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WHITAKER LOGAN

Understanding Crohn's Disease: Immunity, Genes and Microbes

Academic Press

Inflammatory Bowel Disease (IBD) is a chronic debilitating disorder that occurs at any age and in populations around the

world. Its pathogenesis is believed to involve a combination of genetic susceptibility, immune and external environmental factors, including the gut microbiota. Changing factors such as diet and the human gut microbiota may thus be a viable alternative to suppressing the innate and adaptive immune responses. The book at hand starts with a summary of the current understanding of the epidemiology and biologic underpinnings that manifest as IBD. Next, the gut microbiota, its function, and how it may interact with nutritional status in perpetuating IBD are looked at, followed by discussions on the potential for manipulation of the gut microbiota through the use of prebiotics, probiotics, antibiotics, and fecal transplantation. Chapters on the current role of and future prospects for nutritional interventions in the management of IBD complete the topics presented.

Crohn's Disease, an Issue of Gastroenterology Clinics of North America John Wiley & Sons

The human microbiome refers to the complete microorganisms inhabiting the human body sites including skin, ear, nose, oral cavity, the genital, gastrointestinal and respiratory tracts, and body fluids such as breast milk, saliva, and urine. It is a significant and essential organ recognized for the body and has an established involvement in the host wellbeing, in terms of nutritional requirements and immunomodulation. This book talks about how alteration and imbalance in the same can have clinical

implications associated with a multitude of gastrointestinal, lifestyle-associated, and neurodegenerative disorders. How the proliferation of specific groups of bacteria and their metabolic activities, as a result of intestinal dysbiosis leads to the 'leaky gut' condition thereby influences brain activity via the bidirectional gut-brain axis. It also coves the importance of microbial seeding and how it can be influenced by the mode of delivery, nutrition, and medication. This book also provides various therapeutic interventions such as the establishment of stool banks and Faecal microbiota transplantation (FMT) that have recently proved promising in the treatment of ASD, Inflammatory Bowel Disease, and Ulcerative Colitis. This book provides a deeper understanding of the development of the human gut microbiome and the factors driving its dysbiosis. This book is a valuable read for health professionals, medical students, nutritionists, and scientific research communities who are eager to update themselves with recent trends in microbiome research. It will also aid gastroenterologists and nutritionists to make well-informed choices regarding therapeutic regimes.

Gut Microbiota and Inflammation: Relevance in Cancer and Cardiovascular Disease Biota Publishing

The Developing Microbiome: Lessons from Early Life focuses on the establishment of the microbiome in early life, exposing it as a key mediator of diseases and health throughout the lifecycle. The content presents a

comprehensive view of the status of the field and draws real-world correlations to health and disease states. It collates the significant research being done in the pediatric microbiome research space and bridges the knowledge gap showing the factors that impact health and disease states throughout the lifecycle. Finally, it offers knowledge on how the microbiome is and can be manipulated to promote change. This is a perfect reference for both researchers and clinical scientists who are interested in the role of the infant microbiome in health and disease, as well as gastroenterologists and pediatricians looking to affect change in their patients. Provides comprehensive coverage of the factors that influence microbiome development Links research in pediatric patients to later life stages Examines increasing evidence on the impact of the microbiome beyond the gut

The Role of Microbiota in the Onset and Development of Intestine and Liver Diseases and Cancer: Molecular and Cell Mechanisms

Humana

This book provides a comprehensive overview of metabonomics and gut microbiota research from molecular analysis to population-based global health considerations. The topics include the discussion of the applications in relation to metabonomics and gut microbiota in nutritional research, in health and disease and a review of future therapeutical, nutraceutical and clinical applications. It also examines the translatability of systems biology

approaches into applied clinical research and to patient health and nutrition. The rise in multifactorial disorders, the lack of understanding of the molecular processes at play and the needs for disease prediction in asymptomatic conditions are some of the many questions that system biology approaches are well suited to address. Achieving this goal lies in our ability to model and understand the complex web of interactions between genetics, metabolism, environmental factors and gut microbiota. Being the most densely populated microbial ecosystem on earth, gut microbiota co-evolved as a key component of human biology, essentially extending the physiological definition of humans. Major advances in microbiome research have shown that the contribution of the intestinal microbiota to the overall health status of the host has been so far underestimated. Human host gut microbial interaction is one of the most significant human health considerations of the present day with relevance for both prevention of disease via microbiota-oriented environmental protection as well as strategies for new therapeutic approaches using microbiota as targets and/or biomarkers. In many aspects, humans are not a complete and fully healthy organism without their appropriate microbiological components. Increasingly, scientific evidence identifies gut microbiota as a key biological interface between human genetics and environmental conditions encompassing nutrition. Microbiota dysbiosis or variation in metabolic

