

Beyond the Scope

A REPORT OF THE UCLA DIVISION OF DIGESTIVE DISEASES



Motility: A Case Study

FROM THE DIVISION CHIEFS



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Going **Beyond** the Scope

These are challenging times for all of us who are healthcare providers and consumers. Changes are taking place in our country that will ultimately impact how patients will access care one way or another. Simultaneously, we are striving to improve quality and control costs. Limited research funding opportunities continue to challenge our efforts to pursue new knowledge. Yet despite these obstacles, our passion for discovery and innovation has not waned!

We feel it is an auspicious time to send you our latest issue of *Beyond the Scope*, and we wish to share new developments, stories of cutting edge medical science, and future collaborations. We are motivated by the tremendous work of our colleagues and want to share the enthusiasm we have for the opportunities before us all. There has never been a more exciting time for science, medicine and education. Many of the advances we are seeing today will enable us to help people in ways that have never before been possible.

We are continually inspired by our patients and their courage to confront their illnesses. We are energized by our young trainees who are dedicating their lives to making society better for us all. You will read about some of these people in the coming pages, and no doubt you have examples from your own personal and professional lives as well. The Division of Digestive Diseases, the Department of Medicine, the David Geffen School of Medicine at UCLA, and UCLA Health are all parts of the global community that is now working to deliver the highest quality care, conduct the most innovative research, and provide the most enriching educational environment possible. We embrace the challenges that confront us and eagerly anticipate the breakthroughs to come.

XPRIZE



With its innovative approach to solving the world’s “grand challenges” the X PRIZE Foundation aims to motivate and inspire brilliant innovators in all disciplines to leverage their intellectual and financial capital for the benefit of humanity. It does so by creating and managing large-scale, high-profile incentivized prize competitions that stimulate investment in research and development worth far more than the prize itself. In April, the Division of Digestive Diseases hosted a reception for the X PRIZE Foundation – an event kicking off what the two organizations hope will develop into a future collaboration.

The reception, which included a presentation by Dr. Peter H. Diamandis, X PRIZE Foundation chair and CEO, was held at Ronald Reagan UCLA Medical Center and attended by X PRIZE Foundation staff as well as senior administrators, staff and prominent faculty from across the UCLA campus – among them Chancellor Gene Block; Dr. A. Eugene Washington, vice chancellor of UCLA Health Sciences and dean of the David Geffen School of Medicine at UCLA; and Dr. David T. Feinberg, CEO of the UCLA Hospital System.

Known for its \$10 million competitions for private spaceflight and 100 mile-per-gallon equivalent cars, the Playa del Rey, CA-based X PRIZE Foundation currently conducts competitions in five prize groups: Education, Exploration, Energy & Environment, Global

Development and Life Sciences. Dr. Diamandis, who received a medical degree from Harvard as well as degrees in molecular genetics and aerospace engineering from MIT, co-founded the company in 1995, modeled after the days when kings and queens would provide incentives for explorers to discover new lands.

“We at X PRIZE believe that solutions to the world’s grand challenges can come from anywhere,” explains Dr. Diamandis, who returned to UCLA in June to deliver a commencement address at the medical school’s graduation ceremonies. “Through large-scale incentivized competitions, we gather innovators from diverse industries and walks of life, singularly focused on achieving technological breakthroughs that will benefit humankind.”

Continued on page 2

The XPRIZE logo features a stylized white 'X' followed by the word 'PRIZE' in a bold, white, sans-serif font. The background of the top half of the page is a photograph of Earth from space, showing a bright sun in the upper right corner and a blue and white horizon line.

Continued from page 1

The connection between the X PRIZE Foundation and UCLA is rooted in the relationship between Dr. Diamandis and Eric Esrailian, MD, MPH, co-chief of the division. Dr. Esrailian serves on the X Prize Foundation Board of Trustees, along with such notables as James N. Gianopoulos, Chief Executive Officer of Fox Filmed Entertainment Inc.; Elon Musk, CEO and chief technology officer of Space Exploration Technologies (SpaceX) and co-founder of PayPal; critically acclaimed film director James Cameron; and Ratan Tata, chairman of the Mumbai-based Tata Group from 1991 to 2012. “X PRIZE is devoted to innovation, and at UCLA we have leading innovators in wide-ranging fields,” Dr. Esrailian notes. “There is a great potential for a partnership addressing some of the big problems facing society.”

Dr. Esrailian points out that within the Division of Digestive Diseases alone, there are several programs focused on innovation, including:

- The Center for Systems Biomedicine, in which a wide variety of experts collaborate using the latest automated technologies to extract data from large numbers of patient tissue samples in an effort to better

understand how diseases originate, and to develop therapies based on that knowledge.

- The Center for Inflammatory Bowel Diseases, which is developing novel approaches to improving quality of life for patients with chronic conditions – including the use of mobile technology for real-time support and to provide the healthcare team with invaluable data to help track patient care and outcomes.
- The Interventional Endoscopy Program, which develops new minimally invasive non-surgical procedures for the diagnosis and treatment of a wide variety of digestive disorders.

