Prenatal Precautions

Thyroid dysfunction among pregnant women can often result in additional obstetric complications. A new study from The Journal of Clinical Endocrinology & Metabolism shows that thyroid screening could help identify women who might benefit from additional prenatal care.

ENDO 2019 PREVIEW:
Targeting cellular senescence in age-related osteoporosis

MIND THE GAP:
Varsha Vimalananda, MD, MPH, discusses a career in health services research
Get the latest recommendations on how to promptly diagnose, treat, and provide ongoing care for patients with Congenital Adrenal Hyperplasia (CAH) due to steroid 21-hydroxylase deficiency, an inherited endocrine disorder.

**Recommendation Highlights:**

- All newborn screening programs should incorporate screening for CAH.
- Healthcare professionals should inform all parents of pediatric patients with CAH about surgical options.
- Shared decision making among CAH patients, their families, and healthcare professionals should be applied when it comes to the medical, surgical, and psychological treatment of minors.

**READ THE GUIDELINE AT ENDOCRINE.ORG/CPG**
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MIND THE GAP
Frustrated by the gaps she saw between theoretical and actual patient outcomes, Varsha Vimalananda, MD, MPH, decided to pursue a career in health services research, which looks at quality, access, and costs of healthcare, and may even help endocrinologists lead the way for measuring and improving how endocrine care is delivered.

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Publishing peer-reviewed journals is an integral part of the Endocrine Society’s mission to unite, lead, and grow the global community of researchers, educators, and practitioners in endocrinology. It is an endeavor that can only be accomplished when that same community vigorously participates in the Society’s journals as authors, editors, reviewers, and readers – again, on an inclusive, global scale.

I am pleased to highlight just some of the major initiatives that are underway with our journals.

First, we have recently initiated a multi-year plan to revitalize our journals’ volunteer editorial boards — who serve as our primary reviewers — to be more reflective of the Society’s diverse membership, encompassing gender and ethnicity and geographic distribution. The changes will allow us to provide board members with a more focused, personalized experience, improving their ability to learn from the Society and from each other about editorial and publishing trends, and to serve as ambassadors for the journals and the Society. Look for changes in the rosters of our 2019 and 2020 boards.

Second, in mid-2018, Endocrinology began soliciting nominations for early career reviewers (ECRs) to serve on its newly created Early Career Review Board. ECRs could self-nominate or be nominated by an individual who has reviewed for Endocrinology. ECRs must hold a doctoral degree and have published at least three papers, one of which must be a first or co-first author paper. ECRs form a subset of the main reviewer pool and receive mentoring from the journal’s editor-in-chief and associate editors in the manuscript review process. Reviews by ECRs constitute a third review for a manuscript and may be appended to authors’ notifications at the discretion of the associate editor overseeing the reviews. Through this process, ECRs learn how to write good, fair reviews. ECRs who provide reviews will be recognized annually in the journal. The first group of 30 ECRs has been selected for 2019. If this trial is successful, we plan to expand it to our other journals. And all reviewers, early career or not, can benefit from the advice we provide in our online Reviewers Resource Center.

Third, we continue to expand our efforts to publish the very best articles in our journals, across all topic areas. We utilize professional networking, conference attendance, literature research, and a variety of online metrics — including citations, Altmetric scores, h index, and preprints — to identify authors and key topics around the world, then we follow up with personal contacts to authors to encourage and invite submissions of research articles, reviews, commentaries, and other article types. Each month, we publish online a thematic issue of top articles on a given topic drawn from across all our journals.
Our online Author Resource Center provides authors with information, services, and advice, including language editing and graphics preparation services, discount programs for submissions of preprints and transfers to *Journal of the Endocrine Society*, free access links for published articles, primers on Altmetric and ORCiD, and tips on using search engine optimization tricks and social media to attract attention and citations.

We encourage all members to follow and re-tweet our journals' Twitter account, [@EndoSocJournals](https://twitter.com/EndoSocJournals), as it is our primary online communication channel for journal developments and new content releases.

