



WHAT A **CURE** MEANS TO **ME**

“It means
that my work will help people
live happier lives—and that’s the reason why I chose a career
in research.”

RESEARCH REPORT

THE JOURNEY TOWARD A CURE: MANY ROUTES, ONE DESTINATION

Scientific knowledge is never static. Especially when it comes to the fight against chronic diseases, the work of science is very much a work in progress. Each new generation of scientists builds on the accumulated discoveries of the past—adding to them, refuting them, or transcending them through radically new approaches and insights that change our lives for the better—sometimes dramatically.

In the past two decades, the study of medicine has been transformed by successive waves of discovery in genetics and genomics. These discoveries have added layers of complexity to virtually every field of biomedical inquiry, from microbiology to immunology and from pathophysiology to clinical research. Further, traditional disciplines are no longer separated by firm boundaries. It is a given that today's scientists collaborate with their colleagues across a variety of specializations. Cross-disciplinary research means the cross-fertilization of ideas, perspectives, methods, and “lessons learned.” It also means that the pace of discovery is accelerating.

That acceleration is what “translational” research is all about: the speed with which the results of basic science research translate into new treatments that can be tested, retested, and finally, made available to people suffering from Crohn's disease or ulcerative colitis. Speed, breadth, and complexity are the identifying qualities of today's science.

A cure for IBD may not be imminent—but in 2005, research conducted under CCFA's auspices strengthened the links between diverse disciplines, pieced together new parts of the IBD puzzle, and generated important new questions that will drive IBD research over the next five years.

THE BEST SCIENCE: TWO MAJOR STRATEGIES

In 1967, the founding members of CCFA set out to seed and cultivate a new field of research. Little was known about Crohn's disease and ulcerative colitis at the time, but the founders knew that a commitment to the best science, and to attracting the most talented researchers on offer, would result in a bountiful harvest: better diagnostics, better treatments, a deeper understanding of what makes IBD tick, and greater awareness of its impact on patients, families, and society at large.

Thanks to the founders' foresight, and to CCFA's research mission over nearly four decades, we're homing in on the genes, proteins, cells, and microbes that drive and perpetuate Crohn's disease and ulcerative colitis.

For years, CCFA has dedicated itself to attracting the best and the brightest to the field of IBD research. Today, the Foundation can state unequivocally that it has achieved this important goal. Some of the world's most gifted scientists have gravitated to the challenge of solving the IBD puzzle. CCFA continues to fund research of the highest quality via two avenues of support: its traditional grants program and *Challenges in IBD Research*—CCFA's strategic plan for research.

All applications—whether submitted independently or in response to a CCFA Request for Applications (RFA) in a particular *Challenge* area—go through a rigorous peer-review process, in which the Foundation’s National Scientific Advisory Committee selects only those that meet the strictest criteria with respect to study design and scientific worthiness.

What follows is an overview of what has been accomplished by CCFA-funded researchers in 2005, how various studies interrelate and mutually amplify each other, and how, taken together, their efforts are converging toward an IBD-free future.

CONNECTIONS VISIBLE

Genetics

NOD2, the first susceptibility gene for Crohn’s disease, was discovered in 2001 (in part through CCFA-funded research). Since then, scientists have been working overtime to understand its structure and function.

When NOD2 is normal, it produces a protein—also called NOD2—that helps the immune system differentiate dangerous bacteria from those that are harmless, or even beneficial. The NOD2 protein also senses bacteria in the gut and protects it from harmful pathogens such as *listeria* and *salmonella*.

The mutated, or abnormal, NOD2 gene, however, loses some of its native ability to distinguish between harmful and harmless bacteria.

In 2005, CCFA-funded researchers in genetics forged new insights into NOD2 and the genetic underpinnings of IBD. For example, **Li-Chung Hsu, Ph.D.**, found that mice with the mutated gene overproduced IL-1b, a protein that promotes inflammation, and went on to develop severe intestinal inflammation. As a result, IL-1b may emerge as an important molecular target in the drug development process.

NOD2-Bacterial Interactions

As part of its normal bacteria-sensing activity, the NOD2 protein recognizes and binds to a component of the bacterial cell wall called muramyl dipeptide (MDP). This binding interaction prevents inflammation. Conversely,

defects in the NOD2 protein make it insensitive to MDP and the bacterium slips through the “barbed wire” of the intestinal immune system. The failure of NOD2 and MDP to interact fuels inflammation.

This year, researchers probed this and other NOD2-bacterial interactions that keep the gut healthy along with the cellular components that help or hinder the process along the way. For example, CCFA grant recipient **Maria Rescigno, Ph.D.**, discovered a new mechanism by which two cell types—dendritic and epithelial cells—preserve a balanced, anti-inflammatory environment in the intestines of healthy people. Dr. Rescigno and her colleagues will continue to explore the connection between NOD2 mutations and their effects on cellular behavior. Improved understanding of these molecular interactions will help scientists create treatments to short-circuit the inflammatory response before it starts.

Microbiology

In 2005, CCFA supported research that explored the tendency of the IBD patient’s immune system to lose tolerance for normal bacteria. Further, microbiologists scrutinized the role of friendly bacteria, known as probiotics, in maintaining microbial balance in the intestine.

One of the most striking studies carried out in 2005 was spearheaded by **Stephan Targan, M.D.**, a distinguished researcher working at the interface of genetics, microbiology, and immunology. Dr. Targan first established that a bacterial protein called CBir1 triggers a major response from the mouse’s immune system. Approximately 50% of Crohn’s patients have antibodies to CBir1. These patients’ strong immune response to the protein is based on genetic variations, Dr. Targan hypothesizes. Therefore, he will continue to explore how genetic variations are related to the magnitude of the anti-CBir1 response, thus shedding light on one major subtype of Crohn’s disease.

Dendritic Cells and Toll-Like Receptors

It bears repeating that IBD occurs in people whose immune systems react with abnormal intensity to harmless bacteria in the intestines. Scientists believe that this abnormal trait is genetically controlled, and that correcting it would be a powerful strategy for treatment.

However, before such a treatment can be developed, researchers must learn which types of immune cells actually detect the presence of bacteria.

CCFA-sponsored research has found dendritic cells to be a major culprit in the abnormal activation of the immune response in IBD. These specialized immune cells send out balloon-like protrusions into the intestinal cavity, where they “see” bacteria and other microbes via molecules called toll-like receptors (TLRs). The dendritic cells then proceed to pick up samples of bacteria and take them back to the immune system.

Pursuing research along these lines, **Osamu Hitotsumatsu, M.D.**, found a protein produced by dendritic cells, called A20, that blocks the production of the entire gamut of inflammatory chemicals in the body. This suggests that for some people with IBD, drugs that increase the level or activity of A20 may be a powerful way to dampen the excessive inflammation that afflicts people with Crohn’s or colitis.

Immunoregulation

The immune system is a vast complex of organs, tissues, cells, molecules, and signaling pathways—so vast that it used to seem unknowable. However, researchers today have learned a great deal about how the immune system protects the body from disease and injury. The task ahead is to identify the molecular “players” that regulate the immune response and to harness their natural power toward better treatments for IBD.

Among other research projects in the field of immunoregulation, CCFA funded several studies of regulatory T cells, or T-regs. T-regs are specialized T cells that calm inflammation. In the future, treatments that foster the activity of these important immune cells could prevent damage to the intestinal lining and bring enormous relief to people with IBD.

Researcher **Robin Hatton, Ph.D.** has been looking at how T-regs produce IL-10, a type of protein or cytokine that reduces inflammation via its associated gene. After studying distant regions of DNA called “non-coding sequences,” she has identified several potential regulatory elements for IL-10—yet another example of the

interconnectedness of genes, proteins, and the immune response. Dr. Hatton, among other researchers, contends that IL-10 could become a promising therapeutic target in the arsenal of emerging treatments for IBD.

Mucosal Healing

The intestinal lining serves as a barrier that protects the body from harmful substances in the intestinal cavity itself. Once that barrier is breached by a bacterium or other invading substance, the body responds by initiating wound healing to repair the injury. In IBD, the wound-healing response is defective.