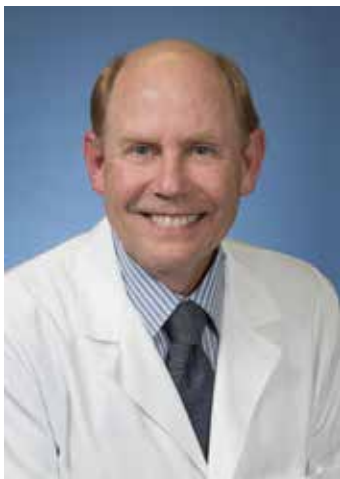
Dr. Diamandis is also co-founder and chair of the Singularity University, a Silicon Valley-based institution that has partnerships with NASA, Google, Autodesk and Nokia. In addition to promoting radical breakthroughs through competitions, he counsels the world’s top enterprises on how to utilize exponential technologies and incentivized innovation to dramatically accelerate their business objectives.

“Peter Diamandis is a visionary, out-of-the-box thinker who imagines things

beyond what is typical and what we take as concrete,” says Nancy Katano, UCLA’s executive director for corporate, foundation and research relations, who is helping to steward the relationship between UCLA and the X PRIZE Foundation. “The competitions established by X PRIZE are in many ways about moving beyond traditional thinking, and that’s something that faculty at a leading research university such as UCLA appreciate. Getting people out of their comfort zones can foster entrepreneurial activity and innovation across our campus, as well as among our donors.”

“We are excited to partner on many dimensions with a forward-looking university such as UCLA,” says Dr. Diamandis. “UCLA is another form of an ‘innovation incubator’ where students from around the globe, representing multiple disciplines and backgrounds, come together to exchange ideas and collaborate to achieve breakthroughs. Merging the intellectual capital of the UCLA ecosystem with X PRIZE’s experience managing incentivized competitions, and capitalizing on the innovative spirit of both entities, could result in a transformational partnership. I look forward to exploring the possibilities.”

Managing Fecal Incontinence in a Patient with Scleroderma: A Case Study



Jeffrey L. Conklin, MD
Medical Director
Center for Esophageal Disorders &
Gastrointestinal Motor Function Laboratory
Professor of Medicine

Jeffrey Conklin, MD, is medical director of the UCLA Center for Esophageal Disorders and Gastrointestinal Motor Function Laboratory. In the following, Dr. Conklin presents a complicated case and how it was properly treated.

Case: MR is a 72-year-old woman with scleroderma who complains of fecal incontinence. She has incontinence when the stool is liquid or soft. It occurs at any time of day, but is cyclical in that the diarrhea occurs every four to six weeks, with 10-15 watery bowel movements per day. These episodes are successfully managed with Imodium, at up to eight dosages per day. MR also has a history of pancreatic insufficiency that was manifest by diarrhea, and responded to pancreatic enzyme replacement.

The patient has a complicated history of pelvic floor surgeries. In 2006 she had a total abdominal hysterectomy, bilateral salpingo-oophorectomy, rectocele repair and bladder suspension. After that, she developed rectal prolapse and fecal incontinence. When the rectal prolapse was repaired, it failed to resolve her incontinence. A colonoscopy with biopsies did not point to any pathological process.

MR had a defecating proctogram, which was incomplete because when the contrast was instilled into the rectum it ran freely out through the anal canal. Her high-resolution anorectal manometry revealed a hypotensive anal sphincter at rest, normal squeeze pressures, loss of the rectoanal inhibitory reflex and rectal hypersensitivity (figure 2). Biofeedback therapy was attempted, but failed to improve her incontinence. A lactulose hydrogen breath test was negative for small bowel bacterial overgrowth. She is now taking Imodium for soft to liquid stools, and when she needs protection.

MR's other GI problem related to scleroderma is heartburn that occurs only at night. It is described as acidic material coming up to and burning the throat. She rarely regurgitates into the mouth, and has rare dysphagia with solid foods. The patient's esophageal manometry revealed hypotensive upper and lower esophageal sphincters and no peristalsis in the smooth muscle esophagus. A gastric emptying study revealed retention of marker in the esophagus, but normal gastric emptying.

Physical Exam: Normal except for tethering of the skin over the hands and face.