activity has been associated with metabolic deregulation (e.g. obesity, inflammatory bowel disease), disease risk factor (e.g. coronary heart disease) and even the aetiology of various pathologies (e.g. autism, cancer), although causal role into impaired metabolism still needs to be established. Metabonomics and Gut Microbiota in Nutrition and Disease serves as a handbook for postgraduate students, researchers in life sciences or health sciences, scientists in academic and industrial environments working in application areas as diverse as health, disease, nutrition, microbial research and human clinical medicine.

Bacterial epithelial interaction in intestinal inflammation Frontiers Media SA

The intestine is constantly exposed to bacteria, invading viruses and ingested food. The intestinal barrier serves as a gate preventing passage of harmful components, and at the same time maintaining absorption of nutrients and water. There are over 300 different bacteria species in the human gastrointestinal tract (GI) comprising over 10 times as many cells as the human body. These bacteria are both of commensal and pathogenic strains in which commensal bacteria and antimicrobial peptides have an important role of controlling the intestinal colonization. The intestinal flora is sampled by the membranous cells (M cells) that are present in the follicle associated epithelium (FAE). Antigens encounter immune cells found

in Peyer's patches located in the distal ileum with FAE overlaying them. Due to environmental factors, genetic predisposition, immune dysregulation or dysbiosis the balance can be shifted which, in turn, will lead to the defect in the barrier function, leading to the development of disorders such as Crohn's disease (CD). CD is a chronic inflammation in the GI tract, often originating in the distal ileum in FAE and associated with an increased number of adherent invasive strains of bacteria. Specifically adherent invasive E.coli (AIEC) that have been isolated from the ileum and colon of CD patients. The aim of the present thesis was to study bacterial epithelial interaction during inflammation in in vivo, ex vivo and in vitro models. In the first project we found that that Faecalibacterium prausnitzii (FP), possess anti-inflammatory properties in the ileum of an in vivo DSS induced colitis mouse model. In the second project, we discovered that infliximab, known to have anti-inflammatory effects by binding soluble TNF and blocking TNF receptors, reduces bacterial transcytosis across colonic biopsies of CD patients and decreases transcytosis and internalization in cell monolayers in vitro. Moreover, we demonstrated that HM427 bacteria, isolated from colonic mucosa of CD patients, uses lipid raft formations to penetrate the barrier under the influence of TNF in an in vitro model. In project three, we demonstrated that LF82 bacteria, which is an adherent invasive strain of E.coli

that has been isolated from the ileum of CD patients, exploits FAE of CD patients and non-IBD control patients to penetrate the barrier via the CEACAM6 receptor and long polar fimbriae. We further demonstrated that there is an increased expression of CEACM6 receptor in the FAE of CD patients, which leads to increased transcytosis of LF82 compared to non-IBD control group. In project four, our results suggested that human ?-defensin 5 significantly decreases the passage of LF82 bacteria in an in vitro and ex vivo models. Moreover, we demonstrated that CD patients have a lower expression of human ?-defensin 5 in the crypts compared to the non-IBD control patients. Taken together, our findings have given a novel insight into the etiology of CD and into the mechanisms involved in bacterial-epithelial interaction in CD.

The Human Microbiota and Chronic Disease Nova Science Publishers

Dr. Loftus is a widely recognized expert in the diagnosis and treatment of Crohn's disease. He has created an issue devoted the current state-of-the-art on Crohn's disease; authors have written comprehensive reviews on the latest research to inform clinical diagnosis and treatment. Articles are devoted to the following topics: Genetics; Epidemiology, natural history, and risk stratification of Crohn's disease; The microbiome in Crohn's disease: Role in pathogenesis and role of microbiome replacement therapies; Endoscopic and radiographic assessment of Crohn's disease; Intestinal

and non-intestinal cancer risk in Crohn's disease; Sexuality, fertility, and pregnancy in Crohn's disease; Interdisciplinary management of perianal Crohn's disease; Targeting specific immunologic pathways in human inflammatory bowel disease; Evolution of treatment paradigms in Crohn's disease; Preventing and managing postoperative recurrence of Crohn's disease; Where and how to use anti-TNF agents and anti-integrins in Crohn's disease; Ustekinumab and anti-interleukin-23 agents in Crohn's disease; Update on therapeutic drug monitoring in Crohn's disease; and Janus kinase antagonists and other novel small molecules for the treatment of Crohn's disease. Readers will come away from this issue armed with the information they need to improve management of this disease as well as patient outcomes.