Normal wound healing involves the modification of histone 3, one of a group of proteins that form an important part of chromosomes. In 2005, **Thomas Karrasch, M.D.**, pursued research based on the idea that wound healing depends on the activation of certain genes, followed by a series of particular molecular “events.” In the process, histones are modified and chromosomal structures undergo remodeling. Finally, healing substances are produced leading to the restoration of the mucous



Children and teens with IBD face a unique set of challenges, which our new strategic plan for pediatric research will address.

membrane that lines the gut. Dr. Karrasch's hypothesis was borne out when he studied mucosal wound healing in mice and saw the process in action.

By understanding how mucosal healing takes place under healthy circumstances, researchers aim to learn how to promote healing in the damaged intestinal tissue often found in people with IBD.

CHALLENGES PRESENT AND FUTURE

IBD researchers aim to unveil the complex processes that characterize health and apply the resulting knowledge to prevent, manage, and cure Crohn's disease and ulcerative colitis. Although we don't yet have a cure for these diseases, the pieces of the IBD puzzle are coming together. CCFA is proud to be at the forefront of the puzzle-solving process.

In addition to funding diverse research projects on an ongoing basis, the Foundation also helps shape the larger IBD research agenda through its *Challenges in IBD Research* program. With the help of its scientific advisors, CCFA launched the *Challenges* in 1990. In 2003, the strategic plan was revised and a new set of priorities established based on new insights, new data, and far-reaching changes in the IBD research paradigm.

Microbial Antigens

CCFA approved nine applications in response to the first *Challenge*: Microbial Antigens. To gain a deeper understanding of the role played by microbes and their various components in triggering IBD, the selected researchers set out to:

- identify specific microbes (mainly bacteria) that drive chronic intestinal inflammation;
- identify bacterial components that activate the dysfunctional immune responses behind that inflammation; and
- investigate the role of probiotics in the treatment of IBD.

Eugene Chang, M.D., a seasoned IBD researcher, focused on *Lactobacillus GG* (LGG)—a probiotic agent known to produce small chemicals with potent anti-inflammatory actions in the bowel. He and his colleagues succeeded in

defining the cellular pathways involved in the probiotic's effect on intestinal epithelial cells. They found that LGG activates several heat shock proteins that protect intestinal tissue against injury.

Dr. Chang's studies have important therapeutic implications. Future studies will focus on identifying the active compounds produced by probiotics. These compounds could then be modified to create treatments that are effective, safe, and easily given to patients with IBD to reduce inflammation, heal wounds, and protect their intestines from further injury.

Biomarkers

One of the most dreaded complications of IBD is increased risk for colon cancer, a risk that rises after people have had ulcerative colitis or Crohn's disease of the colon for more than 8 to 10 years. Unfortunately, present screening methods, principally colonoscopy, are time-consuming, expensive, uncomfortable, and not infallible.

An alternative to colonoscopy is desperately needed in order to identify the relatively small number of patients at risk and focus diagnostic, financial, and human resources on them. Doing so will also allow the majority of patients to avoid unnecessary screening.

For these reasons, CCFA launched its second *Challenge* initiative, "Biomarkers of Colon Cancer in Inflammatory Bowel Disease." Biomarkers are measurable substances in the blood, stool, or tissues that indicate the presence of a disease. Once a reliable biomarker for IBD-related colon cancer is found and its reliability confirmed, we'll have a quick, non-invasive test to detect those patients at greatest risk—approximately 10% of the total IBD population.

By the end of 2005, the Biomarkers initiative was already yielding impressive results. For example, **Teresa Brentnall, M.D.**, has developed a new genetic biomarker test to detect precancerous and cancerous changes in people with both ulcerative colitis and a liver disease called primary sclerosing cholangitis (PSC)—a particularly high-risk patient group. The test, performed with one rectal biopsy, has been shown to detect cancer and pre-cancer in patients with colitis and PSC 95% of the time.

FROM LAB BENCH TO BEDSIDE



Basic researchers study the complex interactions between genes, bacteria (such as *Listeria monocytogenes*, pictured here), and the immune response in IBD.



Clinical researchers focus on translating the findings of basic research into new treatments that promise to calm inflammation, heal damaged tissues, ease painful symptoms, and improve the quality of life for people with ulcerative colitis or Crohn's disease.



Gastroenterologists work with their patients to find the best possible treatment, or combination of treatments, for their particular disease sub-type.

For nearly four decades, CCFA has fostered research of the highest quality, seeded the careers of hundreds of basic and clinical researchers, and contributed to every major advance in the field. As a result, people with Crohn's disease or ulcerative colitis have more and better treatment options today than their counterparts did a short time ago. We don't yet hold a cure in our hands. But the knowledge gleaned from CCFA-led research is being translated into tools and treatments that will—in a rapidly approaching future—help us prevent and even cure IBD.



Richard Blumbeg, M.D.
Immediate Past Chair,
National Scientific
Advisory Committee

The next step will be to validate the test in these patients and, eventually, in people with IBD without PSC.

CHALLENGES IN PEDIATRIC IBD

In September 2005, CCFA sponsored the first national conference to focus exclusively on issues in pediatric IBD. The meeting, held in Boston, provided a forum for 30 leading physicians and scientists to discuss priorities and create a five-year strategic plan for pediatric IBD research.

It is obvious that children with chronic diseases aren't just "little adults." They face a unique set of issues, both physically and psychologically. By advancing our understanding of the factors involved in Crohn's disease and ulcerative colitis in childhood and adolescence, CCFA hopes not only to improve the lives of children, but also to find better ways to treat these illnesses and prevent them from developing in the first place.

This ambitious pediatric research initiative is modeled after our overarching *Challenges in IBD Research* program.

In 2006 and 2007, CCFA will issue RFAs and seek to attract the country's most talented researchers to the following five areas of pediatric research:

- ***The Effects of Inflammation on Growth and Skeletal Development***
Research on the effects of IBD on linear growth (height) and bone accretion (the net addition of bone over time) emerged as the area of highest priority. In 2006, CCFA will support research that will advance our understanding of the link between bone development and IBD in children.

- ***Genetics of Early Onset IBD***

We have much more to learn about the susceptibility genes associated with very early onset disease. Proposals will be sought to identify the susceptibility alleles, or variants of a particular gene, associated with early onset disease. CCFA also intends to support genotype-phenotype research—studies that explore the correlation between a person's genetic constitution and the particular characteristics of his or her disease—among a large group of pediatric patients.

- ***Development of a Quality Improvement Collaborative Network***

Preliminary data show considerable variation in pediatric IBD care due to underuse, overuse, and misuse of medical resources. This unevenness leads to lower quality of care, higher costs, and rising morbidity, or severity of symptoms. To reduce variation in care, improve health outcomes, and enhance the quality of life of children and teens with IBD, CCFA will support prospective studies in which participants are selected and followed forward in time in this area.

- ***Immunology of Early Onset IBD***

Does pediatric IBD differ substantially from adult disease? Recent studies support the hypothesis that IBD develops in distinct phases. Further research is urgently needed to evaluate the intestinal immune response in pediatric IBD, with a view toward developing new therapeutic approaches designed for children. To achieve these goals, it is essential to understand how IBD evolves from the early to the later stages of disease, and how these two phases differ.

- ***Psychosocial Functioning and Development***

While a few studies have begun to identify children with IBD who are at risk for emotional and behavioral problems, much more research is needed to help us understand both risks and potential remedies. Researchers will look at biological factors, such as the effects of cytokines (inflammatory proteins) on depression and environmental factors, such as poor social support.

In 2006, the proceedings of the Boston conference, including in-depth descriptions of the five pediatric Challenges, will be published in *Inflammatory Bowel Diseases*, CCFA's premier professional journal.