If you have any comments regarding your experience publishing in one of our journals, or if you wish to suggest new topics, please forward them to the attention of our Director of Publishing Operations at [publications@endocrine.org](mailto:publications@endocrine.org). Any feedback on our current practices and services, or recommendations for others, is greatly appreciated. What’s next for our journals? Stay tuned for announcements coming soon on graphical abstracts and podcasts!

This is just a snapshot of the tremendous amount of work that our members and staff have been doing over the past year. I would like to thank all the other committees, working groups, and task forces that I did not mention but who are working very hard on numerous projects and initiatives to improve endocrinology worldwide. From the bottom of my heart, thank you. 🌟

— Susan J. Mandel, MD, MPH, President, Endocrine Society

"Publishing peer-reviewed journals is an integral part of the Endocrine Society’s mission to unite, lead, and grow the global community of researchers, educators, and practitioners in endocrinology."

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— Susan J. Mandel, MD, MPH, President, Endocrine Society

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Roger Rittmater, MD
Lauding Endocrine Society Members

As has been tradition for the last several years, the January Endocrine News highlights the latest class of Endocrine Society Laureate Award winners and each year is as impressive as the last. For those of us here at the magazine it lauds some of the members we’ve worked with on various articles through the years as well as introduces us to new subjects who often find their way onto the pages of Endocrine News.

This year we decided to ramp up the coverage from previous years by giving each Laureate his or her own page (page 28), as well as highlighting the distinguished members who have authored the citations for these awards.

Recognizing these living legends gives us a look at the dedication of those who have devoted their professional lives to furthering the practice and science of endocrinology.

The 2019 Laureates are the very essence of what makes the Endocrine Society so vital to furthering the field both in the U.S. and abroad, and this year’s recipients certainly represent an outstanding variety of professionals who are all proudly striving to create a better world to improve human health. Each of these Laureates — past and present — has

“Recognizing these living legends gives us a look at the dedication of those who have devoted their professional lives to furthering the practice and science of endocrinology.”

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secured a place in the history of endocrinology whether as a scientist, clinician, mentor, or educator.

The Laureates will be recognized at ENDO 2019 in New Orleans in March so if you happen to see them be sure to congratulate them on their accomplishments!

Another one of the Society’s members is highlighted in this issue on page 56 when we talk with Leonardo Trasande, MD, MPP, from NYU’s School of Medicine, about his new book, Sicker, Fatter, Poorer: The Urgent Threat of Hormone-Disrupting Chemicals to Our Health and Future… And What We Can Do About It. Trasande’s book is aimed at the lay audience because, as he tells Endocrine News, that although awareness has increased in recent years, knowledge is limited among the public, policy makers, and many clinicians alike and he hopes this will inspire people to take action. “The ban on BPA in sippy cups and baby bottles occurred not because the FDA wanted to do the right thing,” he says, “but because consumers cried out to companies asking whether BPA was in these products.”

Senior editor Derek Bagley got the chance to speak to Sundeep Khosla, MD, from the Mayo Clinic in Rochester, Minn., about his ENDO 2019 plenary on treating osteoporosis via cellular senescence in “Aging Gracefully” on page 50. His chief concept is that aging is a risk factor for a variety of conditions so that is what should be targeted: “So is it possible, instead of targeting each disease separately, can we in fact target a common aging mechanism to maybe try to treat multiple aging diseases?” This will definitely be a lively discussion in New Orleans that you will not want to miss! Register for ENDO 2019 today: endocrine.org/endo2019/register.

