Leopoldina
Nationale Akademie
der Wissenschaften

Curriculum Vitae Prof. Dr. Andreas J. Bäumler

Name: **Andreas J. Bäumler**
Born: **22 November 1962**

Research Priorities: Enteric pathogens, the host and its Microbiota

Andreas J. Bäumler is a microbiologist and immunologist. He pioneered studies on the physiology of enteric pathogens in the natural context of a host-associated microbial community. This work identified epithelial energy metabolism as host control mechanism critical for maintaining homeostasis. Gut dysbiosis resulting from disruption of this host control mechanism underlies a broad range of non-communicable diseases.

Academic and Professional Career

since 2005	Professor of Medical Microbiology and Immunology, University of California, Davis, USA
2001 - 2005	Associate Professor of Medical Microbiology and Immunology, Texas A&M University Health Science Center, College Station, USA
1996 - 2001	Assistant Professor of Medical Microbiology and Immunology, Texas A&M University Health Science Center, College Station, USA
1995 - 1996	Research Assistant Professor of Molecular Microbiology and Immunology, Oregon Health & Science University, Portland, USA
1992 - 1995	Postdoctoral fellow, Department of Molecular Microbiology and Immunology, Oregon Health & Science University, Portland, USA
1989 - 1992	Doctor of Natural Sciences, Department of Microbiology II, Eberhard Karls University of Tübingen, Germany

Functions in Scientific Societies and Committees

since 2017 Editor in Chief of Infection and Immunity

since 2007 Vice Chair of Research, Department of Medical Microbiology and Immunology, University of California, Davis, USA

Honours and awarded Memberships

2021 Robert Koch Award, Robert-Koch-Stiftung, Berlin, Germany

since 2020 Member, German National Academy of Sciences Leopoldina, Germany

since 2010 Fellow, American Academy of Microbiology, USA

Research Priorities

Andreas J. Bäumler is a microbiologist and immunologist. He pioneered studies on the physiology of enteric pathogens in the natural context of a host-associated microbial community. This work identified epithelial energy metabolism as host control mechanism critical for maintaining homeostasis. Gut dysbiosis resulting from disruption of this host control mechanism underlies a broad range of non-communicable diseases.

An imbalance in the colonic microbiota might underlie many human diseases, but the mechanisms maintaining homeostasis remain elusive. Bäumler's work has established the concept that the metabolism of the colonic epithelium functions as a control switch, mediating a shift between homeostatic and dysbiotic communities. During homeostasis, colonic epithelial metabolism is directed towards oxidative phosphorylation, resulting in high epithelial oxygen consumption. The consequent epithelial hypoxia helps maintain a microbial community dominated by obligate anaerobic bacteria, which provide benefit by converting fiber into fermentation products absorbed by the host.

Bäumler's work shows that conditions that alter the metabolism of the colonic epithelium increase epithelial oxygenation, thereby driving an expansion of facultative anaerobic Enterobacteriaceae, a hallmark of dysbiosis in the colon. This dysbiotic shift in the gut microbiota is observed during infection with enteric pathogens, such as Salmonella or Citrobacter, which subvert epithelial metabolism to escape niche protection conferred by the gut microbiota. However, this mechanism also underlies a dysbiotic expansion of Enterobacteriaceae, such as Escherichia coli, in models of inflammatory bowel disease, colorectal cancer or after antibiotic treatment.

Notably, Bäumler's work shows that an increased availability of respiratory electron acceptors is a shared driver of gut dysbiosis in each of these settings. By providing a mechanistic explanation for gut homeostasis, his brand of reductionist microbiota research provides a novel lynchpin for strategies to remediate dysbiosis in a broad spectrum of human diseases by harnessing host

control mechanisms to restore epithelial hypoxia for therapeutic means.