



S. Steinlechner

Stephan Steinlechner

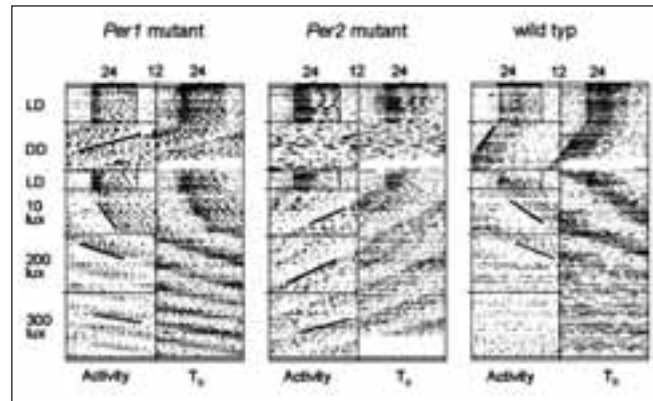
- 1977** Diploma in Biology at Ludwig-Maximilians-University Munich, Germany
- 1980** Dr. phil. nat. at J.W. Goethe-University Frankfurt a. M., Germany

Postdoctoral Fellow at University of Texas, Health Science Center at San Antonio, USA

Research Assistant at Philipps-University Marburg, Germany, Department of Biology
- 1987** Habilitation in Zoology at Philipps-University Marburg, Germany
- Since 1992** Professor of Comparative Physiology, Institut of Zoology, School of Veterinary Medicine Hannover

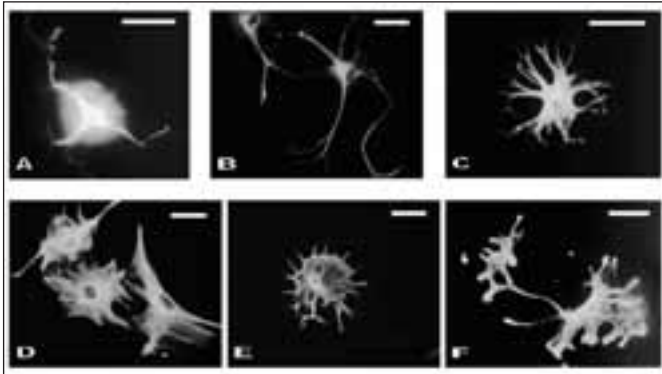
Current Research

The main focus of our interests centers around the question how environmental signals (e.g. light, temperature) are converted into endocrine signals within the central nervous system of small mammals, and how these endocrine signals are converted by the organism into physiological adaptations. The „photoneuroendocrine system“, i.e. the retina, the suprachiasmatic nuclei (SCN, the „master clock“) and the pineal gland, play a major role in this neurochemical transduction pathway. We study aspects of signal transduction from the retina to the SCN (PACAP and NMDA receptors, pCREB, cFos) and the distribution of neuropeptides and neurotransmitters as well as expression of „clock genes“ (*mPer1*, *mPer2*) within the SCN. Cell and organ culture systems are used to study paracrine mechanisms involved in the regulation of melatonin synthesis in the pineal gland which can be considered as one of the main output signals of the circadian clock. Because there are numerous melatonin receptors to be found in the SCN, a feedback regulation of the output signal melatonin to the pacemaker in the SCN is proposed. This hypothesis will be tested by application of melatonin into the SCN at different times of the circadian cycle and by simultaneously measuring with microdialysis techniques the melatonin production in the pineal gland.



Locomotor activity (left side of each panel) and body temperature rhythm (T_b, right side of each panel) of *Per1* mutants, *Per2* mutants and wild type mice under different light conditions as indicated on the left. LD = 12 h of light and 12 h of darkness; DD = constant darkness; 10 lux, 200 lux, 300 lux = illuminance under constant light conditions. Solid lines eye-fitted through onsets of activity illustrate change of endogenous period under the different constant conditions. Note that in *Per1* mutant mice the endogenous clock slows down with increasing illuminance, whereas *Per2* mutants' clock accelerates (Steinlechner et al. 2002).

Recently we started a project in collaboration with the MPI Hannover (Prof. G. Eichele/Dr. H. Oster) and Prof. U. Albrecht (University Fribourg/Switzerland) to characterize physiologically and behaviorally transgenic mice that are deficient of the major clock genes *mPer1* and/or *mPer2*. These genes are expressed with a circadian profile in the SCN of mammals. Mice or other rodents bearing mutations in these or other clock genes are valuable tools to



Different morphologies of astrocytes from the SCN of hamsters (GFAP immunostaining):

(A) + (B) tripolar, prismatic astrocytes, (C) multipolar, fibrillary astrocyte; (D) + (E) multipolar astrocytes with protoplasmic morphology. 400 x; all scale bars 25 μ m.

analyse both the cellular mechanisms of the central pacemaker and the neurological and physiological consequences of malfunctions of the circadian clock. In addition to these phenotyping studies we want to test in wild type and mutant mice how the cellular clocks in individual SCN neurons communicate with each other and how this leads to a synchronized output of the circadian pacemaker(s). In particular, we want to test the hypothesis that SCN glia cells, by calcium signalling via gap junctions, are responsible for this synchronization.

Selected Publications

- [1] Klante G, Secci K, Masson-Pévet M, Pévet P, Vivien-Roels B, Steinlechner S, Wollnik F. (1999) Interstrain differences in activity pattern, pineal function and SCN melatonin receptor density of ACI, BH and LEW rats. **Am. J. Physiol.** 276:R1078-R1086
- [2] Redecker P, Pabst H, Löscher W, Steinlechner S. (2001) Evidence for microvesicular storage and release of glycine in rodent pinealocytes. **Neuroscience Letters** 299:93-96.
- [3] Steinlechner S, Jacobmeyer B, Scherbarth F, Dernbach H, Kruse F, Albrecht U. (2002) Robust circadian rhythmicity of Per1 and Per2 mutant mice in constant light and dynamics of Per1 and Per2 gene expression under long and short photoperiods. **J Biol Rhythms** 17:202-209
- [4] Steinlechner S, Stieglitz A, Ruf T. (2002) Djungarian hamsters; a species with a labile circadian pacemaker? Arrhythmicity under a light-dark cycle induced by short light pulses. **J Biol Rhythms** 17:248-259
- [5] Steinlechner S, Puchalski W. (2002) Mechanisms for seasonal control of reproduction in small mammals. In: Environmental Signal Processing and Adaptation. G. Heldmaier and D. Werner (eds.) Springer Verlag, Berlin, Heidelberg, New York, pp. 233-250

Group Structure

Group leader: Stephan Steinlechner
 Graduate students: Frauke Perl, Robert Dallmann, Frank Scherbarth, Annika Herwig, Nadja Ufer
 Technicians: Siegfried Hilken, Marianne Brüning

Contact

Prof. Dr. Stephan Steinlechner
 Department of Zoology
 School of Veterinary Medicine
 Bünteweg 17
 30559 Hannover
 Germany
 Phone: +49-511-953-8450
 Fax: +49-511-953-8586
 Email: stephan.steinlechner@tiho-hannover.de
 Web: <http://www.tiho-hannover.de/einricht/zoo/sstein/index.htm>