— Mark A. Newman, Editor, Endocrine News

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LETTER TO THE EDITOR

TO THE EDITOR:

In the November issue of Endocrine News, an article on the high cost of insulin affirmed that the cost had tripled between 2002 and 2013 and doubled again since 2012 (1). In the article it was stated that analogue insulins were “better” and that “we don’t have to rehash old data.” Perhaps we should. There have been at least 26 head-to-head studies involving 9,509 patients comparing rapid-acting analogues with regular insulin (2). Effectiveness was similar with only a 0.05% HbA1c level difference. Similarly, there have been at least 26 head to head studies involving 10,512 patients comparing basal analogues with bedtime NPH insulin (2). Again, there was only a 0.04% difference in HbA1c levels. Regarding hypoglycemia, there was no difference in those receiving the rapid-acting analogues versus regular insulin. There was a slight, but statistically significant, difference in overnight hypoglycemia favoring the basal analogues. However, in none of these studies was a small bedtime snack included which some of us recommend to protect against overnight hypoglycemia.

Given that 25% of insulin-requiring patients have cut back on their use of insulin due to its cost (1), we need to seriously consider using the cheaper human insulins. Relion insulins (NPH and regular) can be purchased at Walmart for $25 a vial. Rehashing the old data could lead to all of our patients receiving their full recommended insulin doses. 

Mayer B. Davidson, Professor of Medicine
Charles R. Drew University & David Geffen School of Medicine at UCLA, Los Angeles, California

1. Seaborg, E. Paying the price: coping with the high cost of insulin. Endocrine News, November, 2018, pp 41-44.
Patients, Mentors, and Scientific Innovation

BY KATJA KISELJAK-VASSILIADES, DO, University of Colorado, Denver

There are certain things that shape our lives in the most unexpected ways. Although my initial interest was in oncology, a colleague from medical school sparked my curiosity in endocrinology through his own research on the interaction between the serotonergic system and neuroactive steroids. While I started to grasp the major concepts of medicine, I became intrigued with the complexity of endocrine systems and the crucial role they play in almost every aspect of human body function.

While endocrinology, as an intriguing science, drew my interest since the early days of my medical training, it was the people who I encountered during those years who heightened my fascination with this discipline and strengthened my decision to specialize in this field.

My first interaction with clinical endocrinology was through an elective rotation with Dr. George Lawrence in Baltimore. Despite the heavy clinical load, Dr. Lawrence’s thoughtfulness and comprehensive approach to each patient with endocrine disorders was impressive. Being from the era that pre-dated Up-To-Date and smart phone apps, Dr. Lawrence was an endocrine pathophysiology master with pencil and paper. I was fascinated with the hypothalamic-pituitary axis, negative feedback loops, nuclear steroid receptor action, endocrine-associated autoimmunity, and the critical thinking involved in developing an endocrine-related diagnosis.

As an internal medicine resident, I spent a number of months rotating through endocrine clinics and consult services. During that time I had the privilege to learn from one of the most prominent experts in thyroid disease, Dr. David Cooper. “It is not your thyroid,” are Dr. Cooper’s famous words when faced with non-thyroidal illness, that I remember very well even today. Whether he was on internal medicine wards or in an outpatient endocrine clinic, Dr. Cooper always emphasized the importance of medical history and a thorough physical exam. As a truly superb and meticulous clinician, I have always been in awe of Dr. Cooper’s remarkable career as a scholar, educator, and mentor.

Although I spent almost my entire medical training towards a career as a clinician endocrinologist, the possibility and opportunity to advance the field and contribute to discoveries underlying endocrine pathophysiology were the main motivation that influenced my decision to pursue academic endocrinology.

If you would like to share your story with our readers around the world, contact Editor Mark A. Newman at mnewman@endocrine.org.
At the completion of medical school and through residency, I was also fortunate to cross paths with one of the leading investigators in the field of thyroid cancer, Dr. Mingzhao Xing. In Dr. Xing’s lab, for the first time, I encountered another exciting aspect of endocrinology – research. As an excellent mentor, Dr. Xing gave me a project that I was still able to complete as a busy resident, and met with me on a regular basis to discuss the hypothesis of the research project as well as our approach to investigate the molecular mechanisms and environmental factors that might underlie thyroid tumorigenesis.

