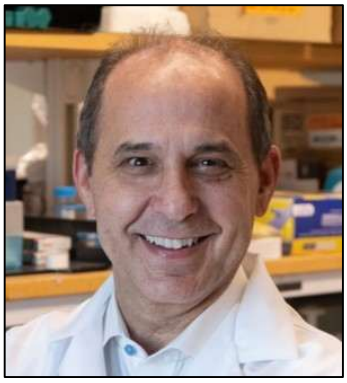
SHERMAN PRIZE
Inspiring Excellence in Crohn's and Colitis



Scott B. Snapper, MD, PhD

Chief, Division of Gastroenterology, Hepatology, and Nutrition, Boston Children's Hospital (BCH);
Wolpow Family Chair and Director, Center for Inflammatory Bowel Disease, BCH;
Physician, Crohn's and Colitis Center, Division of Gastroenterology,
Brigham and Women's Hospital;
Egan Family Foundation Professor of Pediatrics in the Field of Transitional Medicine
and Professor of Medicine, Harvard Medical School, MA

Dr. Snapper received his B.S., summa cum laude, from Tufts University and completed his M.D. and Ph.D. (Microbiology and Immunology) from Albert Einstein College of Medicine. He completed his Residency in Internal Medicine at Brigham & Women's Hospital (BWH) and then Fellowship training in Gastroenterology at Massachusetts General Hospital (MGH) – including post-doctoral work at Boston Children's Hospital (BCH). Dr. Snapper spent 18 years at MGH as a physician-scientist rising up the ranks to Associate Chief of Research. In 2011, Dr. Snapper became the Wolpow Family Chair and Director of the Center for Inflammatory Bowel Disease at BCH and joined the clinical and research faculty at BWH. In January 2020, Dr. Snapper became the Chief of the Division of Gastroenterology, Hepatology and Nutrition at BCH. Dr. Snapper is the Egan Family Foundation Professor of Pediatrics in the Field of Transitional Medicine & Professor of Medicine, Harvard Medical School (HMS).

For over 25 years, the Snapper laboratory has investigated how defective immunity can contribute simultaneously to both immunodeficiency and intestinal inflammation in infants. His group generated the first animal model of a human immune deficiency that presents with inflammatory bowel disease (IBD) and demonstrated that gene therapy was effective for preventing colitis. Working with leading investigators in Germany, Dr. Snapper's group contributed to the identification of IL10 receptor deficiency as causal for IBD, which could be ameliorated by stem cell transplantation. Employing both studies in mice and humans, his group determined that IL10R deficiency results in innate immune cell (macrophage) activation with enhanced production of the pro-inflammatory cytokine IL1β. These findings have had broad implications for therapeutic approaches for the autoimmunity and colitis associated with IL10R deficiency (and other immune deficiencies) as blocking IL1β activity can ameliorate colitis development. Recently, they have identified similar IL1-enriched signals in IBD more generally and have discovered a novel IL1-dependent pathway emanating from endothelial cells that may regulate fibrosis in IBD with targeted therapeutic approaches in development.

A longstanding goal of Dr. Snapper's work has been to learn deeply from rare immune deficiencies that are associated with IBD to decipher underlying mechanisms for more common forms of IBD. They posited that with new advanced molecular tools and the availability of increased numbers of patients and bio-samples, they could begin a coordinated effort to understand more broadly the genetic, immune, microbial and environmental components that lead to infantile and very-early onset IBD and, most importantly, drive the development of novel therapeutics. To facilitate this endeavor, Dr. Snapper with Dr. Aleixo Muise (Toronto) and Dr. Christoph Klein (Munich) established the VEOIBD Consortium (www.veoibd.org). This consortium has collected the largest number of VEOIBD patients in the world. Importantly, they have identified > 10 monogenic gene mutations that are likely causal for VEOIBD and three novel therapeutic approaches. Overall, this work has led to clear changes in clinical practice – with several altered genes resulting in dramatic immune consequences that are corrected by HSC transplantation. Current work seeks to use these rare monogenic causes of IBD as 'molecular guideposts' to inform the basis of the more common causes of IBD and to develop precision therapeutics.

Another interest of the Snapper laboratory has been the development of therapeutics that lead to the expansion of regulatory T cells for the treatment of IBD. They hypothesized that expansion of this suppressive T cell population may control the break in tolerance associated with IBD. They completed a phase 1B clinical trial demonstrating the safety and immune effect of low-dose IL-2 in the expansion of regulatory T cells for the treatment of moderate to severe UC. Based on this successful work, they have initiated a trial evaluating the safety and immune effect of low-dose IL2 in Crohn's disease. IL2 and related therapeutics targeting regulatory T cells are currently under intense investigation by the pharmaceutical industry.

Dr. Snapper has authored over 250 original articles and has received grants from the NIH, the Helmsley Charitable Trust, the Crohn's and Colitis Foundation, the Chan-Zuckerberg Initiative, and from major pharmaceutical companies. Dr. Snapper participates in numerous scientific advisory boards of major pharmaceutical and biotechnology companies, served as a permanent NIH Digestive System Host Defense, Microbial Interactions and Immune and Inflammatory Diseases (DHMI) Study Section member, is a past chair of the National Scientific Advisory Committee of the Crohn's & Colitis Foundation, and is currently the Co-Chair of the Scientific Advisory Board of the Rainin Foundation. Dr. Snapper is a member of the American Society of Clinical Investigation and has won several awards including the Humanitarian of the Year of the New England Chapter of the Crohn's and Colitis Foundation.

Another major focus of Dr. Snapper's efforts has been the training of the next generation of physician-scientists. He is the principal investigator of an NIH T32 Training grant funded for 40 years and has been the mentor of more than 30 graduate or post-doctoral fellows at Harvard. Many of these fellows have become independent investigators at prominent institutions worldwide.

Dr. Snapper received the Sherman Prize for Excellence in Crohn's and colitis in 2025.