HIGHLIGHTS OF CLINICAL RESEARCH

With more than 80 experimental treatments in clinical trials, experts predict that a wave of new therapies for ulcerative colitis and Crohn's disease is on the way. In 2005, infliximab (Remicade®)—the first biologic therapy for Crohn's disease—was approved for ulcerative colitis. Another TNF blocker, adalimumab (Humira®), was shown to induce remission in patients with moderate-to-severe Crohn's disease in a study authored by **Drs. William Sandborn, Stephen Hanauer**, and others that was recently published in the peer-reviewed journal *Gastroenterology*. Targeting other molecular culprits in the inflammatory response, new biologic agents are in various stages of testing and should become available over the next several years.

Dr. Sandborn has also been at the forefront of recent initiatives to get more out of the treatments we already have. For example, he and his colleagues found that budesonide (Entocort® EC), a non-systemic steroid that causes fewer side effects than traditional corticosteroids, is an effective treatment for Crohn's disease that affects the ileum and ascending colon. Budesonide also was shown to be an effective therapy for microscopic colitis, a less severe illness than ulcerative colitis. A majority of patients with pouchitis (inflammation of the internal pouch created during IPAA, a surgical procedure for ulcerative colitis), also were shown to benefit from budesonide therapy.

Developing new treatments and maximizing the benefits of already-available drugs—that's the strategy CCFA has adopted to take clinical research to the next stage. As the pace of basic research continues to quicken, it will soon be matched by results from clinical studies that will broaden patients' treatment choices and ease their suffering today.



TOWARD AN IBD-FREE WORLD

2005 was a year chock-full of accomplishments in research. It was not a year of gigantic breakthroughs, but our understanding of the causes, development, and treatment of IBD deepened significantly. Better yet, CCFA has set in motion the energies and talents of a growing community of researchers dedicated to finding the microbes that drive Crohn's disease and ulcerative colitis...the biomarkers for IBD-related colon cancer...the genetic roots of pediatric IBD...and the treatments that will, one day, eradicate these diseases altogether.

2005 was a pivotal year for yet another reason: CCFA's National Scientific Advisory Committee (NSAC), led with distinction for three years by Richard Blumberg, M.D., passed the torch to a new group of advisors. Jonathan Braun, Ph.D., the newly-elected NSAC Chair, has already begun to make his mark, building on Dr. Blumberg's legacy of excellence as a scientist and as a leader among leaders in the field.

CCFA's gratitude to Dr. Blumberg, and to his colleagues on the NSAC, is immeasurable. Its faith in Dr. Braun, too, is great. We are honored to share the journey toward a cure with such leaders at the helm. And we are confident that they'll help us reach our destination sooner rather than later.

RESEARCH AWARDS

Each year, CCFA's National Scientific Advisory Committee (NSAC) meticulously conducts peer reviews of more than 200 grant applications. Grants are awarded on the basis of scientific merit and relevance to inflammatory bowel disease (IBD). We are pleased to recognize those donors who make significant gifts to research through a naming opportunity. Donors to the CCFA Research Program have the option of meeting the researcher in his or her laboratory and receiving a progress report after one year, as well as a final report at the end of the project. Donors will also have their names listed in our Annual Report and will receive priority invitations to selected CCFA events.

For more information, please call the CCFA National Development Department at 800-932-2423, or contact your local CCFA chapter. Investigators interested in applying for a research grant are encouraged to call CCFA's Research and Scientific Programs Department at 800-932-2423, or visit the Foundation's Web site at www.ccfaprofessionals.org.

CHALLENGES IN IBD RESEARCH

Since 1967, CCFA has been working to identify the needs of the research community and develop action plans to meet those needs. *Challenges in IBD Research*, CCFA's strategic plan, sets forth research priorities to answer the most crucial questions first. In 2005, donors contributed funds in support of three leading-edge areas: Microbial Antigens, Dysplasia and Cancer, and Surrogate Markers.

RESEARCH INITIATIVES

CCFA's Research Initiatives focus on new or undeveloped areas of research that the NSAC deems hold the most promise for finding better treatments for, and ultimately preventing and curing, Crohn's disease and ulcerative colitis. These large-scale projects emerge directly out of the Foundation's overarching research strategies, which are summarized above in the section titled "Challenges in IBD Research."

The DNA and Cell Line Bank

An essential aspect of IBD research is the need to understand the influence of an individual's genotype, or genetic characteristics, on his or her phenotype, or observable traits of disease. CCFA spearheaded the effort to study genotypes and phenotypes by establishing the DNA and Cell Line Bank, a valuable resource that contains DNA and cell lines from patients nationwide.

Clinical Research Alliance

CCFA's Clinical Research Alliance is a national network of major medical centers and smaller, local facilities. The Alliance's institutional members collaborate on clinical studies targeting the management and treatment of IBD.

SENIOR GRANTS PROGRAM

Senior Research Awards

These grants are awarded to established investigators for projects conducted at hospitals, universities, and research laboratories around the world.

First Awards

Candidates for these awards must be independent of a mentor, yet at the beginning stages of their research career.

Note: CCFA is no longer accepting applications for this award category, which has been discontinued.

RESEARCH TRAINING AWARDS PROGRAM

CCFA offers the following awards to nurture careers in independent investigation of IBD. Candidates must be employed by institutions engaged in healthcare and health-related research within the USA and its possessions.

Career Development Awards

Candidates for these awards must have five to 10 years of post-doctoral experience, including two years of relevant research experience. The research project must be in the field of IBD.

Research Fellowship Awards

Candidates must have at least two years of postdoctoral experience. The research project must be in the field of IBD.

Student Research Fellowship Awards

Candidates are undergraduate, medical, or graduate students at accredited North American institutions. They conduct full-time research for a minimum of 10 weeks with a mentor investigating a subject relevant to IBD.

A full summary of all projects is available upon request.

CHALLENGES IN IBD RESEARCH INITIATIVES

MICROBIAL ANTIGENS

Scientists believe that IBD occurs when an environmental agent triggers the abnormal immune response in genetically susceptible individuals. By studying microbes and the body's response to them, researchers may be able to identify these agents and gain essential information about the development and cause of IBD.

The Microbial Antigens Research Initiative was made possible with funding from the following generous donors: *

Anonymous Donor, Illinois Carol Fisher Chapter
Anonymous Donor, Southwest Ohio Chapter
Anthony Balio
Claire Berman
Jacob and Hilda Blaustein Foundation
Judy and Michael Brostoff
Benjamin G. Darnell
Art and Marcy Falcone
The Sanford J. Grossman Charitable Trust¹
Jarret Industries²
Montgomery Community Foundation
Les and Melvin Leeb²
Marti Lemieux
Daniel L. Stone
The Wachovia Foundation, Inc.²

* Footnotes indicate funding was designated for a specific project

¹ Eugene B. Chang, M.D.

The University of Chicago, Chicago, IL
Cellular and molecular mechanisms of probiotic anti-inflammatory and cytoprotective actions

Lora V. Hooper, Ph.D.

The University of Texas, Dallas, TX
Interactions between commensal bacteria and the paneth cell: Role in shaping innate and adaptive immunity

Koichi Kobayashi, M.D., Ph.D.

Dana Farber Cancer Institute, Boston, MA
Roles of NOD proteins in intestinal mucosal immunity

Stephen J. McSorley, Ph.D.

University of Minnesota, Minneapolis, MN
In vivo tracking of antigen presentation and CD4 T cell responses to intestinal bacteria

² Ruslan Medzhitov, Ph.D.

Yale University School of Medicine, New Haven, CT
Role of toll-like receptors in the pathoetiology of inflammatory bowel disease

Maria Rescigno, Ph.D.

European Institute of Oncology, Milan, Italy
In vitro and in vivo models to study the role of mutations in the NOD2 gene, that are associated with susceptibility to Crohn's disease, in bacterial handling and generation of the inflammatory response

Lawrence J. Saubermann, M.D.

Rochester Medical Center, Rochester, NY
Determination of microbial peptides associated with regulatory and effector T cells in the CD4+CD45Rb high T Cell adoptive transfer colitis model

Gerald W. Tannock, Ph.D.

University of Otago, Dunedin, New Zealand
Detection and identification of bacterial substances that activate the adaptive immune response in pouchitis

Vincent B. Young, M.D., Ph.D.