It was these early medical training experiences and interactions with some of the most renowned endocrinologists that led me toward the endocrinology fellowship which I completed at the University of Colorado. Upon completion of my fellowship I found myself at a crossroads again, and probably one of the most exciting and yet difficult decisions I had to make in my medical career thus far. Although I spent almost my entire medical training towards a career as a clinician endocrinologist, the possibility and opportunity to advance the field and contribute to discoveries underlying endocrine pathophysiology were the main motivation that influenced my decision to pursue academic endocrinology.

However, it was also my fellowship mentor Dr. Maggie Wierman, who played a large part in my career choice. Dr. Wierman, initially my clinical mentor, is truly a model clinician-scientist, bridging the gap between the bench and bedside, incorporating cutting-edge scientific knowledge into the management and education of patients, residents, and fellows on all aspects of the disease pathology. I joined Dr. Wierman’s lab as an endocrine research fellow to explore mechanisms of endocrine neoplasia, initially pituitary tumorigenesis and then I expanded my interest to adrenal carcinogenesis. As a mentor, Dr. Wierman has been exceptional, guiding my training in molecular biology techniques, basic, and translational research design, and ensured that I stay focused on my career objectives as well as made sure to enable many networking and collaborative opportunities. I remember my first years of ENDO meetings walking the corridors of the convention centers along with Dr. Wierman where she would introduce me to the most prominent endocrinologists in their fields. By my fifth ENDO meeting she had introduced me four or five times to many of them, and by that time these leaders in endocrinology knew who I was too.

“
It is a challenging science that has undergone a remarkable evolution over the last several decades, and I feel privileged to have been given an opportunity to contribute to the progress of this intriguing science at the intersection of all essential biological functions.
”

I consider myself fortunate to have the opportunity to follow my dream of becoming an endocrinologist and a physician-scientist. My path toward this profession has been inspired by my desire to understand the influence of hormones on the intricacies of the human body and it has been as equally guided by several outstanding leaders in this field. I only mentioned a few of them, but in reality I would have never been here without the positive interactions I encountered with many clinical endocrinologists and research mentors.

It is my belief that endocrinology, unlike other medical subspecialties, tightly knits many aspects of medicine. It is a challenging science that has undergone a remarkable evolution over the last several decades, and I feel privileged to have been given an opportunity to contribute to the progress of this intriguing science at the intersection of all essential biological functions.

EDITOR’S NOTE: The opinions and views of the author do not necessarily represent those of Endocrine News or the Endocrine Society.
Today’s Children Reach Bone Maturity Earlier, Study Reveals

Children born in the most recent century have bones that reach full maturity earlier – by nearly 10 months in girls and nearly seven months in boys, according to a study recently published Clinical Orthopaedics and Related Research.

Researchers led by Dana Duren PhD, director of orthopaedic research at the Thompson Laboratory for Regenerative Orthopaedics at the University of Missouri in Columbia, point out that the timing of epiphyseal fusion (EF) in the bones of the hand and wrist corresponds to late stages of skeletal maturation and signals the end of longitudinal growth. “As such,” the authors write, “assessments of EF status provide a simple means for insight into skeletal development status when evaluating children with disorders such as idiopathic scoliosis, leg length inequality, or constitutional growth delay.”

The authors continue: “We, therefore, sought to answer the following questions: (1) Do children today initiate the process of EF in the hand and wrist earlier than past generations on which maturity standards are based? (2) Do children today complete EF in the hand and wrist earlier than past generations on which maturity standards are based?”

The researchers assessed the radiographs of more than 1,000 children born between 1915 and 2006. The team evaluated radiographs of the bones in the hands and wrists to determine the precise timing of the beginning and ending of a developmental process called epiphyseal fusion.

“We focused on epiphyseal fusion because it signals the end of the growth of the bone,” says Duren. “It begins when the growth plate, which is cartilage at the end of the bone, starts to connect the epiphysis, or bone cap, to the long bone through small calcifications. Eventually, the growth plate completely calcifies and attaches, or fuses, to the long bone. When fusion is complete, so is the growth of that bone.”