The Gut Microbiome in Health and Disease Springer

Individually and collectively, resident microbes play important roles in host health and survival. Shaping and shaped by their host environments, these microorganisms form intricate communities that are in a state of dynamic equilibrium. This ecologic and dynamic view of host-microbe interactions is rapidly redefining our view of health and disease. It is now accepted that the vast majority of microbes are, for the most part, not intrinsically harmful, but rather become established as persistent, co-adapted colonists in equilibrium with their environment, providing useful goods and

services to their hosts while deriving benefits from these host associations. Disruption of such alliances may have consequences for host health, and investigations in a wide variety of organisms have begun to illuminate the complex and dynamic network of interaction - across the spectrum of hosts, microbes, and environmental niches - that influence the formation, function, and stability of host-associated microbial communities. Microbial Ecology in States of Health and Disease is the summary of a workshop convened by the Institute of Medicine's Forum on Microbial Threats in March 2013 to explore the scientific and therapeutic implications of microbial ecology in states of health and disease. Participants explored host-microbe interactions in humans, animals, and plants; emerging insights into how microbes may influence the development and maintenance of states of health and disease; the effects of environmental change(s) on the formation, function, and stability of microbial communities; and research challenges and opportunities for this emerging field of inquiry.

Microbial Ecology in States of Health and Disease CRC Press

The field of microbial endocrinology is expressly devoted to understanding the mechanisms by which the microbiota (bacteria within the microbiome) interact with the host (“us”). This interaction is a two-way street and the driving force that governs these interactions are the neuroendocrine products of both the

host and the microbiota. Chapters include neuroendocrine hormone-induced changes in gene expression and microbial endocrinology and probiotics. This is the first in a series of books dedicated to understanding how bi-directional communication between host and bacteria represents the cutting edge of translational medical research, and hopefully identifies new ways to understand the mechanisms that determine health and disease.

The Developing Microbiome Springer Science & Business Media

Gastrointestinal dysbiosis has been noted in a myriad of conditions in companion animal and human medicine, including inflammatory bowel disease (IBD), immune mediated disease, and critical illnesses. The literature on the microbiota in conditions other than IBD, as well as manipulation of the microbiota through fecal microbial transplant (FMT) on dogs is limited. As a result, an objective of this thesis was to compare the fecal microbiota of healthy dogs, dogs with IBD, and dogs with critical illness. Additional objectives were to compare the efficacy FMT in addition to standard treatment (hypoallergenic diet and immunosuppressive medications) in dogs with IBD versus standard treatment alone in inducing clinical remission, to describe the alteration in the fecal microbiota in dogs undergoing medical therapy for IBD, and to detail a protocol for preparation and administration of FMT in dogs. In the first study, ten healthy dogs, nine dogs with IBD, and 29 dogs with critical illness (nine dogs with

immune mediated hematologic disease (IM), and 20 dogs with other critical illness (NIM)) were recruited. DNA extraction from fecal samples and PCR analysis of the 16S gene were subsequently performed, followed by sequencing using an Illumina MiSeq platform. There was a significant difference between groups for community structure (Yue and Clayton index, p=0.039), and community composition (Jaccard index, p0.001). The Jaccard index was significantly different between healthy and diseased (IBD, NIM, IM) groups (p0.001), but not between individual diseased groups (p0.05). In the second study, 13 client-owned dogs with IBD were enrolled. All patients received corticosteroid therapy and a hypoallergenic diet; patients were randomized to receive either placebo or fecal microbial transplant (FMT). Measured outcomes included the canine chronic enteropathy clinical activity index (CCECAI) along with measurement of serum albumin, C-reactive protein (CRP), and cobalamin levels at 1 week, 1 month, and 3 months after enrolment. Fecal microbiota were analyzed following DNA extraction and profiling using 16S amplicon sequencing. The CCECAI significantly decreased over time regardless of treatment group (p = 0.001). Dogs receiving FMT had improved disease severity scores when assessed 30 days after enrolment (p=0.02), whereas dogs administered a placebo with standard therapy did not (p=0.61). However patient outcome was not affected as differences between