Michigan State University, East Lansing, MI
Interactions between H. Hepaticus probiotics and other members of the intestinal microbiota in the development of colitis in IL-10-/- mice

DYSPLASIA AND CANCER

People who have had IBD for eight to 10 years are at a higher risk for colon cancer. By identifying markers (early signs in blood or tissues) for dysplasia (precancerous changes in a cell), it will be possible to determine which patients are at risk.

The Dysplasia and Cancer Research Initiative was made possible by the following generous donors:

Ethel Wilson Bowles and Robert Bowles Memorial Fund
Leon and Toby Cooperman
The Gillman Family: Shaldine, Richard, Sloane, Marc, Andrea and Scott
Mark and Diane Goldman
Kacyra Family Foundation³
F.M. Kirby Foundation
Mr. and Mrs. Fred H. Miller
Northeast Ohio Research Alliance⁴
Gary and Lanie Sinderbrand

³ Teresa A. Brentnall, M.D.

University of Washington Medical Center, Seattle, WA
Biomarkers of colonic dysplasia and cancer in UC patients with PSC

⁴ Mary P. Bronner, M.D.

The Cleveland Clinic Foundation, Cleveland, OH
Genomic instability biomarkers of cancer in Crohn's disease

Steven Itzkowitz, M.D.

Mount Sinai School of Medicine, New York, NY
Molecular biomarkers of colon cancer in IBD patients: Tissue-stool correlations

SURROGATE MARKERS

Usually found in blood samples, surrogate markers are tools that allow us to measure disease activity. These markers may be used to categorize the diseases into sub-types and to predict patterns of disease activity and how a person will respond to different forms of therapy.

The following have generously provided funding for the Surrogate Markers Research Challenge Initiative:

Bruce and Cynthia Sherman
Jed Manocherian

This Research Initiative will be launched in 2006.

CCFA RESEARCH INITIATIVES

DNA AND CELL LINE BANK

The following have generously provided funding for the DNA and Cell Line Bank:

Joseph Drown Foundation
Friends of the Greater Washington, D.C./Virginia Chapter
F.M. Kirby Foundation
Peter and Joan McKee
Modell Family Foundation
Donald W. Reynolds Foundation
Anne and Henry Zarrow Foundation
Maxine and Jack Zarrow Family Foundation

Robert S. Sandler, M.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
DNA and Cell Line Bank Data Management Center

Kristin Ardlie, Ph.D.

Genomics Cooperative Inc., Cambridge, MA
Purchase of matched control samples; plating, distribution and storage of samples for the DNA and Cell Line Bank

Jeanne C. Beck, Ph.D.
Coriell Institute, Camden, NJ
DNA and Cell Line Repository

Richard H. Duerr, M.D.
University of Pittsburgh School of Medicine, Pittsburgh, PA
DNA and Cell Line Bank-Patient Acquisition Center

Sonia Friedman, M.D.
Brigham and Women's Hospital, Boston, MA
In support of the DNA and Cell Line Bank/Sample Collections

Andrew Ippoliti, M.D.
Cedars-Sinai IBD Center, Los Angeles, CA
DNA and Cell Line Bank-Patient Acquisition Center

Kim L. Isaacs, M.D.
University of North Carolina at Chapel Hill, Chapel Hill, NC
DNA and Cell Line Bank-Patient Acquisition Center

Sunanda V. Kane, M.D.
The University of Chicago Medical Center, Chicago, IL
DNA and Cell Line Bank-Patient Acquisition Center

Lloyd F. Mayer, M.D.
Mount Sinai School of Medicine, New York, NY
DNA and Cell Line Bank-Patient Acquisition Center

Bruce E. Sands, M.D.
Massachusetts General Hospital, Boston, MA
DNA and Cell Line Bank-Patient Acquisition Center

CLINICAL RESEARCH ALLIANCE

William J. Sandborn, M.D.
Mayo Clinic, Rochester, MN
Clinical Research Alliance: Administrative Award

Robert S. Sandler, M.D.
University of North Carolina at Chapel Hill, Chapel Hill, NC
Clinical Research Alliance: Data Management Center

EPIDEMIOLOGY INITIATIVE

Lisa Herrinton, Ph.D.
Kaiser Foundation Research Institute, Oakland, CA
Population-based study of IBD incidence and prevalence by disease subtype and severity

Lisa Herrinton, Ph.D.
Kaiser Foundation Research Institute, Oakland, CA
Practice variation in management of inflammatory bowel disease

NATIONAL GNOTOBIOTIC RODENT RESOURCE CENTER

R. Balfour Sartor, M.D.
University of North Carolina at Chapel Hill, Chapel Hill, NC

SENIOR RESEARCH AWARDS

Clara Abraham, M.D.
The University of Chicago, Chicago, IL
LFA-1 and Regulatory T Cells

THE INFLAMMATORY BOWEL DISEASES EDITORIAL BOARD SENIOR RESEARCH AWARD

Steven R. Brant, M.D.
Johns Hopkins University School of Medicine, Baltimore, MD
The Mid-Atlantic African-American IBD study: Exploring racial disparities

Thomas Brunner, Ph.D.
University of Bern, Bern, Switzerland
The role of in situ produced, intestinal glucocorticoids in the regulation of intestinal inflammation

GREATER WASHINGTON, D.C./VIRGINIA CHAPTER RESEARCH ALLIANCE

James E. Casanova, Ph.D.
University of Virginia Health System, Charlottesville, VA
Epithelial cell responses to salmonella infection

John J. Cebra, Ph.D.
University of Pennsylvania School of Medicine, Philadelphia, PA
Interactions between commensal gut bacteria, the gut epithelium, and the mucosal immune system leading to inflammatory bowel disease

Eugene B. Chang, M.D.
The University of Chicago, Chicago, IL
Role of OCTN1 and OCTN2 in maintaining intestinal homeostasis and in the pathogenesis of IBD

Donald A. Cohen, Ph.D.
University of Kentucky Medical Center, Lexington, KY
Protective role of macrophages in the development of inflammatory bowel disease

WILLIAM AND SHELBY MODELL FAMILY FOUNDATION RESEARCH AWARD

Fong-Fong Chu, Ph.D.
Beckman Research Institute of City of Hope, Duarte, CA
Oxidative stress induced by enterohepatic helicobacter-associated ileocolitis and cancer in GPX-DKO mice

Fabio Cominelli, M.D., Ph.D.
University of Virginia Health System, Charlottesville, VA
Patient preferences and treatment decisions in ulcerative colitis

Anthony DeFranco, Ph.D.
University of California, San Francisco, San Francisco, CA
Regulation of NOD2 innate immune function

NORTHWEST CHAPTER RESEARCH ALLIANCE

Peter J. Dempsey, Ph.D.
Pacific Northwest Research Institute, Seattle, WA
ADAM-mediated ErbB signaling in mucosal injury and repair

FLORIDA CHAPTER RESEARCH ALLIANCE

Ronald DePinho, M.D.
Dana-Farber Cancer Institute, Boston, MA
Examining telomere dysfunction and pathogenesis of ulcerative colitis, inflammatory bowel disease, and increased risk of colorectal cancer

THE INFLAMMATORY BOWEL DISEASES EDITORIAL BOARD SENIOR RESEARCH AWARD

Bonny L. Dickinson, Ph.D.
The Research Institute for Children, New Orleans, LA
FcRn-mediated IgG transcytosis across mucosal surfaces is regulated by calmodulin

MICHAEL S. MODELL MEMORIAL RESEARCH FUND AWARD

Richard H. Duerr, M.D.
University of Pittsburgh, Pittsburgh, PA
Fine mapping an ulcerative colitis locus on chromosome 2q

Lars Eckmann, M.D.
University of California, San Diego, La Jolla, CA
Murine model of the 1007fs mutation of NOD2-associated with Crohn's disease

Peter B. Ernst, Ph.D.
University of Virginia Health Center, Charlottesville, VA
The role of adenosine receptors in controlling intestinal inflammation

WILLIAM AND SHELBY MODELL FAMILY FOUNDATION RESEARCH AWARD

Richard J. Grand, M.D.