Continued on next page

MOTILITY

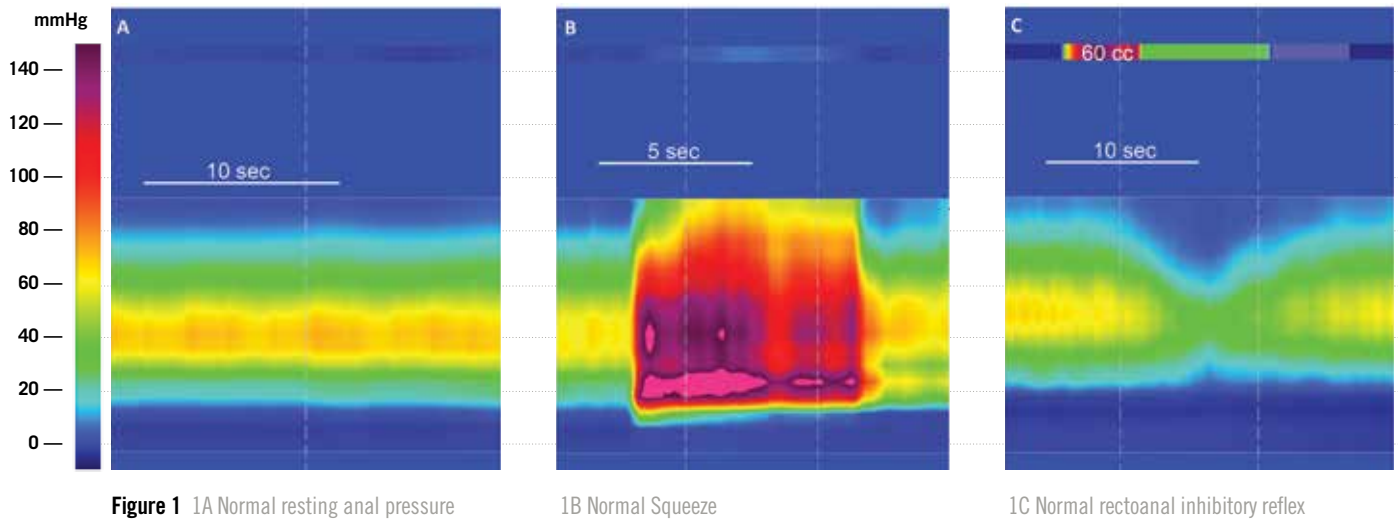


Figure 1 1A Normal resting anal pressure

1B Normal Squeeze

1C Normal rectoanal inhibitory reflex

Discussion: Scleroderma is a multi-system disease of unknown etiology. It is manifest by excess collagen deposition in the skin and internal organs, including those of the gastrointestinal tract; chronic inflammation; and vascular changes. The anorectum is involved in 50-70 percent of patients with scleroderma, and more than 20 percent suffer from fecal incontinence (1,2).

Fecal continence is normally preserved by complex neuromuscular interactions among the central nervous system, pelvic floor, rectum and anal sphincters. The internal anal sphincter (IAS) is composed of smooth muscle that is tonically contracted at rest. It is responsible for about 85 percent of resting anal sphincter pressure. The external anal sphincter (EAS) is a striated muscle that contributes much less to resting anal pressure, but is responsible for the voluntary contraction of the anal sphincter and its reflex contraction during valsalva (3). Weakness or disruption of the IAS typically causes passive fecal incontinence, while dysfunction of the EAS leads to urge and stress incontinence (4,5).

Anorectal dysfunction associated with scleroderma is the result of both myopathic and neuropathic processes. There is a

smooth muscle myopathy characterized pathologically by atrophy and fibrosis of the IAS. It is detected by endoanal ultrasound (EUS) or magnetic nuclear resonance as thinning of the IAS (6,7). Ultrasonography also reveals a hyperechoic IAS, indicating fibrosis. Functionally, it correlates with IAS weakness and a low resting anal canal pressure. MR has a very low resting anal canal pressure consistent with smooth muscle myopathy. Myenteric neuropathy is manifest by loss or attenuation of the rectoanal reflex (RAIR) (8). Our patient has an abnormal RAIR, indicating myenteric neuropathy.

The first step in managing fecal incontinence associated with scleroderma is to solidify liquid stools. This can be done by bulking the stool and using agents that slow colonic transit. Biofeedback has been used, but as with our patient, it frequently is unsatisfactory. Recently, two therapies were approved for the treatment of fecal incontinence. Dextranomer in stabilized hyaluronic acid is a bulking agent that is injected just cephalad to the dentate line. It improves about half of patients with fecal incontinence, but has not been studied specifically in patients with scleroderma (9).

Sacral nerve modulation has been studied specifically in patients with scleroderma, but in limited numbers (10). It decreased the number of incontinent episodes, increased resting and squeeze anal canal pressures and enhanced rectal sensation. Our patient experienced a 60 percent improvement in fecal incontinence with sacral nerve modulation.

Figure 1. Normal anorectal pressure topography. The high-resolution manometry catheter used to obtain these images is comprised of 10 sensors spaced at 6-mm intervals along the anal sensing segment, and rectal pressure is measured in a balloon 3 cm cephalad to the most cephalad anal sensor. Computer software algorithms convert pressure data to the color topographical plots seen in these figures. Panel A is the color contour of a normal resting anal canal pressure. Pressure is represented by color, as indicated by the key along the left side of the figure. Sensor location is on the y-axis and time is on the x-axis. Pressure generated by the resting anal sphincters is seen as a broad band of bright color (double-headed arrow). Panel B is an ARPT of a normal squeeze maneuver. Contraction of the external sphincter and puborectalis is seen as shift to warmer