treatment groups in the CCECAI (p = 0.735), albumin (p=0.43), CRP (p = 0.287), or cobalamin (p = 0.601) were not observed after 90 days of treatment. No adverse effects were reported after FMT. The alpha and beta diversity measurements were not significantly different between pre and one week post FMT samples (p 0.05). Fecal samples were significantly enriched in Faecalibacterium (p=0.047) one week following FMT. In conclusion, dogs with IBD and dogs with critical illness had a similar fecal microbiota community membership, which was significantly different from healthy dogs. Additionally, FMT was an easily applicable treatment option in canine IBD which resulted in improved disease severity scores early after therapy whereas placebo treatment did not, although it did not change longer term patient outcome or fecal microbiota diversity in dogs with IBD in this study.

Fecal Microbiota Alterations in Illness and Efficacy of Fecal Microbiota Transplantation in Treatment of Inflammatory Bowel Disease in Dogs

John Wiley & Sons

This is the first book compiling current research on the gut-bone signaling axis and its implications in the pathophysiology of GI and bone diseases. Rather than focusing on a single mechanism, this book provides the reader with a broad view on gut-bone signaling and the most up-to-date information in this rapidly growing area. The volume is also unique in that it looks at what is known about GI diseases

affecting bone and then examines the role of the microbiome and its modulation by pre and probiotics to treat bone disease, placing this topic within the context of gut-bone signaling pathways. Understanding the Gut-Bone Signaling Axis will thus provide an understanding of how various therapies could be applied to this area.

Advances in Inflammatory Bowel Diseases Springer

In recent years, epidemiological studies have shown a significant increase of incidences regarding ulcerative colitis (UC) in most regions of the world. At present, a common therapeutic modality for inflammatory bowel disorders is the use of anti-inflammatory agents, including sulfasalazine and acetylsalicylic acid, steroid hormone, and other immunosuppressive agents. Most of these treatments are symptomatic and palliative because the etiology of the disease is not yet established. As a result of no proper drug available to treat UC, patients with UC are at a high risk of developing colitis-associated cancer (CAC). It is necessary to understand the genetic and molecular mechanisms involved in the pathogenesis of UC and CAC that lead to the path of new drug discoveries. Diagnostic and Treatment Methods for Ulcerative Colitis and Colitis-Associated Cancer provides innovative insights that describe the fundamental understanding of UC and CAC and the molecular mechanisms behind the etiology as well as modern diagnostic methods that are employed in UC and CAC. Current

prevention and therapeutic strategies practiced in the pre-clinical level are also discussed. The content within this publication examines alternative medicine and dietary intervention and drug delivery techniques. It is designed for healthcare professionals, physicians, academicians, researchers, R&D organizations, and medical students involved in drug discovery and clinical and therapeutic research.

Medical and Surgical Management of Crohn's Disease, An Issue of Gastroenterology Clinics of North America, E-Book Routledge

Despite wide use in our diet, the effects of artificial sweeteners on human health have been inconsistent, with both beneficial and adverse outcomes being reported. Maintaining the balance of gut microbiota and its metabolic functions is vital for human health. However, this balance can be disrupted by various external factors, including chemicals from the diet. Interestingly, it has been reported that artificial sweeteners, such as saccharin, could alter gut microbiota and induce dysbiosis. Inflammation is one of the most common physical conditions associated with the dysbiosis of gut microbiota, which is involved in several diseases, such as inflammatory bowel disease, diabetes, and obesity. Acesulfame-potassium (Ace-K), sucralose and saccharin are three commonly used artificial sweeteners that are found in many foods and beverages and are generally considered to be safe when consumed within the approved amounts. However, little is known about the

functional impact of these artificial sweeteners on gut microbiota. In this dissertation, the effects of Ace-K, sucralose and saccharin on gut microbiota and the changes of fecal metabolic profiles were explored using metagenome sequencing and metabolomics technologies. Also, inflammatory signs were investigated after the consumption of artificial sweeteners. All of these three artificial sweeteners perturbed the gut microbiota and metabolites in mice and increased the bacterial genes that could induce inflammation. In addition, Ace-K consumption for four weeks increased body weight gain and enriched functional bacterial genes of energy metabolism in CD-1 mice with highly sex-specific effects. Consumption of sucralose and saccharin for six months elevated expression of pro-inflammatory genes in the livers of C57BL/6J male mice. Collectively, the results may provide novel understanding of the interaction between artificial sweeteners and gut microbiota, and their potential role in the development of inflammation.

