C U R R I C U L U M V I T A E Helmut KUBISTA

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University and Scientific Career

1986 - 1992	Graduate Studies on Biology/Zoology, Faculty of Natural Sciences and Institute of Zoology, Paris-Lodron-University of Salzburg (Austria).
1990	Specialisation in Neurophysiology und Biochemistry.
1991 - 1992	Diploma thesis at the Department of Animal Physiology, titled ”Localisation and Function of S-100-immunoreactive Material in the central nervous system of *Helix pomatia*”.
12.11.1992	Graduation (title: Mag. rer. nat.) at the Paris Lodron-University of Salzburg.
1992 - 1995	Doctoral thesis at the Department of Animal Physiology, titled ”Electrophysiological Investigations on the Function of Calcium-Binding-Proteins in Nerve Cells“.
10/1995 - 08/1996	Alternative civilian service at a nursing home for the elderly.
20.02.1997	Graduation (title: Dr. rer. nat. PhD) at the Paris-Lodron-University of Salzburg.
02/1997 - 09/1999	Postdoctoral research fellow in an EC-funded project titled “Structure and Function of Annexins” (project number: ERBIO960034) at the Department of Physiology, University College London (lab of Stephen E. Moss).
01/2000 - 03/2007	Postdoctoral research fellow at the Institute of Pharmacology of the Faculty of Medicine, University of Vienna, now Center of Physiology and Pharmacology, Medical University of Vienna.
05/2007 - 7/2011	University Assistant at the Center of Physiology and Pharmacology, Medical University of Vienna.
since 05/2007	Principal Investigator
since 07/2011	Assistant Professor at the Center of Physiology and Pharmacology, Medical University of Vienna.
07/2014	Promotion to Assoc. Professor (ibid.).

Main research area(s):

- Role of L-type voltage-gated calcium channels in neuronal excitability and epileptiform discharge activities.
- Role of paroxysmal depolarization shifts in epilepsy and epileptogenesis
- Role of mitochondria in neuropathogenesis

The main research achievements of Dr. Kubista can be summarized as follows:

- Demonstration of a channel subtype-dependent bimodal regulation exerted by L-type calcium channel-mediated Ca^{2+} influx on the excitability of central neurons (publications # 7 and 9, listed below).
- Demonstration of a crucial implication of L-type calcium channels and in particular of the $Ca_v1.3$ subtype of L-type calcium channels in the formation of paroxysmal depolarization shifts (PDS) (publications # 2, 5 and 8).
- A definition of bona fide PDS and of the main research questions that need to be solved to understand the role of PDS in epileptology (publications # 4).
- Identification of a bimodal regulation of mitochondrial function by L-type calcium channels, including the demonstration of the induction of mitoprotective mechanisms by high L-type calcium channel-mediated Ca^{2+} loads (publication # 3).
- Demonstration that rises in cytosolic Ca^{2+} via L-type Ca^{2+} channels adjust mitochondrial ATP production to energetic needs during neuronal activity and an involvement therein of glycerol-3-phosphate shuttle, which is also driven by Ca^{2+} influx via this type of channels (publication # 1).
- Subtype-selective modulation of $GABA_A$ receptors by the anticonvulsant drug retigabine (publication # 6)
- Evidence of a physiological role of higher order complexes of SNARE proteins in neuronal exocytosis (publication # 10).