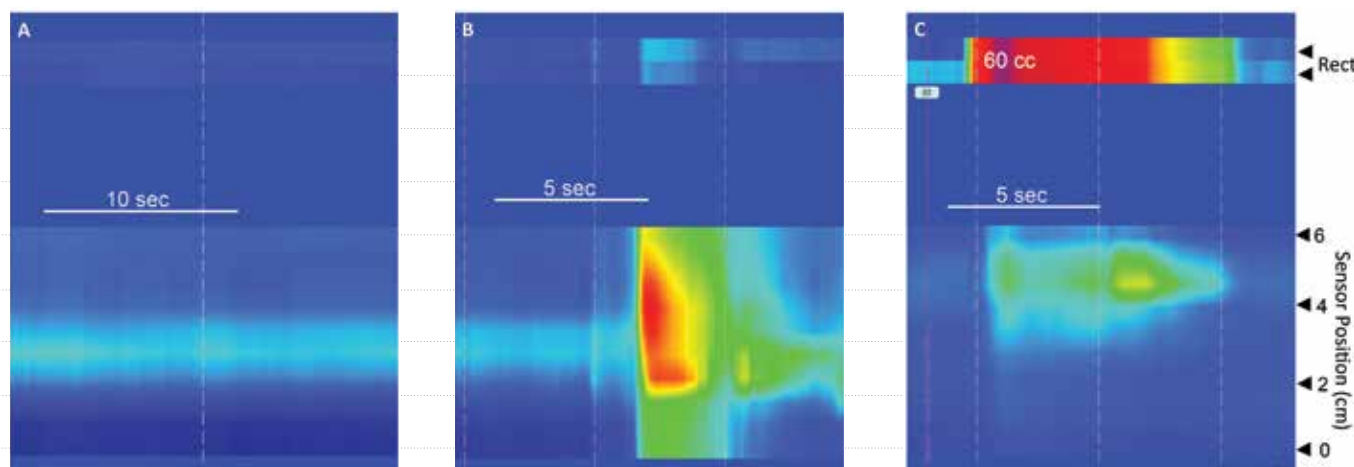


Figure 2 2A Scleroderma, hypotensive anal sphincter

2B Scleroderma, normal squeeze

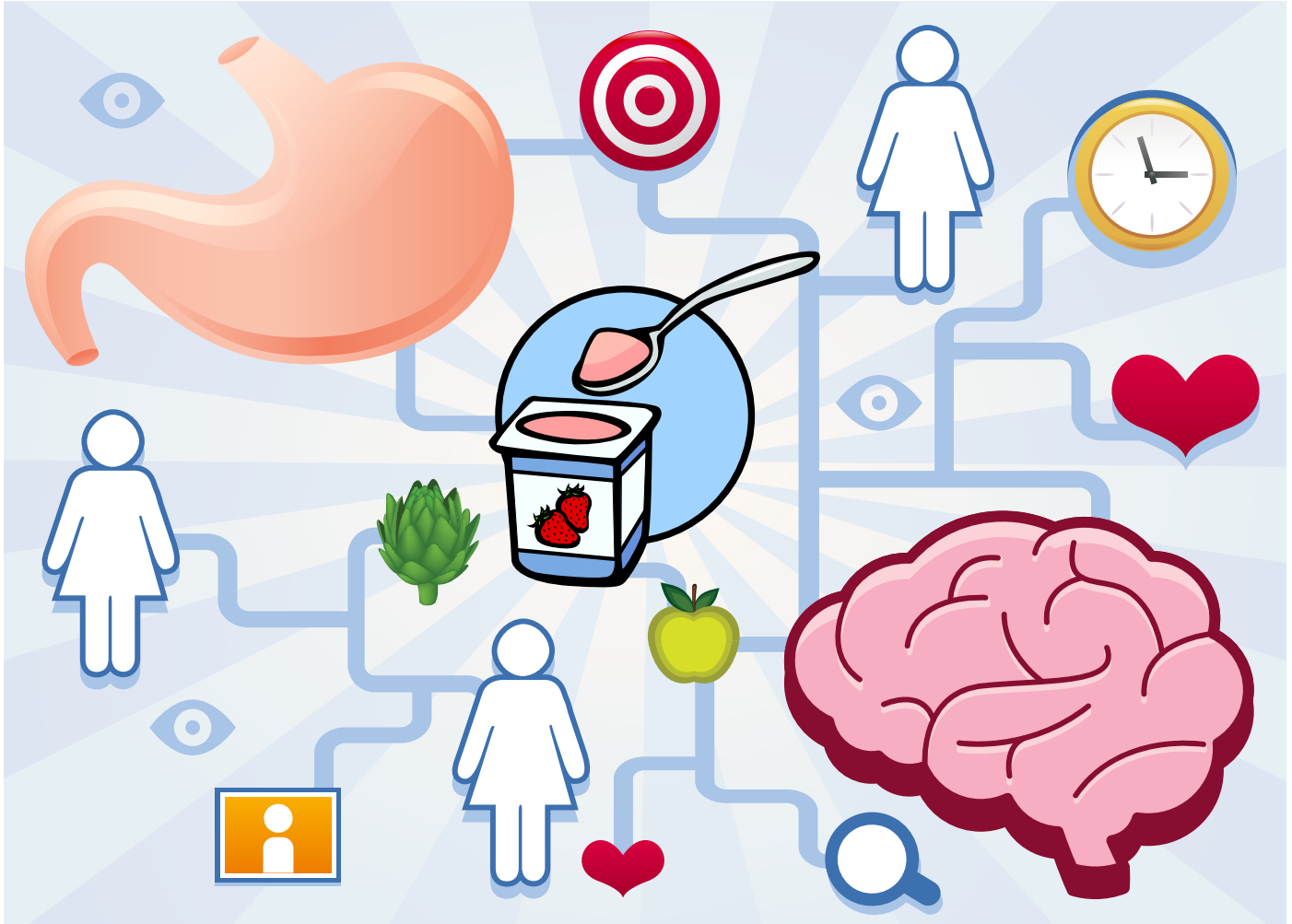
2C Scleroderma, failed RAIR

colors. Notice that there is little change in rectal pressure, indicating that there was no valsalva during the squeeze maneuver. Panel C is an ARPT of a normal rectoanal inhibitory reflex (RAIR). It is produced by inflation of the intrarectal balloon, in this case with 60 cc of air. Relaxation is seen as a change in color from red and yellow to green and blue. Most of the time, the RAIR appears to start on the rectal side of the anal canal and spread caudal as a function of balloon volume. This reflex is mediated at the level of the myenteric plexus, and it is the internal anal (smooth muscle) sphincter that relaxes.