Intestinal Bacteria Associated with Colitis and Inflammatory Bowel Disease MDPI

Crohn's disease (CD) is a chronic, relapsing, inflammatory bowel disease resulting in considerable morbidity and reduced quality of life. Although still under intense debate, CD seems to result from an enhanced and uncontrolled immune response to the gut microbiota. CD is thought to be multifactorial depending on genetic and environmental determinants. In recent

years, nearly 100 single nucleotide polymorphisms (SNPs) were associated with increased risk of developing CD (some of the SNPs also associated with susceptibility to ulcerative colitis, another type of IBD). These SNPs are mostly located in genes involved in innate and adaptive immunity mechanisms, such as autophagy, expression of pattern-recognition receptors and citokine signaling. Epigenetics is also probably playing a role in CD susceptibility, as it is sensitive to environmental conditions and may mediate gene-environment interactions. Environmental factors possibly involved in CD development include diet, gut microbiota composition and infection with specific pathogens, of which the most consistently associated to CD are Mycobacterium avium subsp. paratuberculosis and adherent-invasive Escherichia coli. This Topic aimed at bringing together contributions covering different genetic, epigenetic, immunological and microbial processes involved in the development of CD, helping to drive forward the understanding of CD immunopahtology.

Understanding the Gut-Bone Signaling Axis Frontiers Media SA

Inflammatory bowel disease (IBD) is a set of chronic, relapsing inflammatory diseases of the intestine. The two major subtypes of IBD are Crohn's disease (CD) and ulcerative colitis (UC). Although the pathogenesis of IBD remains largely unknown, Crohn's disease is considered to result from the interaction of environmental factors, including

intestinal microbiota, with host immune mechanisms in genetically susceptible individuals. Recent advances in sequencing technologies have allowed us to characterize the IBD associated dysbiosis in unprecedented depth. However, phylogenetic profiling can only provide limited information on the functional implication of these alterations. To address this analytical challenge, we developed the novel mucosal lavage sampling approach, which enabled the profiling of multi'omic molecular features including microbiome, metaproteome and metabolome. Combined with host genomic information, these tools can provide us with unprecedented understanding of the dynamics of host-microbial interaction, and help us to investigate the pathogenesis of inflammatory bowel diseases. Another analytical challenge to identify microbial taxa consistently representing IBD associated dysbiosis is the high complexity and low inter-individual overlap of intestinal microbial composition. This difficulty can be overcome by an ecologic analytic strategy to identify modules of interacting bacteria (rather than individual bacteria) as quantitative reproducible features of microbial composition in normal and IBD mucosa. We developed the strategy to analyze microbial composition using microbial co-occurrence network approach. This strategy uncovered 5 reproducible functional microbial communities (FMCs) detectable in the mucosa of all

individuals. The quantitative levels of two FMCs were significantly associated with IBD states. Imputed metagenome analysis indicated the functional importance of the disease associated modules reflected by the enrichment of virulent and pathogenic pathways. Thus, these modules appear to define novel microbial communities within the intestinal microbial ecology, some of which are commonly and stably modified by the IBD disease state, and may be of particular relevance for microbial pathogenesis and intervention. Using this experimental and bioinformatic framework, we investigated the microbial gardening effect of FUT2 gene and its link to Crohn's disease. Fucosyltransferase 2 (FUT2) is an enzyme that is responsible for the synthesis of the H antigen in body fluids and on the intestinal mucosa. Non-secretors, who are homozygous for the loss-of-function alleles of FUT2 gene (sese), have increased susceptibility to Crohn's disease. In healthy individuals, imputed metagenomic analysis revealed perturbations of energy metabolism in the microbiome of non-secretor and heterozygote individuals, notably the enrichment of carbohydrate and lipid metabolism, cofactor and vitamin metabolism, and glycan biosynthesis and metabolism related pathways; and, the depletion of amino acid biosynthesis and metabolism. Similar changes were observed in mice bearing the FUT2−1− genotype. Metabolomic analysis of human specimens revealed concordant as well as novel changes in the levels of

