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Academic Background:

Ph.D., Pharmaceutical Sciences, University of Nebraska Medical Center, 1999
B.S., Medical Technology, University of Nebraska Medical Center, 1988
B.S., Computer Science, Winona State University, 1986

Professional Experience:

01/2004 – present	Assistant Professor, Pharmacology, University Missouri-Kansas City
10/1999 – 01/2004	Research Associate/ Postdoctoral Fellow in the Pharmacology Department, College of Medicine, University of Arizona - Tucson, AZ
07/1990 – 07/1993	Clinical Instructor, Medical Technology Program, University of Nebraska Medical Center (1990-1993)

Research Interests:

Blood-brain barrier (BBB), inflammation, cytokines, tight junctions, neurological diseases

Research interests in the Mark laboratory are focused on understanding cellular signaling mechanisms and pathways of endothelial barrier systems, especially the cerebral microvasculature, i.e., the blood-brain barrier (BBB). In particular, our research investigates functional and structural disturbances of the BBB due to stressors such as chronic inflammation and the milieu of cytokines and mediators that are released into the circulation as part of the pathology.

Our research program examines intracellular mechanisms and pathways (i.e., signal transduction, gene regulation, protein expression, etc.) of endothelial barrier systems that are stimulated by pathological insults, which lead to perturbations of the BBB. This is particularly important in several diseases with an inflammatory component where cerebral edema and neurological dysfunction have been reported.

These research efforts are aimed to provide a better understanding of the cellular mechanisms involved

in the cytoskeleton and tight junctional complexes (structural changes), changes of microvascular barriers that result in disturbances in cellular homeostasis (i.e., altered transport, increased paracellular permeability, edema, and multi-organ failure). Understanding the mechanisms by which these pathological stressors induce endothelial cell alterations will ultimately lead to improved pharmacological therapy for patients with neurological dysfunction such as multiple sclerosis, ischemic stroke, Alzheimer's disease, and chronic inflammation such as liver fibrosis.

Models and techniques used in our laboratory include: *in vitro* cell culture, Western blotting, permeability assays, immunohistochemistry, RT-PCR, enzyme assays, and ELISA.

Representative Publications:

Huber, J.D., Campos, C.R., **Mark, K.S.**, and Davis, T.P., Alterations in Blood-brain Barrier ICAM-1 Expression and Brain Microglial Activation Following λ-carrageen-induced Inflammatory Pain, *Am J Physiol (Heart Circ Physiol)*, 290: H732-H740, (2006).

Witt, K.A., **Mark, K.S.**, Huber, J.D., and Davis, T.P., Hypoxia Inducible Factor and Nuclear Factor Kappa-β Activation in Blood-Brain Barrier Endothelium Under Hypoxic/Reoxygenation Stress, *J Neurochem*, 92: 203-214, (2005).

Brown, R.C., **Mark, K.S.**, Egleton, R.D., and Davis, T.P., Protection Against Hypoxia-Induced Blood-Brain Barrier Disruption: Changes in Intracellular Calcium, *Am J Physiol (Cell Physiol)*, 286 (5): C1045-52 (2004).

Mark, K.S., Burroughs, A.R., Brown, R.C., Huber, J.D., and Davis, T.P., Nitric Oxide Mediated Changes in Permeability During Hypoxia and Reoxygenation, *Am J Physiol (Heart Circ Physiol)*, 286: H174-H180, (2004).

Witt, K.A., **Mark, K.S.**, Hom, S., and Davis, T.P., Effects Of Hypoxia-Reoxygenation On Rat Blood-Brain Barrier Permeability And Tight Junctional Protein Expression, *Am J Physiol (Heart Circ Physiol)*, 285: H2820 - 2831 (2003).

Brown, R.C., **Mark, K.S.**, Egleton, R.D., Huber, J.D., Burroughs, A.R., and Davis, T.P., Protection Against Hypoxia-induced Increase in Blood-Brain Barrier Permeability: Role of Tight Junction Proteins and NFκB, *J Cell Science*, 16 (pt 4): 693-700 (2003).

Mark, K.S. and Davis, T.P., Cerebral Microvascular Changes In Permeability And Tight Junctions Induced By Hypoxia And Reoxygenation, *Am J Physiol (Heart Circ Physiol)*, 282 (4): H1485-1494 (2002).

Mark, K.S., Trickler, W.J., and Miller, D.W., Tumor Necrosis Factor-α Induces Cycyclooxygenase-2 Expression and Prostaglandin Release in Brain Microvessel Endothelial Cells, *JPET*, 297 (3): 1051-1058 (2001).

Huber, J.D., Witt, K.A., Hom, S., Egleton, R.D., **Mark, K.S.**, and T.P. Davis, Inflammatory Pain Alters Blood-brain Barrier Permeability and Tight Junctional Protein Expression, *Am J Physiol (Heart Circ Physiol)*, 280 (3): H1241-H1248 (2001).

Mark, K.S. and Miller, D.W., Increased Permeability of Primary Cultured Brain Microvessel Endothelial Cell Monolayers Following TNF-α Exposure, *Life Sci*, 64 (21): 1941-1953 (1999).

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