Figure 2. Abnormal anorectal pressure topography in scleroderma. Panel A demonstrates a hypotensive anal sphincter at rest. In this case, the resting pressure is <20 mmHg. The majority of resting anal canal pressure is produced by tonic contraction of the internal anal sphincter. Panel B demonstrates a normal squeeze maneuver, indicating intact neuromuscular function of the external anal sphincter and puborectalis muscle. Panel C demonstrates loss of the RAIR and paradoxical sphincter contraction in response to rectal balloon distention, which suggests a neuropathic process.

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Can What We Eat Affect How We Think?

Researchers in UCLA's Division of Digestive Diseases have reported the first evidence suggesting it can

Specifically, scientists in the division's Gail and Gerald Oppenheimer Family Center for Neurobiology of Stress found that healthy women who regularly consumed yogurt containing beneficial bacteria known as probiotics showed altered brain function, both while in a resting state and in response to an emotion-recognition task. The early proof-of-concept study, which was performed at the Ahmanson-Lovelace Brain Mapping Center at UCLA, was sponsored by Danone Research and published in the journal *Gastroenterology*.

The discovery that changing the bacterial environment, or microbiota, in the gut can affect the brain carries significant implications for future research that could point the way toward dietary or drug interventions to improve brain function.

"Many of us have a container of yogurt in our refrigerator that we may eat for enjoyment, for calcium or because we think it might help our health in other ways," says Kirsten Tillisch, MD, an associate professor of medicine in the

division and lead author of the study. "Our findings indicate that some of the contents of yogurt may actually change the way our brain responds to the environment. When we consider the implications of this work, the old sayings 'you are what you eat' and 'gut feelings' take on new meaning."

Researchers have known that the brain sends signals to the gut, which is why stress and other emotions can contribute to gastrointestinal symptoms. The study by

Dr. Tillisch's group shows what has been suspected but until now had been proved only in animal studies: that signals travel the opposite way as well. "Time and time again, we hear from patients that they never felt depressed or anxious until they started experiencing problems with their gut," Dr. Tillisch says. "Our study shows that the gut-brain connection is a two-way street."

The small study involved 36 women, ages 18 to 55, who were divided into three groups. One group ate a specific yogurt containing a mix of several probiotics – bacteria believed to have a positive effect on the intestines – twice a day for four weeks. A second group consumed a dairy product that looked and tasted like the yogurt, but contained no probiotics. The third group ate no product.

Functional magnetic resonance imaging (fMRI) scans, conducted both before and after the four-week study period, looked at the women's brains in a state of rest and in response to an emotion-recognition task in which they viewed a series of pictures of people with angry or frightened faces and matched them to other faces showing the same emotions. The task, designed to measure the engagement of affective and cognitive brain regions in response to a visual stimulus, was chosen because previous research in animals had linked changes in gut flora to changes in affective behaviors.

The researchers found that, compared with the women who didn't consume the probiotic yogurt, those who did showed a decrease in activity in both the insula – which processes and integrates internal body sensations, like those from the gut – and the somatosensory cortex during the emotional reactivity task. In response to the task, these women also had a decrease in the engagement of a widespread network in the brain that includes emotion-, cognition- and sensory-related areas. The women in the other two groups showed a stable or increased activity in this network.

During the resting brain scan, the women consuming probiotics showed greater connectivity between a key brainstem region known as the periaqueductal grey and cognition-associated areas of the prefrontal

cortex. The women who ate no product at all, on the other hand, showed greater connectivity of the periaqueductal grey to emotion- and sensation-related regions, while the group consuming the non-probiotic dairy product showed results in between. Dr. Tillisch says she and her colleagues were surprised to find that the brain effects could be seen in many areas, including those involved in sensory processing and not merely those associated with emotion.

The knowledge that signals are sent from the intestine to the brain and that they can be modulated by a dietary change is likely to lead to an expansion of research aimed at finding new strategies to prevent or treat digestive, mental and neurological disorders, says Emeran Mayer, MD, PhD, a professor in the Division of Digestive Diseases, as well as of physiology and psychiatry, and the study's senior author.

"There are studies showing that what we eat can alter the composition and products of the gut flora – in particular, that people with high-vegetable, fiber-based diets have a different composition of their microbiota, or gut environment, than people who eat the more typical Western diet that is high in fat and carbohydrates," Dr. Mayer says. "Now we know that this has an effect not only on the metabolism, but also affects brain function."

The researchers are now seeking to pinpoint particular chemicals produced by gut bacteria that may be triggering the signals to the brain. They also plan to study whether people with gastrointestinal symptoms such as bloating, abdominal pain and altered bowel movements have improvements in their digestive symptoms that correlate with changes in brain response after consuming probiotics. Dr. Mayer notes that other researchers are studying the potential benefits of certain probiotics in yogurts on mood symptoms such as anxiety – and that other nutritional strategies may also be found to be beneficial.

The discovery that changing the bacterial environment, or microbiota, in the gut can affect the brain carries significant implications for future research that could point the way toward dietary or drug interventions to improve brain function.