The Children's Hospital of Boston, Boston, MA
Use of intranasally administered calcitonin in the treatment of osteopenia and osteoporosis in children, adolescents, and young adults with IBD: A pilot study

ELIZABETH CALHOUN RESEARCH AWARD

Norman R. Harris, Ph.D.

Louisiana State University Health Sciences Center, Shreveport, LA
Microvascular flow in inflammatory bowel disease

Peter D. Higgins, M.D., Ph.D.

The University of Michigan, Ann Arbor, MI
A novel, valid disease activity index for clinical research in ulcerative colitis

I-Chen Ho, M.D., Ph.D.

Brigham and Women's Hospital, Boston, MA
Ets-1 as a therapeutic target of Crohn's disease

Bruce H. Horwitz, M.D., Ph.D.

Brigham and Women's Hospital, Boston, MA
Role of ERK 1/2 in the control of helicobacter hepaticus-induced IL-12 p40 expression

Christian Jobin, Ph.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Role of bacteria in colitis-associated colon cancer

Charlotte S. Kaetzel, Ph.D.

University of Kentucky Medical Center, Lexington, KY
Regulation of pro- and anti-inflammatory gene expression in intestinal epithelial cells by colonic bacteria

Stephen J. Keely, Ph.D.

Royal College of Surgeons in Ireland, Dublin, Ireland
Epidermal growth factor in chronic regulation of intestinal epithelial chloride secretion

Stephen M. Krane, M.D.

Massachusetts General Hospital, Charlestown, MA
Roles of collagenolysis and collagenolytic matrix metalloproteinases in the experimental inflammatory bowel disease that accompanies mucosal injury in mice

HONL FAMILY RESEARCH AWARD

Subra Kugathasan, M.D.

Medical College of Wisconsin, Milwaukee, WI
Genotype/phenotype correlation in new onset pediatric IBD patients

James W. Lillard, Jr., Ph.D.

Morehouse School of Medicine, Atlanta, GA
Cellular and molecular mechanisms of I-TAC-mediated colitis: Induction and maintenance

GET YOUR GUTS IN GEAR RESEARCH AWARD

Pauline K. Lund, Ph.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Role of suppressor of cytokine signaling 3 in epithelial repair and tumorigenesis during injury and inflammation

Stephen J. Meltzer, M.D.

University of Maryland School of Medicine, Baltimore, MD
Progression biomarker development in inflammatory bowel disease

Andrew S. Neish, M.D.

Emory University, Atlanta, GA
TLR signaling in intestinal inflammatory injury

GEORGIA CHAPTER RESEARCH ALLIANCE

Asma Nusrat, M.D.

Emory University, Atlanta, GA
Role of occludin in regulation of epithelial tight junctions

PHILADELPHIA/DELAWARE VALLEY CHAPTER RESEARCH ALLIANCE

Young Chul Park, Ph.D.

Fox Chase Cancer Center, Philadelphia, PA
Structural and functional study of NOD2 and its interaction

Theresa T. Pizarro, Ph.D.

University of Virginia Health Center, Charlottesville, VA
Development of novel diagnostic tools for the non-invasive imaging and evaluation of intestinal inflammation

WESTERN PENNSYLVANIA/WEST VIRGINIA CHAPTER RESEARCH ALLIANCE

Scott E. Plevy, M.D.

University of Pittsburgh, Pittsburgh, PA
Carbon monoxide, cigarette smoking, and IBD

D. Brent Polk, M.D.

Vanderbilt University School of Medicine, Nashville, TN
Mechanism of KSR regulation in intestinal cell survival

Charalabos Pothoulakis, M.D.

Beth Israel Deaconess Medical Center, Boston, MA
Toll-like receptors and intestinal inflammation

Hans-Christian Reinecker, M.D.

Massachusetts General Hospital, Boston, MA
Dendritic cells in intestinal inflammation

John D. Rioux, Ph.D.

Montreal Heart Institute, Montreal, Canada
A biological pathway approach to understanding IBD

William J. Sandborn, M.D.

Mayo Clinic and Foundation, Rochester, MN
A randomized, double-blind placebo-controlled trial of ciprofloxacin of metronidazole for the treatment of Crohn's disease perianal fistulas

MICHAEL LIBRETTI MEMORIAL RESEARCH FUND AWARD

Cynthia Sears, M.D.

The Johns Hopkins University School of Medicine, Baltimore, MD
Induction of colonic inflammation and hyperplasia by enterotoxigenic bacteriodes fragilis

MICHAEL S. MODELL MEMORIAL RESEARCH FUND AWARD

Robert J. Shulman, M.D.

Baylor College of Medicine, Houston, TX
Intestinal permeability in inflammatory bowel disease

Shanthi V. Sitaraman, M.D., Ph.D.

Emory University, Atlanta, GA
Role of metalloproteinase-9 in inflammatory bowel disease

ALABAMA/NORTHWEST FLORIDA CHAPTER RESEARCH ALLIANCE

Lesley E. Smythies, Ph.D.

University of Alabama at Birmingham, Birmingham, AL
Cellular and molecular biology of intestinal macrophages in normal mucosa and IBD

Thaddeus S. Stappenbeck, M.D., Ph.D.

Washington University School of Medicine, St. Louis, MO
Cellular and molecular factors of the epithelial stem cell niche of the colonic mucosal injury response

WILLIAM AND SHELBY MODELL FAMILY FOUNDATION RESEARCH AWARD

Daniel S. Straus, Ph.D.

University of California, Riverside, Riverside, CA
Peroxisome proliferator-activated receptors: Roles in normal colonic physiology and novel targets for treatment of inflammatory bowel disease

William F. Stenson, M.D.

Washington University School of Medicine, St. Louis, MO
Increased expression of indoleamine 2-3, dioxygenase diminishes the severity of TNBS colitis

Manjunath N. Swamy, M.D.

CBR Institute for Biomedical Research, Boston, MA
Role of CD27-CD70 costimulatory pathway in inflammatory bowel disease

Stephen R. Targan, M.D.

Cedars-Sinai IBD Center, Los Angeles, CA
Antibodies to the Flagellin, CBir1, define a subtype of Crohn's disease

Cornelis P. Terhorst, Ph.D.

Beth Israel Deaconess Medical Center, Boston, MA
Study of CD84 induced innate and adaptive immune responses in experimental colitis

THE SANFORD J. GROSSMAN CHARITABLE TRUST RESEARCH AWARD

Cornelis P. Terhorst, Ph.D.

Beth Israel Deaconess Medical Center, Boston, MA
The role of GITR-ligand/GITR interactions in control of regulatory T cell functions in experimental colitis

BRANDON GILLMAN RESEARCH AWARD

Jerrold R. Turner, M.D., Ph.D.

The University of Chicago, Chicago, IL
Barrier dysfunction in Crohn's disease: Myosin light chain phosphorylation as mediator and therapeutic target

MICHAEL S. MODELL MEMORIAL RESEARCH FUND AWARD

Jay Unkeless, Ph.D.

Mount Sinai School of Medicine, New York, NY
Modulation of MAST205 to treat experimental colitis

HOUSTON GULF COAST/SOUTH TEXAS CHAPTER RESEARCH ALLIANCE

James Versalovic, M.D., Ph.D.

Baylor College of Medicine, Houston, TX
Identification of probiotic genes in lactobacillus reuteri

DR. CARL LYSS RESEARCH AWARD, SPONSORED BY A FRIEND OF THE GATEWAY CHAPTER, ST. LOUIS, MO

Joel V. Weinstock, M.D.

Tufts-New England Medical Center Hospitals, Inc., Boston, MA
Substance P and the regulation of intestinal inflammation

Mark T. Worthington, M.D.

University of Virginia Health System, Charlottesville, VA
Post-transcriptional regulation of tumor necrosis factor gene expression in tolerized macrophages by 3' untranslated region and poly A tail regulatory proteins

FIRST AWARDS

WESTERN PENNSYLVANIA/WEST VIRGINIA CHAPTER RESEARCH ALLIANCE

Nathan Bahary, Ph.D.

