National Jewish Health
Breathing Science is Life.

David W H Riches, PhD

Conditions Treated:

Research Areas:

- *Rheumatoid Arthritis (RA)*
- *Pulmonary Fibrosis*

Programs & Services:

- *Cell Biology*

Dr. Riches' career interests are focused on the intersection between lung immunity and fibrosis, and the role of macrophages and fibroblastic cells in these processes. Based on a combination of basic and translational approaches, his lab has made significant contributions to our understanding of the mechanisms underlying the development of pulmonary fibrosis, the mechanisms of macrophage functional programming, cytokine-induced signal transduction and the control of fibroblast and myofibroblast apoptosis. Dr. Riches' lab utilizes cutting edge approaches to model and analyze the development and resolution of pulmonary fibrosis in mice together with the study of comparable phenomena in lung tissues and primary cultured cells from patients with idiopathic pulmonary fibrosis and other fibrotic lung conditions.

Research Interests

Research into lung inflammation and fibrosis. 1. TNF-receptor family members play a key role in inflammation, innate and adaptive immunity and apoptosis. The goals of our lab are two fold. First, we are addressing fundamental questions about how the prototypic receptor, TNF-R1, initiates different responses. Second, we are investigating how TNF-R1 and other family members regulate apoptosis in pulmonary myofibroblasts. These latter studies are aimed at furthering our understanding of the mechanisms that lead to the development of pulmonary fibrosis. 2. The subcellular localization of the TNF receptor, TNF-R1, determines signaling responses. 3. TRUSS, a TNF receptor scaffolding protein. 4. Mechanism of myofibroblast apoptosis and survival in pulmonary fibrosis.

Education

1979 University of Birmingham, U.K., PhD

Teaching or Professional Positions

2015-Present: Director, Thomas L. Petty Aspen Lung Conference
2014-Present: Permanent Member, Lung Injury Repair and Remodeling study section
2009-Present: Member Editorial Board, American Journal of Respiratory Cell and Molecular Biology
2005: Chair, Innate Immunity and Inflammation study section
2001-2005: Permanent Member, Innate Immunity and Inflammation study section

Affiliations with the University of Colorado Denver

Professor, Department of Immunology & Microbiology, University of Colorado Denver

Professor, Division of Pulmonary Sciences and Critical Care Medicine, Department of Medicine, University of Colorado Denver

Professional Memberships

American Thoracic Society
American Association of Immunologists

Awards & Recognition

2011: Awarded the Recognition Award for Scientific Accomplishments by the American Thoracic Society.

Publications

M.W. Wynes, B.L. Edelman, A.G. Kostyk, M.G. Edwards, C. Coldren, S.D. Groshong, G.P. Cosgrove, E.F. Redente, A. Bamberg, K.K. Brown, N. Reisdorph, R.C. Keith, S.K. Frankel and **D.W.H. Riches**. 2011. Increased Cell Surface Fas Expression is Necessary and Sufficient to Sensitize Lung Fibroblasts to Fas Ligation-Induced Apoptosis. Implications for Fibroblast Accumulation in Idiopathic Pulmonary Fibrosis. *J. Immunol*. 187:527-537 PMID: 21632719

E.F. Redente, K.M. Jacobsen, J. Solomon, A. Lara, S. Faubel, P.M. Henson, G.P. Downey and **D.W.H. Riches**. 2011. Age and gender dimorphisms contribute to the severity of bleomycin-induced lung injury and fibrosis. *Am. J. Physiol*. (Lung Cell. Molec. Physiol.). 301:L510-L518. PMID: 21743030

R.C. Keith, J.L. Powers, E.F. Redente, A. Sergew, R.J. Martin, A. Gizinski, V.M. Holers, S. Sakaguchi and **D.W.H. Riches**. 2012. A novel model of rheumatoid arthritis-associated interstitial lung disease in SKG mice. *Exp. Lung Res*. 38:55-66 PMID: 22185348

J.M. Gump, L. Staskiewicz, M.J. Morgan, A. Bamberg, **D.W.H. Riches** and A. Thorburn. 2014. Autophagy variation within a cell population determines cell fate via selective degradation of Fap-1. *Nat. Cell Biol*. 16:47-54. PMID: 24316673

E.F. Redente, R.C. Keith, W. Janssen, P.M. Henson, G.P. Downey, L.A. Ortiz, D.L. Bratton and **D.W.H. Riches**. 2014. TNF-α accelerates the resolution of established pulmonary fibrosis in mice by targeting pro-fibrotic lung macrophages. *Am. J. Respir. Cell. Mol. Biol*. 50:825-837. PMID: 24325577

Doctor's Contact Information

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Locations

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