The research team used radiographs gathered in the Fels Longitudinal Study, which is the world’s only century-long study of human growth and development, to track when fusion started and when it was complete in children born as far back as 1915. The results showed that the skeletons of children born in the 1990s are reaching fusion completion, and thus skeletal maturity, faster and sooner than children born in the 1930s.

These findings directly impact the timing of the clinical care of certain pediatric orthopaedic conditions, such as leg-length differences, scoliosis and the timing of using growth hormone.

“Our findings show there is a ‘new normal’ for timing when kids’ skeletons will reach full maturity,” says Duren.

Mel Boeyer, MS, predoctoral orthopaedic research fellow and co-author of the study, works closely with pediatric orthopaedic surgeons to understand how physicians time this care.

“The timing for the treatments of these conditions is a critical component to a good outcome,” Boeyer says. “What this research shows us is physicians will need to start looking for the beginning of epiphyseal fusion sooner than they once thought.”

The study does not address what might be the cause of this new normal. However, Duren and many of her colleagues think an increase in exposure to environmental hormones and hormone mimickers could be a contributing factor.

Findings: Based on their findings, the authors conclude: “In general, EF [initiation] traits exhibited greater changes in age than did EF [complete] traits with the magnitude of the observed trends appearing to be larger in females than in males. As the contemporary population shifts the timing of maturational milestones, expectations regarding the average age of occurrence for a maturational milestone, including EF, must shift as well.”
Hysterectomy can impair some types of memory in the short term following the surgery, according to a rat study published in *Endocrinology*.

Researchers led by Heather Bimonte-Nelson, PhD, Arizona State University in Tempe, Ariz. Point out that one in three women in the U.S. undergo a hysterectomy by age 60, but few studies have looked at hysterectomy in a preclinical animal model and the cognitive effects of hysterectomy with and without ovarian conservation have not been fully explored in a systematic experimental context. “Given the large number of women who undergo variations in gynecological surgeries, it is essential to understand how ovarian morphology and function may be altered following hysterectomy, as well as to elucidate how hysterectomy with and without ovarian preservation relates to the trajectory of brain aging and cognitive decline,” the authors write.

A hysterectomy can cause some women to experience menopause, or the process a woman goes through that causes her monthly periods to end, earlier than they would have otherwise. The current view in the medical field is that the uterus has no function when it’s not in the pregnant state. “Specifically, we assessed serum hormone levels of ovarian-derived hormones and the gonadotropins and ovarian follicle morphology, as well as monitored estrous cyclicity and body weight changes, to gain a comprehensive understanding of the far-reaching impact that variations in gynecological surgeries have on the body’s reproductive anatomy, physiology, and function, and how these factors ultimately may lead to cognitive changes,” the authors write.

This study is the first of its kind to link the uterus to brain function by using a rat model of hysterectomy to show its effect on cognitive abilities, including memory and thinking skills.

“Our novel findings indicate that the nonpregnant uterus is not dormant and is in fact linked to brain function,” says the study’s senior author, “We studied several different menopause surgeries and found that a hysterectomy may impair short-term memory in rats.”

Researchers developed a rat model of hysterectomy to assess the effects of surgically removing the uterus on brain function. They found that surgical removal of the uterus alone can impair some types of memory in the short term, two months after surgery. Removing the ovaries alongside the uterus did not result in a memory impairment, indicating a unique negative effect of hysterectomy on memory, and suggesting that the uterus and ovaries are part of a system which communicates with the brain for functions such as cognition.

Based on their findings, the authors conclude: “Translationally, these findings are impactful in that they can inform clinical understandings of, and lead to additional human studies testing, the intricate connections between the brain and the female reproductive system. This will provide fundamental stepping stones to initiate further exploration into how common variations in gynecological surgery impact quality of life, as well as cognitive and brain aging, in women throughout their lifetimes.”