University of Pittsburgh School of Medicine, Pittsburgh, PA
Developmental expression and function of IBD genetic determinants in the zebrafish

SOUTHWEST OHIO CHAPTER RESEARCH ALLIANCE

Lee A. Denson, M.D.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH
Molecular mechanisms of growth hormone resistance in chronic colitis

WISCONSIN CHAPTER RESEARCH ALLIANCE

Michael B. Dwinell, Ph.D.

Medical College of Wisconsin, Milwaukee, WI
Chemokine receptor regulation of the epithelial barrier

MINNESOTA/DAKOTAS CHAPTER RESEARCH ALLIANCE

Laurence J. Egan, M.D.

Mayo Clinic and Foundation, Rochester, MN
Regulation of intestinal epithelial cell migration by NF-kappa B

GEORGIA CHAPTER RESEARCH ALLIANCE

Andrew Gewirtz, Ph.D.

Emory University, Atlanta, GA
Regulation of intestinal epithelial chemokine secretion

Andrew C. Keates, Ph.D.

Beth Israel Deaconess Medical Center, Boston, MA
Regulation of MIP-3 alpha gene expression in IBD

Uma Mahadevan, M.D.

University of California, San Francisco, San Francisco, CA
Pregnancy outcomes in women with inflammatory bowel disease

Rodney D. Newberry, M.D.

Washington University School of Medicine, St. Louis, MO
Lymphotoxin beta receptor function and blockade in intestinal inflammation

INDIANA CHAPTER RESEARCH ALLIANCE

Mythily Srinivasan, Ph.D.

Indiana University School of Medicine, Indianapolis, IN
Structural and functional characterization of B7 competitive antagonist peptides as immunomodulatory agents in inflammatory bowel disease

Ramnik J. Xavier, M.D.

Massachusetts General Hospital, Boston, MA
The elucidation of the function of dendritic cells in inflammatory bowel disease

CULLMAN FAMILY RESEARCH AWARD

Huabao Xiong, M.D., Ph.D.

Mount Sinai School of Medicine, New York, NY
Inhibition of interleukin-12 (IL-12) gene expression by nitric oxide and inositol hexakisphosphate (IP6): Possible role in the treatment of inflammatory bowel diseases

CAREER DEVELOPMENT AWARDS

ILLINOIS CAROL FISHER CHAPTER RESEARCH ALLIANCE

Jeffrey B. Brown, M.D.

Children's Memorial Hospital, Chicago, IL
The role of P-selectin in effector T-cell recruitment to the colon

NORTHWEST CHAPTER RESEARCH ALLIANCE

Daniel J. Campbell, Ph.D.

Benaroya Research Institute at Virginia Mason, Seattle, WA
Control of intestinal inflammation by regulatory T cells

Raja Fayad, M.D.

University of Illinois at Chicago, Chicago, IL
Role of adipokines in inflammatory bowel disease

PHILADELPHIA/DELAWARE VALLEY CHAPTER RESEARCH ALLIANCE

Christopher R. Gasink, M.D.

University of Pennsylvania School of Medicine, Philadelphia, PA
Transcriptional regulation of regulatory T cells in experimental colitis

Xiaonan Han, Ph.D.

Children's Hospital Medical Center, Cincinnati, OH
Characterization of STAT5b as a novel therapeutic target in Crohn's disease

Manisha Harpavat, M.D.

Children's Hospital of Pittsburgh, Pittsburgh, PA
Altered bone mineral metabolism in children with inflammatory bowel disease

Laurie E. Harrington, Ph.D.

University of Alabama at Birmingham, Birmingham, AL
Dynamic regulation between TH1 and TH17 cells during inflammatory bowel disease

Ossama A. Hatoum, M.D.

Medical College of Wisconsin, Milwaukee, WI
Microvascular dysfunction in inflammatory bowel disease

ALABAMA/NORTHWEST FLORIDA CHAPTER RESEARCH ALLIANCE

Robin D. Hatton, Ph.D.

University of Alabama at Birmingham, Birmingham, AL
Identification of distant IL-10 regulatory elements

Eric Houpt, M.D.

University of Virginia/Digestive Health Center, Charlottesville, VA
Control of innate intestinal inflammation in mice

WILLIAM AND SHELBY MODELL FAMILY FOUNDATION RESEARCH AWARD

Li-Chung Hsu, Ph.D.

University of California, San Diego, La Jolla, CA
The molecular mechanism of NOD2 in the pathogenesis of Crohn's Disease

MICHAEL S. MODELL MEMORIAL RESEARCH FUND AWARD

Andrei Ivanov, Ph.D.

Emory University, Atlanta, GA
The role of endocytic recycling of junctional proteins during mucosal restitution IBD

IN HONOR OF MELISSA ANNE MILLER IN HONOR OF AMANDA BASH CAROLINAS CHAPTER RESEARCH ALLIANCE

Sandra C. Kim, M.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Different bacterial species selectively induce TH1 cells

Christine McDonald, Ph.D.

The University of Michigan, Ann Arbor, MI
Molecular mechanisms of activation and regulation of the Crohn's disease associated protein NOD2

GREATER SAN DIEGO AND DESERT AREA CHAPTER RESEARCH ALLIANCE

Declan F. McCole, Ph.D.

University of California, San Diego, San Diego, CA
The influence of reactive oxygen species on signaling underlying secretory diarrhea in IBD

J. Rodrigo Mora, M.D., Ph.D.

CBR Institute for Biomedical Research, Boston, MA
Microenvironmental programming of intestinal dendritic cells to imprint gut-tropism in lymphocytes

Brian M. Necela, Ph.D.

Mayo Clinic Jacksonville, Jacksonville, FL
Cellular and molecular targets essential for the protection against the development of ulcerative colitis

MICHIGAN CHAPTER RESEARCH ALLIANCE

Mary X. O'Riordan, Ph.D.

University of Michigan, Ann Arbor, MI
Identification of regulators of the pro-inflammatory host intracellular surveillance pathway

Steven Polyak, M.D.

University of Florida, Gainesville, FL
Targeted gene delivery to the intestinal epithelium for the study and treatment of colitis

Brian K. Reuter, Ph.D.

University of Virginia Health Center, Charlottesville, VA
Characterization and role of Beta-Defensins and CCR6-positive immune cells in murine models of Crohn's disease-like ileitis

LAURA ELLEN AND ROBERT MUGLIA RESEARCH FUND AWARD

ILLINOIS CAROL FISHER CHAPTER RESEARCH ALLIANCE

Suzana D. Savkovic, Ph.D.

University of Illinois at Chicago, Chicago, IL
Signaling pathways of inflammation in intestinal epithelia in response to enteropathogenic E. Coli

WILLIAM AND SHELBY MODELL FAMILY FOUNDATION RESEARCH AWARD

Jishu Shi, Ph.D.

Auburn University, Auburn, IL
Role of defensins in the pathogenesis of inflammatory bowel disease

LAWRENCE F. SCHWARTZ MEMORIAL FUND FOR PEDIATRIC RESEARCH IN IBD AWARD

Steven J. Steiner, M.D.

Indiana University, Indianapolis, IN
Metabolic response to anti-tumor necrosis factor-alpha (infliximab) in pediatric Crohn's disease

Matthew John Tyska, Ph.D.

Vanderbilt University Medical Center, Nashville, TN
Investigating the role(s) of actin-based motor proteins in the maintenance of brush border structure and composition, and the response to bacterial pathogens implicated in inflammatory bowel disease

Brian K. Weaver, Ph.D.

Washington University School of Medicine, St Louis, MO
Elucidating the anti-inflammatory functions of a novel IL-10 induced gene

Bo Wei, M.D., Ph.D.

University of California, Los Angeles, Los Angeles, CA
Regulatory B cells in mucosal homeostasis and inflammatory bowel disease

MICHAEL S. MODELL MEMORIAL RESEARCH FUND AWARD

Xianyang Yio, M.D., Ph.D.