*Kirsten Tillisch, MD
Associate Professor of
Medicine*



*Emeran A. Mayer, MD, PhD
Director, Gail and Gerald
Oppenheimer Family Center
for Neurobiology of Stress
Professor of Medicine,
Physiology and Psychiatry*

Study Suggests Anti-Cancer Properties for Diabetes Drug

Metformin Appears More Effective than Other mTOR Inhibitors in Pancreatic Cancer Model



*J. Enrique Rozengurt, DVM, PhD
Chief of Research, Division of
Digestive Diseases
Director, CURE Digestive
Diseases Research Center
Distinguished Professor*

A new study by a UCLA Division of Digestive Diseases and CURE: Digestive Diseases Research Center team headed by Dr. Enrique Rozengurt provides further evidence that metformin, a front-line drug for the treatment of type 2 diabetes, has anti-cancer properties.

It's well recognized that normal cells become cancerous through a series of steps resulting in the continuous activity of a signaling pathway that stimulates their multiplication and dissemination to other sites. Researchers have focused on identifying drugs that target these dysregulated pathways in an effort to restore normalcy, halting the proliferation of cancer cells. One pathway that has become the subject of growing interest because of its central role in the development of many cancers is mammalian target of rapamycin (mTOR).

Recently there has been a concerted effort in the pharmaceutical industry to move mTOR inhibitors – first rapamycin, and now a second generation of drugs – to the clinic as a potential treatment for certain cancers. But these compounds, while successful at inhibiting the mTOR pathway, have shown limited therapeutic benefits. Rapamycin was found to be an incomplete inhibitor of the pathway. The second-generation inhibitors are more effective in blocking the pathway, but they have their own shortcomings. The reasons for these limitations were explored in a paper by Dr. Rozengurt's group that appears in the journal PLoS ONE.

Working with pancreatic cancer cells, Dr. Rozengurt and colleagues followed up on previous work showing that the new mTOR-inhibiting drugs turn on other pathways involved in cancer, counterbalancing the inhibitory effects. "mTOR drives cell proliferation through insulin and other growth factors, but it also has a regulatory feedback mechanism," explains Dr. Rozengurt, professor of medicine in the Division of Digestive Diseases, director of the CURE: Digestive Diseases Research Center and Hirshberg Professor in Pancreatic Cancer Research. "In addition to sending stimulatory signals to the cell, it moderates the intensity of the growth factors. As a result, by suppressing the growth-promoting effects, you also suppress these moderating effects, which has the unintended consequence of overactivating pathways upstream of mTOR."

Dr. Rozengurt's group found that mTOR inhibitors activate a pathway called MEK/ERK that is linked to Ras – the most commonly activated oncogene in pancreatic cancer. "We know there are very good inhibitors for the MEK/ERK pathway," says Dr. Rozengurt. "What this means is that the future may lie in rational new combinations of drugs to inhibit mTOR and MEK/ERK."

The researchers also examined the impact of an existing drug, metformin, on the MEK/ERK pathway. Metformin is the most common drug used in the control of type 2 diabetes. A series of recent reports, including a set of papers from Dr. Rozengurt's team, indicates that the drug also exerts anti-cancer effects. This is believed to be at least partly due to its ability to efficiently block mTOR, albeit through different mechanisms than rapamycin and the second-generation mTOR inhibitors. "The question became, what are the consequences of metformin's blocking of mTOR – whether it turns on the same feedback loops and shows the same patterns as the other inhibitors," explains Dr. Rozengurt. What his team found was significant: Metformin not only inhibits mTOR in pancreatic cancer cells, but it also suppresses the MEK/ERK pathway – just the opposite of the other mTOR inhibitors.

Pancreatic cancer is one of the most devastating cancers. It is the fourth-leading cause of cancer mortality in the developed world, with a life expectancy of 5-8 months in diagnosis.

Could metformin, when used for patients with type 2 diabetes, be providing the unintended therapeutic benefit of reducing the risk of pancreatic cancer? Given the epidemic of type 2 diabetes in the United States, millions of people have used the drug, providing fertile ground to mine epidemiological data in search of the answer. Sure enough, several groups from different institutions found that people with type 2 diabetes who were treated with metformin showed a reduced susceptibility to pancreatic cancer.

Paired with the laboratory findings of Dr. Rozengurt's group, and consistent with other studies in the field this observational evidence paves the way toward clinical studies, some of them already underway to determine the potential efficacy of metformin, perhaps in combination with other drugs, in pancreatic cancer patients. "If our new findings with cells in culture can be extended to cancer cells in tumors, the results could explain important aspects of the anti-cancer activity of different drugs that target mTOR – and, most excitingly, might suggest the use of rational drug combinations to combat pancreatic cancer in the future," Dr. Rozengurt says.

Pancreatic cancer is one of the most devastating cancers. It is the fourth-leading cause of cancer mortality in the developed world, with a life expectancy of 5-8 months from diagnosis. Dr. Rozengurt believes his group's findings together with findings from other researchers will add to the growing interest in studying metformin's potential as a chemopreventive agent in pancreatic and other cancers (the MEK/ERK pathway has been implicated as a key pathway in a number of cancers).

"There have been many efforts made to bring mTOR inhibitors to cancer patients, but they have been less than successful because of an unintended consequence — they over-activate other pathways," Dr. Rozengurt concludes. "Now we have this drug, metformin, that has been used in millions of people with type 2 diabetes. It's an old drug that we know a lot about, so if it turns out to be effective in cancer, that will be extremely exciting."