**Findings:** “We hope these basic science findings will lead to more attention around how different menopause surgeries might impact the brain and its functioning in women, ultimately impacting their quality of life,” Bimonte-Nelson says. “The overarching goal of our research is to promote and discover optimal health outcomes for women throughout their entire lifespan.”
55th Clinical Diabetes and Endocrinology Institute Annual CME Conference
Snowmass, Colorado, January 15 — 19, 2019
The 55th Clinical Diabetes and Endocrinology Institute Annual CME Conference will address gender-affirming hormone therapy, gestational diabetes, precision medicine for thyroid tumors, Cushing’s disease, neuroendocrine diseases, obesity therapies, the new ADA/EASD guidelines for type 2 diabetes management, menopause, diabetes technologies, and much more.
www.njhealth.org/diabetes-conference

ENDO 2019
With over 7,000 attendees, nearly 2,000 abstracts, and over 200 other sessions, ENDO 2019 is the leading global meeting for endocrinology research and clinical care. Join us for the most well attended and valued translational endocrinology meeting in the world. Bringing together leading experts, researchers, and the most respected clinicians in the field, ENDO 2019 represents a convergence of science and practice that highlights and facilitates breakthrough discoveries in the field of endocrinology. Spend time connecting with peers and colleagues, exchanging ideas and information, and getting out in front of the latest trends and advancements in hormone health. The meeting also hosts other satellite and pre-conference events.
www.endocrine.org/endo2019

55th Clinical Diabetes and Endocrinology Institute Annual CME Conference
Snowmass, Colorado, January 15 — 19, 2019
The 55th Clinical Diabetes and Endocrinology Institute Annual CME Conference will address gender-affirming hormone therapy, gestational diabetes, precision medicine for thyroid tumors, Cushing’s disease, neuroendocrine diseases, obesity therapies, the new ADA/EASD guidelines for type 2 diabetes management, menopause, diabetes technologies, and much more.
www.njhealth.org/diabetes-conference

MEN 2019: 16th International Workshop on Multiple Endocrine Neoplasia
Houston, Texas, March 26 — 29, 2019
In keeping with the spirit of the original MEN workshop, MEN2019 will focus on emerging topics in the genesis and therapy of malignant endocrine tumors associated with multiple endocrine neoplasia. The goal of the workshop will be to provide an outline for basic and clinical research focused on these malignant manifestations. The meeting will bring together local and international experts on multiple endocrine neoplasia to focus on these subjects. A significant portion of the meeting will be spent in workshops centered on emerging topics and the development of an international roadmap for future research and clinical trials, and the remainder of the meeting will be composed of large group didactic sessions.
https://www.mdanderson.org/conference

Endocrine Fellows Series: Type 1 Diabetes Care and Management
New Orleans, Louisiana, March 19—21, 2019
This comprehensive conference is for adult and pediatric endocrine fellows interested in type 1 diabetes. The unique and highly sought-after program is an opportunity to learn from leaders in the field through interactive sessions. The curriculum is specially designed to support early career endocrinologists by enhancing skills with comprehensive education not typically taught in
fellowships and providing the opportunity to connect with thought-leaders and peers. https://www.endocrine.org/T1Dfellsows

**International Pituitary Congress**  
**New Orleans, Louisiana,**  
**March 20 – 22, 2019**  
The Sixteenth International Pituitary Congress will present an exciting group of member and guest international experts in pituitary problems. It will include distinguished clinicians and clinical researchers, fellows in training, and experts in basic science. There will be cutting-edge in-depth topics that will permit each attendee to become familiar with the latest trends in pituitary endocrinology. The format of the meeting is intended to facilitate maximum interaction and free exchange of ideas among the participants and speakers. http://pituitarysociety.org

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**2019 Clinical Endocrinology Update**  
**Seattle, Washington**  
**September 17 – 19, 2019**  
Each year CEU brings together hundreds of endocrine clinicians for a unique learning experience and opportunities to network with expert faculty and colleagues. Attend the 71st CEU to receive the most trusted and clinically relevant information about recent advances in the field of endocrinology. The educational programming at CEU appeals to clinicians at all levels of practice, as well as fellows and other members of the clinical practice team. www.endocrine.org/ceu

