent basic research environment, but also provides a great translational research environment. The translational research resources are required to carry out our research into the causes of immunosuppression and metabolic suppression in the tumor microenvironment. The clinical success of immune checkpoint inhibitors has revealed the power of harnessing immune cells (such as CD8⁺ T cells) in the treatment of cancers. Currently available immunotherapies are very promising, but also have limitations. In one clinical trial, about 35% of melanoma patients had no disease progression after receiving anti-PD-1 treatment, suggesting that the currently available checkpoint inhibitors alone probably cannot eradicate cancers for all patients. Our research combines human samples with mouse models to identify the key metabolic pathways causing T cell immunosuppression in the tumor microenvironment. HI-TRON has enormous pre-clinical and clinical resources that I need to advance our research with the hope to develop more powerful immunotherapies.

2) What is your research focus?

My group studies CD8⁺ T cell immunometabolic checkpoints in cancer and infection. I use human samples and mouse models (inducible and transplanted melanoma, LCMV infection, influenza infection and listeria infection) to identify the key metabolic pathways that support or suppress protective CD8⁺ T cell function. To identify these key pathways in CD8⁺ T cells, my laboratory has established a research pipeline. Using this research pipeline, we have identified a panel of metabolism-related proteins that essentially regulate effector T cell functions. For instance, we found that SPTLC2, a sphingolipid synthetic enzyme, promotes antiviral and antitumor effector CD8⁺ T cell survival and function (Wu et al., *Immunity*, 2019). Furthermore, we identified that oxidized lipids are enriched in the tumor microenvironment. These oxidized lipids were imported into CD8⁺ T cells through the lipid transportation protein CD36. Genetic or pharmacological inhibition of CD36 reversed the immunosuppression of CD8⁺ T cells, and inhibited tumor growth (Xu et al., *Immunity*, 2021). Some of the metabolic pathways can be targeted using antibodies and small-molecule compounds. My group is exploring the potential synergy of inhibiting these metabolic checkpoints together with the well-established immune checkpoints for the treatment of cancer and infectious diseases.

3) What did you do before?

I earned my PhD in Chinese Academy of Sciences in Shanghai (2010) and worked in GlaxoSmithKline until 2012. I returned to academia and did my postdoctoral training at Yale University (2012-2016). With the generous support of a Helmholtz Young Investigator Award, I started my junior research group at German Cancer Research Center (DKFZ, Heidelberg) in September, 2016. My junior research group was promoted to a Research Division at HI-TRON in September, 2021. Our research has attracted 5.5 million euro third-party funds by October 2021.