Accreditation

The Office of Continuing medical Education, David Geffen School of Medicine at UCLA is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

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The 2nd Annual UCLA-Mellinkoff Gastroenterology Symposium

will focus on novel medical, endoscopic and surgical therapeutics for a wide array of gastrointestinal and hepatic disorders. Distinguished faculty will share their specialized expertise through formal lecture, Q & A panel discussion and hands-on endoscopy opportunities. The hands-on sessions provide a valuable learning opportunity, though no CME credits will be issued for participants. This agenda has been specifically designed to offer practical approaches and solutions that healthcare professionals may readily integrate into their daily practice.

Overnight Accommodations

A limited block of rooms, at a special rate of \$249 (deluxe) or \$309 (studio suite) + tax, has been reserved at the Beverly Hilton. This special room block **expires February 14, 2014**. Call the reservation line at 1-800-Hiltons (1-800-445-8667) and ask for the "UCLA GI" block.

For more information about the hotel, visit www.beverlyhilton.com



Agenda

Friday, March 7

Welcome and Introduction

Gary Gitnick, MD, UCLA

Course Overview

V. Raman Muthusamy, MD, UCLA & Bennett Roth, MD, UCLA

Esophageal Disorders

Moderator: Bennett Roth, MD, UCLA

New Options for Treating GERD - Beyond PPIs

David C. Chen, MD, UCLA

Barrett's Esophagus – Who, When and How to Treat

David Fleischer, MD, Mayo Clinic, Arizona

Achalasia – Treatment Options: Dilation/ Surgery/Endoscopic?

Jeffrey L. Conklin, MD, UCLA

Eosinophilic Esophagitis (EoE) – Drugs/Diet/Dilate?

Kevin Ghassemi, MD, UCLA

Case Presentations / Q & A

Colonic Disorders

Moderator: Rabindra R. Watson, MD, UCLA

Management of the Difficult Polyp

G.S. Raju, MD, University of Texas, Anderson Cancer Center

Managing Complications of Colonoscopy

G.S. Raju, MD, University of Texas, Anderson Cancer Center

Functional Disorders of the Ano-rectum – Incontinence and Pelvic Floor Dysfunction

Christopher Tarnay, MD, UCLA

Common Ano-rectal Problems: What the Gastroenterologist Should Know

Anne Lin, MD, MSHS, UCLA

Case Presentations / Q & A

Lunch

Afternoon Session – Track 1

Moderator: Thomas Kovacs, MD, UCLA

Case Presentations / Video – GI Bleeding

Dennis M. Jensen, MD, UCLA

Lumps and Bumps Along the Way: GISTS

Fritz C. Eilber, MD, UCLA

Neuro-endocrine Tumors

Joseph R. Pisegna, MD, UCLA

Afternoon Session – Track 2

Non-CME Hands-on Session*

Adjourn

* Hands-on Session Faculty

David Fleischer, MD, Mayo Clinic, AZ
Karoush Ghassemi, MD, UCLA
Chris Hamerski, MD
California Pacific Medical Center

Saturday, March 8

Welcoming Remarks

Eric Esrailian, MD, MPH, UCLA

Pancreatico-biliary Disorders

Moderator: V. Raman Muthusamy, MD, UCLA

Chronic Pancreatitis – Managing Complications

Alireza Sedarat, MD, UCLA

Pancreatic Fluid Collections – Acute/Chronic, All Shapes and Sizes

Rabindra R. Watson, MD, UCLA

Acute and Chronic Pancreatitis – The Surgeon's Perspective

O. Joe Hines, MD, UCLA

Getting into the Inaccessible Duct

Janak N. Shah, MD, California Pacific Medical Center

Case Presentations / Q & A

Inflammatory Bowel Disease

Moderator: Daniel Hommes, MD, PhD, UCLA

New Therapeutic Approaches – Drugs/Worms/Transplant

William J. Sanborn, MD, UC San Diego

Monitoring and Optimization of Drug Therapy

Christina Ha, MD, UCLA

Dealing with the Pouch Problem

Jennifer M. Choi, MD, UCLA

Treating IBD - A Surgeon's Perspective

Jonathan Sack, MD, UCLA

Case Presentations / Q & A

Lunch

Afternoon Session – Track 1

Moderator: Lynn S. Connolly, MD, MSCR, UCLA

Challenging Cases – Patients with Functional GI Disease

Chronic Abdominal Pain, Disordered Bowel Function, Dyspepsia, Chronic Nausea/Vomiting, Gastroparesis, Cyclic Vomiting Syndrome

Panel Members:

Lin Chang, MD, UCLA

Emeran A. Mayer, MD, PhD, UCLA

Kirsten Tillisch, MD, UCLA

Afternoon Session – Track 2

Non-CME Hands-on Session*

Adjourn

V. Raman Muthusamy, MD, UCLA
G. S. Raju, MD, University of Texas
Anderson Cancer Center
Bennett Roth, MD, UCLA

Sunday, March 9

Welcoming Remarks

Bennett Roth, MD, UCLA

Liver Disease

Moderator: Simon W. Beaven, MD, PhD, UCLA

Treatment of Hepatitis B

Myron Tong, MD, PhD, UCLA

Treatment of Hepatitis C

Sammy Saab, MD, MPH, UCLA

Treating Complications of Cirrhosis

Steven-Huy Han, MD, MPH, UCLA

Treatment of Hepatic Neoplasia

Francisco Durazo, MD, UCLA

Case Presentations / Q & A

Joining Panel: Mohamed El Kabany, MD, UCLA

Bugs and GI Diseases – Cause or Cure?