several metabolites. Human metaproteomic analysis indicated that these functional changes were accompanied by sub-clinical levels of inflammation in the local intestinal mucosa. In an extended cohort containing both healthy and CD individuals, the phylogenetic composition of intestinal mucosal microbiota was affected by an interaction of Crohn's disease status and FUT2 genotype. Decreased abundances of Firmicutes were associated with both CD and FUT2 risk allele. At metagenomic level, a distinct signature of amino acid metabolism deficiency was identified in CD and non-secretor microbiome. Such changes were also reflected at metabolomic level in the proximal gut region. Taken together, FUT2 gene increased the risk of Crohn's disease by changing the microbial composition and function to a disease-like state. The CD associated perturbations of metagenome and metabolome were driven by the FUT2 risk allele. The same experimental and bioinformatic approach can also be applied to study the composition and functional changes of mucosal associated microbiota in other chronic inflammatory disease, namely HIV-1 infection. In the rectal mucosa, microbial composition and imputed function in HIV-positive individuals not receiving cART was significantly different from HIV-negative individuals. Genera including Roseburia, Coprococcus, Ruminococcus, Eubacterium, Alistipes and Lachnospira were depleted in HIV-infected subjects not receiving cART,

while Fusobacteria, Anaerococcus, Peptostreptococcus and Porphyromonas were significantly enriched. HIV-positive subjects receiving cART exhibited similar depletion and enrichment for these genera, but were of intermediate magnitude and did not achieve statistical significance. Imputed metagenomic functions, including amino acid metabolism, vitamin biosynthesis, and siderophore biosynthesis differed significantly between healthy controls and HIV-infected subjects not receiving cART. In the cervicovaginal mucosa, significant differences in alpha and beta diversity were observed between HIV-negative and HIV-positive women, with the latter enriched of organisms associated with bacterial vaginosis and depleted of Lactobacilli. These ecologic changes occurred concomitantly with significant metagenomic and immunologic differences. Such functional pathways may represent novel interventional targets for HIV therapy if normalizing the microbial composition or functional activity of the microbiota proves therapeutically useful.

Flu Frontiers Media SA

The gastrointestinal (GI) tract is home to trillions of microorganisms and contains more genetic information than that which exists in the human genome. It is, in fact, the largest immune system in the body. Study of the GI tract microbiome and its influence on both health and disease states have demonstrated the importance in maintaining health. The microbiome has a significant role in the assembly of micronutrients and vitamins

and immune system processing. Recently, there has been a focus on the cross-talk between gut immunity and the host microbiome and the subsequent effect of this interaction on a broad range of diseases. The application of next-generation sequencing technologies to the study of human-associated microbial communities has markedly advanced our understanding of these effects. Changes in human-associated microbial communities have been implicated in the etiology and increased incidence of ever growing chronic conditions including obesity, diabetes, and inflammatory bowel disease. Although recognizably understanding the full spectrum of the role of the "gut microbiome" in health and disease is still in a relative infant states, it is clear that our bacterial flora play a much larger role in systemic diseases than previously appreciated. Healthcare for disease management has typically focused on specific therapy with pharmacologic, device or surgical intervention. As we further expand our understanding of the importance of gut microbiome, it is certain that we will see major changes to disease management strategies. Presently, we can see "footprints" of specific bacterial shifts in healthy ones versus those with a disease. Whether shifting the bacteria colonization away from the perceived imbalance in disease, will modify the disease expression remains to be seen. Clearly in the next decade, we will see profound changes in the way we approach current disease

intervention/prevention. The intent of the authors of this book is to provide the most current assessment and analysis of what will likely in the coming decade to be the most exciting expansion in a new understanding of complex relationships of disease pathophysiology as well as therapeutic options for therapy. Additionally, it is the intent not to provide specific answers, but rather hopefully push clinicians to "think outside of the box" and raise great questions to direct research and/or translational therapies for redefining and optimizing "best practice" treatment strategies for our patients!

