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CV

Education:

1982-1987	Student of Biology, FA University Erlangen/Nürnberg
1987	Diploma (Biology), emphasize on Microbiology, Human Genetics and Computer Sciences, University of Erlangen/Nürnberg.
1988-1992	Doctoral thesis: "Molecular mechanisms of the regulation of tetracycline resistance determinants", major supervisor: Prof. Dr. Wolfgang Hillen
1992-1995	Postdoctoral research fellow in the lab of Professor Gary Ruvkun at the Harvard Medical School / Mass. General Hospital, Boston MA, working on the role of the gene unc-86 for the specification and differentiation of the C.elegans nervous system
1995-2000	Group leader at the Genzentrum of the Ludwig-Maximilians-University of Munich
since Nov. 2000	Professor of Biochemistry at the University of Munich, Medical Faculty

Research visits:

1989	Karolinska Institute Stockholm, Sweden. Work in the lab of Prof. Dr. Alexander von Gabain on the translation initiation and decay of tetR mRNA
1991	Institute of Crystallography, Free University of Berlin. Molecular modelling of Tet repressor-tet operator interactions in the lab of Prof. Dr. Wolfram Saenger.

Awards and Prizes:

1987	Eva-Schleip Stipend, awarded from the University of Erlangen
1993	VAAM-Promotionspreis (Graduation Prize) 1993, awarded by the "Vereinigung für Allgemeine and Angewandte Mikrobiologie e.V.", Germany
2001	Philip-Morris-Research Prize, Europe

CONTACT INFORMATION

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C.elegans, an Animal Model for the Functional Analysis of Human Disease Genes

The different genome projects have resulted in an exponential increase in sequence information available in the databases. At the same time, the number of functionally characterized genes is only increasing linearly. How can we increase the speed of functional genomics to make full use of the data mining? Model organisms have helped significantly to understand the roles of particular genes in an organism. The classical approaches to address gene function first involves the inactivation of a given gene and the monitoring of the resulting consequences. For single factors, this method was successfully used in the model organisms Drosophila melanogaster and mouse. However, in order to upscale this knock-out methodology and subsequent analysis, these models have a significant disadvantage: the time and effort to perform even single targeted gene manipulations is significant, and the complexity of the organism prevents in many cases the detailed analyses of the KO consequences. Here, the nematode C. elegans offers several advantages: 40-60 % of the human disease genes are represented by homologues in C. elegans. In addition, the animals are small enough to be kept in large numbers in a format that allows mass manipulations (microtiter plates) and knock-outs of candidate genes can be obtained in a matter of 4-6 weeks. Animal facilities are cheap, and C. elegans is the only multicellular organism for which the development of each single cell and the entire connectivity of its nervous system are known. At the same time, the cellular diversity of the C. elegans nervous system is in the same range as that of a vertebrate brain, although the total number of neurons is only 302. Remarkably, C. elegans neurons use the same neurotransmitters as humans, and the receptor pharmacology is astonishingly similar.

In this seminar, examples of functional conservation of human genes and their C. elegans counterparts/homologues will be discussed. In particular, I will focus on genes involved in human neurodegenerative diseases and discuss the contribution C. elegans models can make to understand the function of the relevant human disease genes.