Thomas Meitinger

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CV

EDUCATION:

1973-1981	Biology, Ludwig-Maximilians-Universität München (LMU)
1978-1982	Medicine, Ludwig-Maximilians-Universität München (LMU)
1982-1983	Final year medical student, Bharagwanath Hospital, Soweto, University of Witwatersrand, Johannesburg
1983	MD thesis, "Sequence and copy number of a repetitive element in the mouse genome" - Institut für Physiologische Chemie der Universität München

(Prof. Dr. H.-G. Zachau)

DEGREES:

1991	Diploma, Biology, LMU München
1983	MD, LMU München
1993	Board Certification for Medical Genetics
2000	Professor of Human Genetics, Technische Universität München

POSITIONS HELD:

1981	Research Fellow, Institut für Physiologische Chemie der Universität München (LMU) (Prof. Dr. H.-G. Zachau)
1982	Final year student, Gold Fields West Hospital, Westonaria, Johannesburg
1984-1985	Assistent, Kinderchirurgische Klinik, Karlsruhe, Akad. Lehrkrankenhaus der Universität Freiburg (Prof. Dr. W. Maier).
1985-1988	DFG/Royal Society fellowship, Genetics Laboratory, Dept. of Biochemistry, University of Oxford (Prof. J. Edwards)
1988-2000	Head, Molecular Genetics Laboratory, Department of. Medical Genetics, Kinderklinik der LMU München (Prof. J. Murken)
Oct. 2000	Director, Institute of Human Genetics, Klinikum rechts der Isar, Technische Univeristät München Director, Institute of Human Genetics, GSF National Research Centre, Neuherberg

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Complexity in Genetic Eye Disease

Over the past decade, remarkable progress has been made on molecular genetic characterisation of the eye. More than 50 genes with mutations causing retinal degenerations have been identified, many more have been mapped. Allelic heterogeneity is also a common theme with for instance more than 100 different mutations known in the rhodopsin gene. Experimental strategies used for the elucidation of monogenic disease are now being adapted to the study of multifactorial disease such as glaucoma and age related macular dystrophy, with limited success so far. This is hardly surprising given the underlying heterogeneity observed in monogenic disorders which is augmented by phenomena such as phenocopies and clinical misclassifications. The identification of genes involved in degenerative eye disorders has increased our knowledge base about mechanisms of neurodegeneration but in general it has still not provided clues for individual therapies. There is hope however that a unifying concept of neuroprotective strategies will emerge to combat the complex issue of genetic eye disease.