Probiotic Bacteria and Enteric Infections Clinics: Internal Medicine This book uses new thinking on precision medicine and the interplay of genetic factors, the microbiome, and external triggers to build on the core concepts of inflammatory bowel disease. It outlines the latest findings in targeting therapies to the individual patient with Crohn’s and colitis, management of chronic infections in the setting of immunomodulators and biologics, non-surgical therapy of dysplasia in colitis patients, and redefining and structuring the problematic pouch. In addition, this book features useful chapters dedicated to the economic aspects of IBD in an increasingly constrained healthcare system, as well as the patient experience and the role of subspecialist telemedicine care. Written by specialists and thought leaders in the field, Inflammatory Bowel Disease: Pathogenesis, Diagnosis and

Management provides a concise but highly relevant account of the latest thinking and concepts in IBD.
Gardening Effect of Host Genetics on Human Intestinal Mucosal Microbiome and Its Link to Inflammatory Bowel Disease National Academies Press
In the intestine, a unique immunological system that is different from the systemic immune system exists to provide adaptive immunity in response to luminal bacteria and dietary antigens. There are many lymphoid cell aggregates called gut-associated lymphoid tissue (GALT) including Peyer's patches (PPs), which function as important induction sites for the mucosal immune response. M-cells are present in the epithelium of PPs, having a specialized structure for uptake of macromolecules such as bacteria. In addition to GALT, there are abundant lymphoid cells in the intestinal lamina propria, where they mainly play a role as immune effector cells. A strong innate immune system that mainly consists of dendritic cells, macrophages, and γδT lymphocytes also exists in the intestinal mucosa to assist the barrier function of intestinal epithelial cells. The intestinal mucosa thus shows a unique morphological structure with many immune cells being present under physiological conditions. This condition is known as "controlled inflammation." These abundant immune cells also have characteristic functions: they are "negatively regulated" and have been educated not to overreact unnecessarily to the intestinal luminal milieu. Main

players that control inflammation of the intestinal mucosa include regulatory cytokines and regulatory T cells which induce oral tolerance to intestinal bacteria and food antigens, and the secretory IgA system. The maintenance of unique immunological activity in the intestine is also related to an organized, orchestrated lymphocyte migratory mechanism called the "common mucosal immune system." These negative regulatory mechanisms of the intestinal immune system are disturbed in certain disease conditions, causing the immunocompetent cells to respond to food components and commensal bacteria by becoming activated and to overproduce inflammatory cytokines and chemokines. These disease conditions include food allergies, such as celiac disease, and the inflammatory bowel diseases, such as ulcerative colitis and Crohn’s disease, although their exact etiological mechanisms remain to be revealed. Table of Contents: Introduction / GALT: Its Structure and Formation / Intestinal Epithelial Cells and Their Immune Function / Innate Immunity in the Intestinal Mucosa / Intraepithelial Lymphocytes (IELs) / Lymphoid Cell Trafficking in Intestinal Immunology / Site of Induction of Mucosal Immunity and Antigen Presentation by Dendritic Cells / Production of Secretory IgA (SIgA) / Effector Site of Acquired Immunity and T Helper Cell Subpopulation / Immune Regulatory System and Oral Tolerance / Food Allergy and Celiac Disease / Inflammatory Bowel Diseases / Enteric Infection with Pathogenic Microbes and

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Understanding Crohn's Disease: Immunity, Genes and Microbes Academic Press
Ulcerative colitis and Crohn's disease remain a great therapeutic challenge to the medical community. In recent years knowledge about the pathogenesis of these diseases has progressed rapidly but the cause of the diseases remains completely unknown. It has become clear that dysregulation of the mucosal immune system is the basis for the chronic evolution of the diseases in a genetically susceptible population. Exciting new therapeutic approaches have been attempted in the last couple of years and cytokine and anti-cytokine treatments in particular seem very promising, especially in intractable disease. The format of the Falk Symposium 106 on `Advances in Inflammatory Bowel Diseases', held in Brussels, Belgium, June 18-20, 1998, was somewhat innovative as each session attempted to link the new insights into pathogenetic mechanisms with new therapeutic approaches, resulting in optimal information transfer. The classic therapeutic schemes were updated with a special focus on step-wise build-up of therapy.
Interventional Inflammatory Bowel Disease: Endoscopic Management and Treatment of Complications Springer Nature
Describes the great flu epidemic of 1918, an outbreak that killed some forty million people worldwide, and discusses the efforts of scientists and public health

officials to understand and prevent another lethal pandemic

Modulation of the Brain-Gut-Microbiota Axis in a Murine Model of Inflammatory Bowel Disease Springer

The book provides an overview on how the gut microbiome contributes to human health. The readers will get profound knowledge on the connection between intestinal microbiota and immune defense systems. The tools of choice to study the ecology of these highly-specialized microorganism communities such as high-throughput sequencing and metagenomic mining will be presented. In addition the most common diseases associated to the composition of the gut flora are discussed in detail. The book will address researchers, clinicians and advanced students working in biomedicine, microbiology and immunology.