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**1st BES-Mayo Course in Advanced Endocrinology 2019**  
**Dhaka, Dhaka, Bangladesh, January 24 – 25, 2019**  
The Advanced Course in Endocrinology is a collaboration between the Bangladesh Endocrine Society (BES) and the Mayo Clinic, Rochester, Minn. It is a two-day intensive and interactive learning course covering all aspects of clinical endocrinology. http://www.bes-mayo.com

**Keystone Symposia on Functional Neurocircuitry of Feeding and Feeding Disorders**  
**February 10—14, 2019, Alberta, Canada**  
The goal of this conference is to gather international leaders in the neural control of feeding and energy homeostasis, along with leaders in the pathophysiology of feeding and energy homeostasis. The conference will broaden the field by covering fundamental advances in the neural circuitry underlying feeding, while including entire sessions devoted to anorexia nervosa, disease cachexia, and feeding disorders across the lifespan. Additionally, while past Keystone Symposia conferences on the neuronal control of appetite have been heavily focused on the hypothalamic control of homeostatic feeding, this symposium will feature entire sessions devoted to brainstem and telencephalic control of feeding. http://www.keystonesymposia.org/19J8

**ATTD 2019**  
**Berlin, Germany, February 20 – 23, 2019**  

**World Peptide Congress**  
**April 17–18, 2019, Tokyo, Japan**  
The World Peptide Congress will bring together world-class biochemists, scientists, professors, and scholars to concentrate on “Accelerating Current Innovations in Peptide Research.” Peptides play important roles in living body systems by controlling, directing, and coordinating inter- and intra-cellular communications and cellular function and this conference will focus on the latest stimulating patterns and advancements in the field of peptide science. https://www.meetingsint.com/conferences/peptide

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Photo: Shutterstock.com
Thyroid dysfunction is fairly common among pregnant women and can often result in additional obstetric complications. A new study published in *The Journal of Clinical Endocrinology & Metabolism* shows that thyroid screening — despite some controversy as to its usefulness — could help identify women who might benefit from additional prenatal care.

BY KELLY HORVATH
Pregnancy places quite a physiologic demand on the thyroid gland, which resulting increases 40% in size and increases production of thyroxine (T₄) and triiodothyronine (T₃). It therefore stands to reason that the effects of any thyroid dysfunction will be amplified during pregnancy, and this can have negative implications for the long-term health of both the mother and child.

“Abnormalities in thyroid function are relatively prevalent among pregnant women and have been linked to several obstetric complications,” says Cuilin Zhang, MD, PhD, of the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) in Bethesda, Md. “In addition, given the important role that thyroid hormones play in glucose metabolism and homeostasis, thyroid dysfunction has been suggested to play a role in the etiology of gestational diabetes mellitus (GDM), a common metabolic complication in pregnancy.” Some previous studies have demonstrated increased incidence of GDM with varying levels of hypothyroidism and others with low levels of free T₄ (fT₄). Others found no significant association between thyroid hormone levels and development of GDM.
Finding the Likely Culprit

With existing evidence thus conflicting, and sparse longitudinal data formerly available, senior author Zhang and team fill the gap by focusing more closely on fT₃, which is the biologically active hormone involved in glucose homeostasis, in “A Longitudinal Study of Thyroid Markers Across Pregnancy and the Risk of Gestational Diabetes,” published in The Journal of Clinical Endocrinology & Metabolism. They reasoned that conducting a comprehensive analysis of the fT₃/fT₄ ratio, which signals the conversion of fT₄ to fT₃ and has been linked with insulin resistance and other mechanisms of diabetes, might help elucidate the pathogenesis of GDM. They also studied how thyroid markers shift over the course of pregnancy. Their longitudinal case-control study (for which Zhang is a co-principal investigator) tapped into the NICHD Fetal Growth Studies — Singleton Cohort (2009–2013), a multiracial and multicenter pregnancy cohort consisting of 2,334 non-obese and 468 obese women. Their subset comprised 107 GDM cases and 214 non-GDM controls, and eligibility criteria were age between 18 and 40 years, gestational status between eight and 13 weeks at enrollment, and a healthy state (i.e., no history of hypertension, diabetes, renal or autoimmune diseases, psychiatric disorder, cancer, HIV/AIDS, pregnancy complications like GDM or preeclampsia, or recent smoking). Using electrochemiluminescence immunoassay, the researchers measured fT₃, fT₄, and thyroid-stimulating hormone (TSH) at each of four visits to one of 12 clinics in the U.S.