Mount Sinai School of Medicine, New York, NY
Functional characterization of CD8+ intestinal regulatory T cell (TrE) and defects associated with its activation in IBD

RESEARCH FELLOWSHIP AWARDS

SHAPIRO FAMILY RESEARCH AWARD ANONYMOUS DONOR, ORANGE COUNTY

A. Abadia-Molina, Ph.D.

Beth Israel Deaconess Medical Center, Boston, MA
The role of co-stimulatory molecules, SLAM in experimental colitis

RONALD A. KRANCER RESEARCH AWARD

David Artis, Ph.D.

University of Pennsylvania School of Medicine, Philadelphia, PA
Role of NF- κ B in preventing chronic intestinal inflammation

GREATER WASHINGTON, D.C./VIRGINIA CHAPTER RESEARCH ALLIANCE

Giorgios Bamias, M.D.

University of Virginia Health Center, Charlottesville, VA
Role of bacterial flora in the initiation and perpetuation of ileitis in a spontaneous model of Crohn's disease

GREATER NEW YORK CHAPTER RESEARCH ALLIANCE

Geraldine O. Canny, Ph.D.

Brigham and Women's Hospital, Boston, MA
Regulation of intestinal epithelial BPI

Elmer Yeong-Shin Chang, M.D.

University of California, Los Angeles, Los Angeles, CA
The role of RIP2 in NOD2 signal transduction and murine enterocolitis

ELVIN AND JANET PRICE RESEARCH AWARD

Laetitia Charrier, Ph.D.

Emory University, Atlanta, GA
Colonic leptin: Source of a novel pro-inflammatory cytokine involved in IBD

Alex Chung Kyu Chin, Ph.D.

Emory University, Atlanta, GA
Effect of neutrophil transmigration and epithelial pathophysiology in inflammatory bowel disease

Beckley K. Davis, Ph.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
A Novel CATERPILLER gene involved in mucosal inflammation

GATEWAY CHAPTER RESEARCH ALLIANCE

Dawn Renae Ebach, M.D.

Washington University School of Medicine, St. Louis, MO
Tumor necrosis factor receptors in colitis

THE INFLAMMATORY BOWEL DISEASES EDITORIAL BOARD RESEARCH AWARD

Benjamin Faustin, Ph.D.

The Burnham Institute, La Jolla, CA
Biochemical mechanisms regulating NAC function in IL-1 β processing

TENNESSEE CHAPTER RESEARCH ALLIANCE

Mark Frey, Ph.D.

Vanderbilt University School of Medicine, Nashville, TN
Regulation of intestinal epithelial cell migration and proliferation by EGFR and p38 MAPK

FAIRFIELD/WESTCHESTER CHAPTER RESEARCH ALLIANCE

Cosmas Giallourakis, M.D.

Massachusetts General Hospital, Boston, MA
IBD gene identification on chromosome 19P using a comprehensive haplotype approach

LEON AND MARYSUE WECHSLER RESEARCH AWARD

James B. Hamburger, Ph.D.

University of Pennsylvania School of Medicine, Philadelphia, PA
The biophysical characterization of NOD2 interactions with muramyl dipeptide and synthetic bacterial peptidoglycans

Refaat A.F. Hegazi, M.D., Ph.D.

University of Pittsburgh, Pittsburgh, PA
Immunomodulatory effects of carbon monoxide in mouse models of Th1 and Th2 colitis

Osamu Hitotsumatsu, M.D.

University of California, San Francisco, San Francisco, CA
The role of dendritic cell toll-like receptors in IBD

Shien Hu, M.D.

The University of Chicago, Chicago, IL
Post-transcriptional regulation of heat shock proteins by pro-inflammatory cytokines: Contribution to IBD pathogenesis

Ivaylo Ivanov, Ph.D.

New York University School of Medicine, New York, NY
Role of ROR γ t+ cells and cryptopatches in the pathogenesis of IBD and mucosal immunity

Rheinallt M. Jones, Ph.D.

Emory University, Atlanta, GA
A novel genetic system to study host bacterial interactions

Thomas Karrasch, M.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Molecular mechanisms of wound-healing induced gene expression in the intestine

LONG ISLAND CHAPTER RESEARCH ALLIANCE

Arthur Kaser, M.D.

Brigham and Women's Hospital, Boston, MA
Role and function of CD1d-restricted natural killer T cells in experimental colitis

Hon Wai Koon, Ph.D.

Beth Israel Deaconess Medical Center, Boston, MA
Mechanisms of the healing effects of substance P in intestinal inflammation

Matam V. Kumar, Ph.D.

Emory University, Atlanta, GA
Role of TLR5 and flagellin in inflammatory bowel disease

Narendra Kumar, Ph.D.

University of Tennessee Health Science Center, Memphis, TN
Role of JAK3-villin interaction in the restitution of intestinal epithelial cells

THE CCFA/CENTOCOR SEARCH FOR THE CURE RESEARCH AWARD

Gaëlle Anne Marie Le'Negrate, Ph.D.

The Burnham Institute, La Jolla, CA
Cloning and functional characterization of bacterial proteins similar to SUMOYases

JOSEPH MARINO RESEARCH ALLIANCE

Alusha A. Mamchak, Ph.D.

University of California, San Francisco, San Francisco, CA
Examination of the mechanism by which inflammatory bowel disease arises as a genetic loss of FYN kinase, a molecule involved in antigen receptor signaling

Brian G. Morris, M.D.

University of Alabama at Birmingham, Birmingham, AL
Bystander regulation of colitigenic T cells

Takashi Nagaishi, Ph.D.

Brigham and Women's Hospital, Boston, MA
Characterization of CEACAM1, a novel regulatory molecule in mucosal T cells

ELLEN CROWN PROFILE IN COURAGE RESEARCH AWARD

Makoto Naganuma, M.D., Ph.D.

University of Virginia Health Center, Charlottesville, VA
Factors controlling the differentiation of regulatory T cells and their effect on intestinal inflammation

ROCKY MOUNTAIN CHAPTER RESEARCH ALLIANCE

Jan Hendrik Niess, M.D.

Massachusetts General Hospital, Boston, MA
Antigen sampling and presentation by intestinal dendritic cells

Marie Anne O' Donnell, Ph.D.

Mount Sinai School of Medicine, New York, NY
Regulation of TNF-induced NF-Kb activation and cell death by ubiquitination of RIP

MICHIGAN CHAPTER RESEARCH ALLIANCE

THOMAS H. COBB RESEARCH AWARD

Yasunori Ogura, M.D., Ph.D.

Yale University, New Haven, CT
Structure-function analysis of Nod2-LPS interaction

LONG ISLAND CHAPTER RESEARCH ALLIANCE

Asit Parikh, M.D., Ph.D.

Massachusetts General Hospital, Boston, MA
Molecular mechanisms of colorectal cancer in inflammatory bowel disease

Stephen F. Parsons, M.D., Ph.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Effects of chronic maternal inflammation on fetal development, pulmonary and neurologic outcomes using a murine model of TH-1 mediated colitis

BiFeng Qian, Ph.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Defective response to immunoregulatory signals in HLA-B27 transgenic rats

Sang Hoon Rhee, Ph.D.

Beth Israel Deaconess Medical Center, Boston, MA
The role of toll-like receptor 5-mediated signaling pathways in IBD

JOAN AND HARRY FALK RESEARCH FELLOWSHIP AWARD OF THE CROHN'S & COLITIS FOUNDATION OF AMERICA GREATER NEW YORK CHAPTER

Svend Tafford Rietdijk, M.D.

Beth Israel Deaconess Medical Center, Boston, MA
Control of experimental colitis by the cell surface receptor Ly108

Rachael Jane Rigby, Ph.D.

University of North Carolina at Chapel Hill, Chapel Hill, NC
Non-immune roles of suppressor of cytokine signaling 3 (SOCS3)

Marcos W. Steinberg, Ph.D.

La Jolla Institute for Allergy and Immunology, San Diego, CA
Interaction of LIGHT with HVEM in APC delays colitis and inflammation in the CD+CD45RBhigh T cell transfer model of colitis

Ken Sugimoto, M.D., Ph.D.

Massachusetts General Hospital East, Boston, MA
Role of interleukin-22 in epithelial cell function and colitis

Monisha Sundarajan, Ph.D.

