## **Meet Our Editorial Board Member**

## **Gustav Akk**

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Dr. Gustav Akk obtained his Ph.D. in biophysics from the State University of New York at Buffalo. Following postdoctoral training in the Department of Neurology and Neurological Sciences at Stanford University, he joined the Department of Anesthesiology at Washington University School of Medicine, initially, as a research associate and then, as a faculty member. Dr. Akk is presently an Associate Professor of Anesthesiology, a member of the Center for Investigation of Membrane Excitability Diseases (CIMED), and a core member of the Taylor Family Institute for Innovative Psychiatric Research. Gustav Akk is an author of over 90 peer-reviewed publications. His research interests include physiology and pharmacology of members of the Cys-loop family of transmitter-gated ion channels, with particular emphasis on how the interplay between endogenous and clinical GABAergic compounds, including neurosteroids and anesthetic drugs, shapes the inhibitory activity in the brain.



## SELECTED PUBLICATIONS

- Akk, G., Auerbach, A. Inorganic, monovalent cations compete with agonists for the transmitter binding site of nicotinic acetylcholine receptors. *Biophys. J.*, 1996, 70, 2652-2658.
- [2] Auerbach, A., Akk, G. Desensitization of mouse nicotinic acetylcholine receptor channels. A two-gate mechanism. J. Gen. Physiol., 1998, 112, 181-197.
- [3] Akk, G., Steinbach, J.H. Activation and block of recombinant GABA<sub>A</sub> receptors by pentobarbitone: a single-channel study. *Br. J. Pharmacol.*, **2000**, *130*, 249-258.
- [4] Akk, G. Aromatics at the murine nicotinic receptor agonist binding site: mutational analysis of the αY93 and αW149 residues. J. Physiol., 2001, 535, 729-740.
- [5] Akk, G., Bracamontes, J., Steinbach, J.H. Pregnenolone sulfate block of GABA<sub>A</sub> receptors: mechanism and involvement of a residue in the M2 region of the α subunit. J. Physiol., 2001, 532, 673-684.
- [6] Akk, G., Bracamontes, J.R., Covey, D.F., Evers, A., Dao, T., Steinbach, J.H. Neuroactive steroids have multiple actions to potentiate GABA<sub>A</sub> receptors. J. Physiol., 2004, 558, 59-74.
- [7] Akk, G., Shu, H.J., Wang, C., Steinbach, J.H., Zorumski, C.F., Covey, D.F., Mennerick, S. Neurosteroid access to the GABA<sub>A</sub> receptor. J. Neurosci., 2005, 25, 11605-11613.
- [8] Li, P., Shu, H.J., Wang, C., Mennerick, S., Zorumski, C.F., Covey, D.F., Steinbach, J.H, Akk. G. Neurosteroid migration to intracellular compartments reduces steroid concentration in the membrane and diminishes GABA<sub>A</sub> receptor potentiation. *J. Physiol.*, 2007, 584, 789-800.
- [9] Li, P., Akk, G. The insecticide fipronil and its metabolite fipronil sulphone inhibit the rat  $\alpha 1\beta 2\gamma 2L$  GABA<sub>A</sub> receptor. *Br. J. Pharmacol.*, **2008**, *155*, 783-794.
- [10] Li, P., Eaton, M.M., Steinbach, J.H., Akk, G. The benzodiazepine diazepam potentiates responses of α1β2γ2 γ-aminobutyric acid type A receptors activated by either γ-aminobutyric acid or allosteric agonists. *Anesthesiology*, **2013**, *118*, 1417-1425.
- [11] Li. P., Bracamontes, J.R., Manion, B.D., Mennerick, S., Steinbach, J.H., Evers, A.S., Akk, G. The neurosteroid 5β-pregnan-3α-ol20one enhances actions of etomidate as a positive allosteric modulator of α1β2γ2L GABA<sub>A</sub> receptors. *Br. J. Pharmacol.*, **2014**, *171*, 5446-5457.
- [12] Shin, D.J., Germann, A.L., Steinbach, J.H., Akk, G. The actions of drug combinations on the GABA<sub>A</sub> receptor manifest as curvilinear isoboles of additivity. *Mol. Pharmacol.*, 2017, 92, 556-563.
- [13] Cao, L.Q., Montana, M.C., Germann, A.L., Shin, D.J., Chakrabarti, S., Mennerick, S., Yuede, C.M., Wozniak, D.F., Evers, A.S., Akk, G. Enhanced GABAergic actions resulting from the coapplication of the steroid 3α-hydroxy-5α-pregnane-11,20-dione (alfaxalone) with propofol or diazepam. Sci. Rep., 2018, 8, 10341.
- [14] Germann, A.L., Steinbach, J.H., Akk, G. Application of the co-agonist concerted transition model to analysis of GABA<sub>A</sub> receptor properties. *Curr. Neuropharmacol.*, 2019, 17, 843-851.
- [15] Germann, A.L., Pierce, S.R., Senneff, T.C., Burbridge, A.B., Steinbach, J.H., Akk, G. Steady-state activation and modulation of the synaptic-type α1β2γ2L GABA<sub>A</sub> receptor by combinations of physiological and clinical ligands. *Physiol. Rep.*, 2019, 7, e14230.