

Dr. med. Sebastian Wieland

Current position: Resident and Principal Investigator
Address: Department of General Internal Medicine and Psychosomatics, Heidelberg University Hospital
Thibautstraße 4, 69115 Heidelberg
Phone: +49 (0) 6221 - 56 34180
Mail: sebastian.wieland@med.uni-heidelberg.de
Birth Date: 21.08.1984

Scientific vita:

Since 2019 Principal Investigator at CRC1158, Project Bo4: "Translational studies in pain chronicity: role of corticothalamic pathways in stress-sensitization and comorbidity development", Heidelberg University, Germany
Since 2018 Resident, Department of General Internal Medicine and Psychosomatics, Heidelberg University Hospital
2016-2018 Postdoctoral fellow, Prof. Dr. Thomas Kuner, Institute of Anatomy and Cell Biology, Heidelberg University
2014-2015 Residency, Department of Sports Medicine, Heidelberg University Hospital
2010-2016 Doctoral degree in psychiatry (summa cum laude), Heidelberg University, Germany.
Mentor: Prof. Dr. Wolfgang Kelsch
2009 Medical approbation
2003-2009 Medical school, Heidelberg University, Germany

Grants, Awards and Honors:

2019 Project Bo4 CRC1158, Principal Investigator
2016 Physician-Scientist- Fellowship, Heidelberg University

Key publications:

- Oettl LL*, Scheller M*, Filosa C*, Wieland S, Haag F, Loeb C, Durstewitz D, Shusterman R, Russo E*, Kelsch W* (* equal contribution); *Phasic dopamine reinforces distinct stimulus encoding in the olfactory tubercle driving dopaminergic reward prediction.* **Nat commun.** 2020 Jul 10;11(1):3460
- Wieland S, Schindler S, Huber C, Köhr G, Oswald MJ, Kelsch W. *Phasic Dopamine Modifies Sensory-Driven Output of Striatal Neurons through Synaptic Plasticity.* **J. Neurosci.** 2015 Jul 8;35(27):9946-56.
- Kelsch W*, Li Z*, Wieland S*, Senkov O, Herb A, Göngrich C, Monyer H. *GluN2B-containing NMDA receptors promote glutamate synapse development in hippocampal interneurons.* **J. Neurosci.** 2014 Nov 26;34(48):16022-30. (* equal contribution)
- Wieland S, Du D, Oswald MJ, Parlato R, Köhr G, Kelsch W. *Phasic dopaminergic activity exerts fast control of cholinergic interneuron firing via sequential NMDA, D₂, and D₁ receptor activation.* **J. Neurosci.** 2014 Aug 27;34(35):11549-59. (cover article, highlighted by the editor)