10 most important publications:

1: Dhoundiyal A, Goeschl V, Boehm S, **Kubista H**, Hotka M. Glycerol-3-phosphate shuttle is a backup system securing metabolic flexibility in neurons. J Neurosci. 2022 Aug 19:JN-RM-0193-22. doi: 10.1523/JNEUROSCI.0193-22.2022. Epub ahead of print. PMID: 35999055.
2: Meyer C, Kettner A, Hochenegg U, Rubi L, Hilber K, Koenig X, Boehm S, Hotka M, **Kubista H**. On the Origin of Paroxysmal Depolarization Shifts: The Contribution of Cav1.x Channels as the Common Denominator of a Polymorphous Neuronal Discharge Pattern. Neuroscience. 2021 Aug 1;468:265-281. doi: 10.1016/j.neuroscience.2021.05.011.
3: Hotka M, Cagalinec M, Hilber K, Hool L, Boehm S, **Kubista H**. L-type Ca^{2+} channel-mediated Ca^{2+} influx adjusts neuronal mitochondrial function to physiological and pathophysiological conditions. Sci Signal. 2020 Feb 11; 13(618):eaaw6923, doi: 10.1126/scisignal.aaw6923.
4: **Kubista H**, Boehm S, Hotka M. The Paroxysmal Depolarization Shift: Reconsidering Its Role in Epilepsy, Epileptogenesis and Beyond. Int J Mol Sci. 2019 Jan 29;20(3). doi: 10.3390/ijms20030577.

5: Stiglbauer V, Hotka M, Ruiß M, Hilber K, Boehm S, **Kubista H.** $Ca_v1.3$ channels play a crucial role in the formation of paroxysmal depolarization shifts in cultured hippocampal neurons. Epilepsia. 2017 May;58(5):858-871. doi: 10.1111/epi.13719.
6: Treven M, Koenig X, Assadpour E, Gantumur E, Meyer C, Hilber K, Boehm S, **Kubista H.** The anticonvulsant retigabine is a subtype selective modulator of GABAA receptors. Epilepsia. 2015 Apr;56(4):647-57. doi: 10.1111/epi.12950.
7: Hasreiter J, Goldnagl L, Böhm S, **Kubista H.** Cav1.2 and Cav1.3 L-type calcium channels operate in a similar voltage range but show different coupling to Ca(2+)-dependent conductances in hippocampal neurons. Am J Physiol Cell Physiol. 2014 Jun 15;306(12):C1200-13. doi: 10.1152/ajpcell.00329.2013.
8: Rubi L, Schandl U, Lagler M, Geier P, Spies D, Gupta KD, Boehm S, **Kubista H.** Raised activity of L-type calcium channels renders neurons prone to form paroxysmal depolarization shifts. Neuromolecular Med. 2013 Sep;15(3):476-92. doi: 10.1007/s12017-013-8234-1.
9: Geier P, Lagler M, Boehm S, **Kubista H.** Dynamic interplay of excitatory and inhibitory coupling modes of neuronal L-type calcium channels. Am J Physiol Cell Physiol. 2011 Apr;300(4):C937-49. doi: 10.1152/ajpcell.00219.2010.
10: **Kubista H**, Edelbauer H, Boehm S. Evidence for structural and functional diversity among SDS-resistant SNARE complexes in neuroendocrine cells. J Cell Sci. 2004 Feb 29;117(Pt 6):955-66. PubMed PMID: 14762114.

Grants received:

as project leader:

FWF P-36145: The role of paroxysmal depolarization shifts in epilepsy. 01.01.2023-31.12.2025
FWF P-28179: The link between PDS and mitochondria in epileptogenesis. 04.01.2016-03.07.2020
Herzfelder`sche Familienstiftung: Are paroxysmal depolarizaton shifts a common pathological factor in neurological diseases of the elderly? 01.04.2015-31.03.2018
FWF P-19710: New targets for anti-epileptic drugs. 01.05.2007-30.04.2012

and as co-applicant:

FWF P-33799: Glutamate toxicity mediated by mitochondria. Ongoing (starting date 01.03.2021); together with project leader Prof. Andrey Kozlov (Ludwig Boltzmann Institute for Experimental and Clinical Traumatology, Vienna, Austria).

Important recent conference contributions:

Organization and chairing of a parallel session at the 13th European Conference on Epileptology, titled: “Epilepsy and mitochondria” (http://epilepsyvienna2018.org/scientific-programme/parallel-sessions/).