Moderator: Mary Farid, DO, UCLA

Treatment Paradigms for Resistant H. Pylori

Daniel Z. Uslan, MD, UCLA

Bacterial Overgrowth and IBS: Causation, Association or Neither?

Brennan M.R. Spiegel, MD, MSHS, UCLA

Fecal Microbial Transplant – Role in GI Disease

Eric Esrailian, MD, MPH, UCLA

Probiotics in GI Disease – What the Gastroenterologist Needs to Know

Kirsten Tillisch, MD, UCLA

Case Presentations / Q & A

Adjourn – Boxed Lunch

Janak N. Shah, MD, California Pacific
Medical Center
Alireza Sedarat, MD, UCLA
Rabindra R. Watson, MD, UCLA

UCLA Division of Digestive Diseases Welcomes Three New Faculty Members

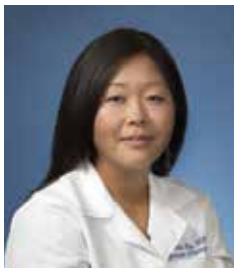


Michael J. Albertson, MD
*Assistant Clinical Professor
of Medicine*

Michael J. Albertson, MD | Assistant Clinical Professor of Medicine

Dr. Michael J. Albertson is a clinical gastroenterologist with extensive experience in endoscopic retrograde cholangiopancreatography and biliary tract intervention whose expertise also includes inflammatory bowel disease and hepatology.

Board-certified in internal medicine and gastroenterology, Dr. Albertson earned his medical degree at the University of Southern California's Keck School of Medicine, where he also did his internship in medicine, residency in internal medicine, and a fellowship in gastroenterology at Los Angeles County+USC Medical Center. He then returned to Keck School of Medicine as director of student health and clinical instructor of gastroenterology. Dr. Albertson's medical career also includes stints as a gastroenterologist at Southern California Digestive Disease Consultants and Valley Gastroenterology Associates, both in Burbank, CA. Immediately prior to coming to UCLA, he served as assistant professor of medicine and gastroenterology at State University of New York (SUNY)/Downstate Medical School in Brooklyn.

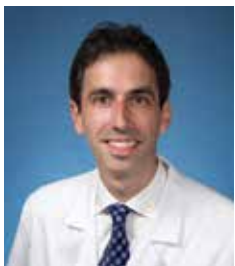


Christina Ha, MD
*Assistant Clinical Professor
of Medicine*

Christina Ha, MD | Assistant Clinical Professor of Medicine

Dr. Christina Ha focuses her clinical work on the inflammatory bowel diseases (IBDs) – Crohn's disease and ulcerative colitis. She also conducts research in these diseases, with a particular focus in the natural history and clinical outcomes of IBD in the elderly.

Dr. Ha graduated from Harvard University and earned her medical degree from Albert Einstein College of Medicine. She completed both her internal medicine residency and gastroenterology fellowship at Washington University in St. Louis School of Medicine. Following her GI fellowship, Dr. Ha spent a year as the Present-Levison Inflammatory Bowel Disease Fellow at the Mount Sinai School of Medicine in New York. Subsequently, she joined the faculty at The Johns Hopkins School of Medicine as part of the Meyerhoff Inflammatory Bowel Disease Center, where she was co-associate director of IBD clinical research and associate GI fellowship director. She is board-certified in internal medicine and gastroenterology.



Jeffrey R. Lewis, MD
Clinical Instructor of Medicine

Jeffrey R. Lewis, MD | Clinical Instructor of Medicine

Dr. Jeffrey R. Lewis begins his UCLA career as a health sciences clinical instructor of medicine in the Division. His research includes studies on capsule endoscopy in healthy individuals and on the use of high-resolution manometry to identify predictors of favorable outcomes after fundoplication for refractory gastroesophageal reflux disease (GERD).

Dr. Lewis previously served as attending physician in the Department of Medicine at the Icahn School of Medicine at Mount Sinai – New York City. He earned his medical degree from the University of Chicago Pritzker School of Medicine, completed his residency in internal medicine at the University of Chicago Medical Center, and, following his residency, served as chief fellow in the Division of Gastroenterology at the Icahn School of Medicine at Mount Sinai – New York City. Dr. Lewis has received multiple honors, including election to the national medical honor society Alpha Omega Alpha.

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The UCLA Division of Digestive Diseases continues to be rated in the top ten among digestive diseases centers in the United States by *U.S. News & World Report* in its annual survey.

UCLA DIVISION OF DIGESTIVE DISEASES

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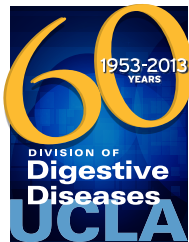
to learn more about the UCLA Division of Digestive Diseases.

UCLA Physician Referral Service **1-800-UCLA-MD1 (800-825-2631)**

Digestive Disease Call Center **(310) 825-1597**



U.S. News & World Report's Best Hospital Survey ranks UCLA as the No. 5 hospital and UCLA Digestive Diseases as No. 8 in the country.



UCLA Division of Digestive Diseases celebrates 60 years of education, research and innovation.