La Jolla Institute for Allergy and Immunology, San Diego, CA
Role of CD8aa+CD4 T cells in pathogenesis and prevention of colitis

COWLES CHARITABLE TRUST RESEARCH AWARD

Guo-Zhong Tao, Ph.D.

Palo Alto VA Medical Center, Palo Alto, CA
Keratin mutations as a risk factor for inflammatory bowel disease

Marcela K. Tello-Ruiz, Ph.D.

Broad Institute/Massachusetts Institute of Technology, Cambridge, MA
Identification of the genetic variation on chromosome 3p conferring susceptibility to IBD

Michal F. Tomczak, M.D.

Brigham and Women's Hospital, Boston, MA
Inhibitory role of NF-kb within the innate immune system in H. Hepaticus-induced colitis

Truc Thi Trinh, M.D.

University of Virginia Health Center, Charlottesville, VA
The role of IL-18 promoter polymorphisms in inflammatory bowel disease

Flavia A. Wald, Ph.D.

University of Miami, Miami, FL
Biogenesis of the apical ezrin scaffold and diarrheal disorders

Thomas Robert Walker, M.D.

The Children's Hospital of Boston, Boston, MA
Phase I evaluation of urinary isoprostane levels in pediatric patients with inflammatory and non-inflammatory gastrointestinal disease

Marc-Andre Wurbel, Ph.D.

The Children's Hospital of Boston, Boston, MA
CCR9 and CCL25 in the gut-specific T lymphocytes homing

Yutao Yan, Ph.D.

Emory University, Atlanta, GA
Role of integrin associated protein CD98 in intestinal permeability

Masaru Yoshida, M.D., Ph.D.

Brigham and Women's Hospital, Boston, MA
The role of the Fc receptor for IgG (FcRN) in regulating colitis

David Alexander Ziring, M.D.

University of California, Los Angeles, Los Angeles, CA
Manipulation of immunoregulatory B cells in colitis

CCFA/CDHNF CO-SPONSORED YOUNG INVESTIGATOR AWARD

CHILDREN'S DIGESTIVE HEALTH AND NUTRITION FOUNDATION (CO-SPONSORED AWARD THROUGH NASPGHAN)

Michael C. Stephens, M.D.

Medical College of Wisconsin, Milwaukee, WI
ABC Transporter Pharmacogenetics: Impact on Thiopurine Therapy

STUDENT FELLOWSHIP AWARDS

WARREN AND ROBERTA SIRZYK RESEARCH AWARD

Ksenia Borisova

Medical College of Wisconsin, Milwaukee, WI
Characterization of the bacterial flora in gastrointestinal health and disease

Nicholas E. Burjek

The University of Chicago, Chicago, IL
Role of inducible heat shock protein 72 in immunoregulation of the gastrointestinal tract

CHRISTOPHER D. MAIER, SR. RESEARCH AWARD

Jennifer V. Chillemi

Fox Chase Cancer Center, Philadelphia, PA
Characterization of peptidoglycan recognition by NOD2

Christian Hudert

Massachusetts General Hospital, Boston, MA
Dextran sodium sulfate induced colitis in fat-1 transgenic mice

WARREN AND ROBERTA SIRZYK RESEARCH AWARD

Camalla M. Kimbrough

University of Alabama at Birmingham, Birmingham, AL
P-Glycoprotein and responses to microbial ligands

TENNESSEE CHAPTER RESEARCH ALLIANCE

Brandon T. Larsen

Medical College of Wisconsin, Milwaukee, WI
Cytochrome P450 enzymes and microvascular dysfunction in inflammatory bowel disease

TENNESSEE CHAPTER RESEARCH ALLIANCE

Laura McDaniel

University of Illinois at Chicago, Chicago, IL
Bacteria mediated suppression of inflammation

Brian Pabst

Northwestern University, Chicago, IL
The capacity for 5-ASA to prevent cancer in IBD

Jennifer L. Pasko

Massachusetts General Hospital, Boston, MA
*The role of the probiotic yeast, *saccaromyces boulardii*, during infection with shigella flexneri*

Vikram Prasanna

University of Pittsburgh, Pittsburgh, PA
HMGB1 as an endogenous danger signal in inflammatory bowel disease

Sadeea Qureshi

University of Nevada School of Medicine, Reno, NV
Role of phospholamban in PGE2-induced relaxation of colonic smooth muscles

Jami A. Rothe

The University of Chicago, Chicago, IL
Does degree of inflammation increase risk for colorectal cancer in patients with chronic inflammatory bowel disease?

Jacob Russal

Fox Chase Cancer Center, Philadelphia, PA
Characterization of NOD2-RIP2 interaction

Aliese Sarkissian

Massachusetts General Hospital, Boston, MA
Investigation of the effect of bacterial superantigen on epithelial tight junctions

Rupak Shivakoti

Massachusetts General Hospital, Boston, MA
*Identification and characterization of receptor specificity of probiotic *Lactobacillus* during salmonella and listeria infection in human intestinal epithelium*

Kathryn Straub

University of Illinois at Chicago, Chicago, IL
*Determining if enteropathogenic *E. coli* (EPEC) Orf3 is a pathogenic effector*

TENNESSEE CHAPTER RESEARCH ALLIANCE

Vyas Viswanathan

Northwestern University, Chicago, IL
The role of PI3K/Akt pathway in T cell-induced crypt cell apoptosis

TENNESSEE CHAPTER RESEARCH ALLIANCE

Amy Yang

University of Illinois at Chicago, Chicago, IL
The role of NF- κ B, regulator of inflammation, in intestinal epithelial proliferation

TENNESSEE CHAPTER RESEARCH ALLIANCE

Feilin Alicia Zhu

Brigham and Women's Hospital, Boston, MA
CD1d-restricted natural killer T cells in experimental colitis

WORKSHOPS/CONFERENCES

Theodore M. Bayless, M.D.

The Johns Hopkins University School of Medicine, Baltimore, MD
IBD Junior Faculty Symposium

LONG ISLAND CHAPTER RESEARCH ALLIANCE

Judy H. Cho, M.D.

The University of Chicago, Chicago, IL
Eighth Meeting of The IBD International Genetics Consortium

Richard B. Colletti, M.D.

University of Vermont, Burlington, VT
PIBDNET improvement collaborative meeting

Peter B. Ernst, Ph.D.

University of Virginia Health Center, Charlottesville, VA
Microbes and Mucosal Immunity

Torsten Kucharzik, M.D.

University Hospital of Muenster, Muenster, Germany
Inflammatory Bowel Disease — Research Drives Clinics, Genetics, Barrier Function, Immunologic and Microbial Pathways

Lloyd F. Mayer, M.D.

Mount Sinai School of Medicine, New York, NY
International Congress of Mucosal Immunology

Asma Nusrat, M.D.

Emory University, Atlanta, GA
Epithelial mesenchymal transition and oncogenesis

Jerrold R. Turner, M.D., Ph.D.

The University of Chicago, Chicago, IL
Gastrointestinal Tract XI: Innovations in GI research and therapy

Severine Vermeire, M.D., Ph.D.

University Hospital Gasthuisberg, Belgium, Germany
Ninth Meeting of the IBD International Genetics Consortium

RESEARCH ALLIANCES

This program allows individuals in a chapter to pool their resources, thereby raising the funds to sponsor a research grant approved by CCFA's National Scientific Advisory Committee.

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- Carolinas Chapter Research Alliance
- Central Ohio Chapter Research Alliance
- Fairfield/Westchester Chapter Research Alliance
- Florida Chapter Research Alliance
- Gateway Chapter Research Alliance
- Georgia Chapter Research Alliance
- Greater Los Angeles Chapter Research Alliance
- Greater New York Chapter Research Alliance
- Greater San Diego and Desert Area Chapter Research Alliance
- Greater Washington, D.C./Virginia Chapter Research Alliance
- Houston Gulf Coast/South Texas Chapter Research Alliance
- Illinois Carol Fisher Chapter Research Alliance
- Indiana Chapter Research Alliance
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