Fries laboratory

Prof. Dr. Jochen Fries

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Schwerpunkte: Glomeruläre Erkrankungen, chronische Niereninsuffizienz, Signaltransduktion

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Grants

Research has been and is continuously supported by different funding sources outside and inside the university.

Outside funding sources:

1. Marga und Walter Boll Stiftung
2. Private donations

Inside funding sources:

1. Köln Fortune
2. Imhoff-Stiftung

Research background Prof. Dr. Jochen Fries

In the experimental thesis (Prof. Thoenes, Pathology, Univ. Clinic, Mainz, Germany) a backleak of tubular fluid caused by tubular necrosis in the S3 segment due to a continued ischemic environment was shown by light and electron microscopy as cause of prolonged acute renal failure. A morphometric analysis of glomerular epithelial damage in proteinuric renal disease (1980-1984) demonstrated that only a small amount of all glomerular epithelial podocytes is altered in proteinuric/hematuric diseases in adults and children independent of the degree of...
proteinuria. Studying mechanism of proteinuria in a DFG-funded project at the Brigham and Women’s Hospital, Boston, MA, USA (Pathology, Laboratory of Prof. Dr. H. Rennke; 1984-1987) an immune complex mediated versus hypertensive model of glomerular disease was established showing the importance of the location immune complex deposition versus hemodynamic forces as determinants for progressive glomerular damage. A molecular biology project at the Harvard School of Public Health (Public Health; Prof D. Wirth, 1987-1989) in molecular mycobacteriology provided the basis for future studies at the molecular level, establishing DNA probes for diagnostic analyses for M. avium and the genus Mycobacteria. Studies in molecular vascular research (PDGF-transgenic mouse; cloning and characterisation of E-selectin and vascular cell adhesion molecule-1; Pathology, Brigham and Women’s Hospital; Prof. T. Collins; 1987-1997) as well as working as a consultant for experimental animal research (Prof. J. Halperin; Lab. of Transport Physiology; Harvard Medical School; 1994-1997) became important steps for future studies. In Köln (Pathology; since 1997) a microdissection method for analysing glomerular disease in formalin-fixed, paraffinized biopsies was established funded by the DFG.

The availability of virus-targeted gene delivery as well as the stem cell technology have been part of the research repertoire for renal and cardiac disease through scientific collaboration. The participation in the program of the DSO (Deutsche Stiftung Organtransplantation) in pre-transplant organ evaluation as well as the evaluation of non-allocated livers and kidneys have led to an intense interest in the problems of organ transplantation.

Over the last 5 years, research efforts have focussed on the role of endothelin in progressive renal disease and renal tumors. The signalling pathway via the ET-A-receptor and a cytoplasmic transcription complex consisting NF-κBp65, MAPKp38 and PKCα migrating into the nucleus has been analysed in normal and tumorous proximal tubule cells/human tumors.

Current interests of the Fries laboratory

1. Chronic Renal Disease
2. Cardiac/Vascular Disease
3. Molecular Therapy/Transplantation/Stem Cell Technology

Future projects

1. Activation of nuclear p16
2. Role of an ET-1 induced microRNA in proteinuric renal disease
3. Role of PKC in oncocytic transformation
4. Molecular analysis of a subtype of clear cell renal carcinoma

Publications

1. Kidney | Molecular Nephropathology


1. Fries JWU, Pakula A, Roth T, Dienes H-P, Odenthal M.


1. H Loeser, M von Brandenstein, A Herschung, M Schlosser, Buettner R, JWU Fries Endothelin-1 influences Multiple Drug Resistance in the human renal proximal tubule via microRNA 133a downregulating the MRP2 transporter, Life Sciences, under review


2. Heart | Vascular Research


1. Novel cis-acting elements in the human platelet-derived growth factor


3. Molecular Therapy | Transplantation | Stem cell Technology

1. Philipp J, Unruh M, Wagener A, Fries JWU, Gottstein C. The in vivo coagulating activity of soluble tissue factor is selectively enhanced on tumor endothelium by endotoxin. Arteriosklerosis, Thrombosis and Vascular Research 2003; 23: 905-910


