

GRK 2576 Guest Lecture

Title: Beta Cell Development and Regeneration

Speaker: Heiko Lickert, PhD

Director, Institute of Diabetes and Regeneration Research (IDR), Helmholtz Diabetes Center München, and Professor and Chair of Beta Cell Biology, Medical Faculty, Technical University Munich (TUM)

Date: 26. April 2023

Time: 14:00 h CET

Location: Oskar Minkowski-Saal & Paul Langerhans-Saal, DDZ
(please register with nicole.rockel@ddz.de by April 24th)

Zoom:

<https://us06web.zoom.us/j/86746758833?pwd=RHRkcmpDMW5saU5hcGdaOHdwQ3BIQT09>

Meeting-ID: 867 4675 8833; Kenncode: 425053

Biography



Heiko Lickert is a Full Professor and Chair of beta-cell biology in the Medical Faculty of the Technical University Munich (TUM) and is the Director of the Institute of Diabetes and Regeneration Research (IDR) and Adjunct Professor in the Institute of Stem Cell Research (ISF) at the Helmholtzzentrum München. He is in the Executive Committee of the Helmholtz Diabetes Zentrum (HDC) and is the newly elected incoming President of the GSCN, as well as in the Research Coordination Board of the German Center for Diabetes Research (DZD).

He obtained his PhD from the Albert-Ludwig University and Max-Planck Institute in Freiburg and his Postdoctoral studies

were carried out at the Mount Sinai Hospital, Toronto, Canada. He is an expert on organ development and tissue homeostasis with emphasis on endocrine lineage formation in the gut and pancreas, insulin-producing β -cell development, regeneration and replacement, as well as metabolic signaling and stem cell-based drug screening. His research has been funded by the European Research Council (ERC), a prestigious Emmy-Noether fellowship of the German Research Foundation (DFG), the Ministry of Education and Research (BMBF), the Alexander-von-Humboldt Foundation, the Helmholtz Association and the European Union.

The primary objective of his institute (IDR) at the Helmholtz Diabetes Center München is to pave the way for causal regenerative therapeutic approaches to stop or revert disease progression. Therefore the aim is 1) to understand the β -cell development, homeostasis and function for triggering *in vivo* regeneration of endogenous β -cells and 2) to improve current strategies for functional β -cell production *in vitro* with the ultimate goal to provide alternative sources of β -cells for cell replacement therapy in diabetes.

Selected recent publications

Aliluev, A; Tritschler, S; Sterr, M; Oppenländer, L; Hinterdobler, J; Greisle, T; Irmler, M; Beckers, J; Sun, N; Walch, A; Stemmer, K; Kindt, A; Krumsiek, J; Tschöp, MH; Luecken, M; Theis, F; Lickert, H and Böttcher, A (2021) *Diet-induced alteration of intestinal stem cell identity and lineage allocation underlies obesity and pre-diabetes in mice*. **Nature Metabolism** 3, 1202–1216.

Siehler, J; Blöchliger, A; Meier M and Lickert, H (2021) *Engineering islets from stem cells for advanced therapies of diabetes*. **Nature Reviews Drug Discovery** <https://doi.org/10.1038/s41573-021-00262-w>

Scheibner, K; Schirge, S; Burtscher, I; Büttner, M; Sterr, M; Yang, D; Böttcher, A; Ansarullah; Irmler, M; Beckers, J.; Cernilogar, FM; Schotta, G; Theis, FJ and Lickert H (2021) *Epithelial cell plasticity drives endoderm formation during gastrulation*. **Nat Cell Biol.** 23, 692–703

Ansarullah; Jain, C; Far, FF; Wißmiller, K; Homberg, S; Gräfin von Hahn, F; Raducanu, A; Schirge, S; Sterr, M; Bilekova, S; Siehler, J; Wiener, J; Oppenländer, L; Morshedi, A; Bastidas-Ponce, A; Collden, G; Irmler, M; Beckers, J; Feuchtinger, A; Grzybek, m; Ahlbrecht, C; Feederle, R; Plettenburg, O; Müller, T; Meier, M; Tschöp, M; Coskun, Ü and Lickert, H (2021) *Inceptor counteracts insulin signalling in β -cells to control glycaemia*. **Nature** 590, 326–331

***Information on access:** please visit <https://www.vivid.hhu.de/qualification-program/guest-lectures>

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Talk teaser

Guest lecture with Prof. Dr. rer. nat. Heiko Lickert

Beta Cell Development and Regeneration

Diabetes mellitus is a multifactorial disease with steadily increasing numbers of patients world-wide. Progression to insulin-dependent diabetes is characterized by the loss or dysfunction of pancreatic β -cells, but the pathomechanisms underlying β -cell failure in type 1 and 2 diabetes are still poorly defined. Regeneration of β -cell mass from residual β -cells or replacement by stem cell-derived β -cells holds great promise to stop or reverse disease progression. Thus, understanding how β -cells are formed from progenitors during embryonic development or are regenerated in diabetic mouse models is warranted to design novel cell-replacement and regenerative therapies for diabetic patients.