The best/most optimal way of lowering high T₃ remains controversial, especially for pregnant women. As such, these women may need close monitoring of their glucose levels through pregnancy.”

— CUILIN ZHANG, MD, PHD, EUNICE KENNEDY SHRIVER NATIONAL INSTITUTE OF CHILD HEALTH AND HUMAN DEVELOPMENT, BETHESDA, MD.
The first visit was in the first trimester (between gestational weeks 10 to 14), but, because thyroid levels fluctuate during pregnancy, measurements were also taken during the second trimester (gestational weeks 15 to 26), and researchers differentiated first- and second-trimester analysis. Third and fourth blood samples were obtained between weeks 27–31 and weeks 33–39, respectively. Thyroid markers were analyzed as quartiles, and conditional logistic regression was used to establish initial and adjusted odds ratios between the cases and their matched controls.

Their findings showed that women with GDM weighed more prior to pregnancy and had higher family history rates of diabetes. After adjusting for these potential cofounders, the team found that their fT₃ levels were significantly lower, and, at the first two measurements, their fT₃ and fT₄;fT₃ significantly higher. Conversely, women with the highest levels of fT₄ during the second trimester were significantly less likely to develop GDM. TSH levels did not correlate with GDM risk at any measurement point. As for the researchers’ secondary study goal of mapping the trajectory of certain thyroid markers, they found that fT₃ and fT₄;fT₃ are higher in the setting of GDM, fT₄ is lower, and TSH is higher only during the last trimester. Normal function aligns with a decrease in TSH early in pregnancy accompanying the temporary rise in human chorionic gonadotropin, followed by a gradual increase in TSH levels as well as a progressive reduction in fT₃ and fT₄. Collectively, their results implicate high fT₃ and fT₄;fT₃ as independent risk factors for GDM and represent a novel way to monitor glucose homeostasis and assess GDM risk. Further support for this notion comes from the size and multiracial (white, black, Hispanic, and Asian/Pacific Islander) composition of the study sample.

To Screen or Not to Screen

“Findings from our longitudinal study suggest that higher fT₃ levels may be involved in the pathophysiology of GDM,” Zhang says. The team surmises that this elevation could be from an upsurge of conversion of fT₃ to fT₄ or de novo synthesis of fT₄ by the thyroid gland. What this means for clinicians is very important. “At present, the utility of routine screening for thyroid function during pregnancy is controversial,” Zhang explains. “This study adds an important piece of evidence to this debate, as our findings show that women with thyroid abnormalities in early to middle pregnancy are at an increased risk for GDM. Our findings, in conjunction with previous evidence of thyroid-related adverse pregnancy outcomes, support the potential benefits of thyroid screening among pregnant women.”

Screening, therefore, could identify women who may need additional care during pregnancy. “The best/most optimal way of lowering high T₃ remains controversial, especially for pregnant women. As such, these women may need close monitoring of their glucose levels through pregnancy,” Zhang says. Although lowering high T₃ with exogenous methods may not be currently recommended, making

**AT A GLANCE**

- Compared to euthyroid pregnant study participants, pregnant participants with abnormal, trimester-specific thyroid function, specifically, elevated levels of circulating triiodothyronine (fT₃), were at increased risk for gestational diabetes mellitus (GDM).

- Hypothyroxinemia (defined by 2017 American Thyroid Association [ATA] guidelines as normal thyroid-stimulating hormone [TSH] and low circulating thyroxine [fT₄] levels) in the second trimester of pregnancy was significantly associated with risk for developing GDM.

- Neither overt nor subclinical hypothyroidism (defined by ATA guidelines as high TSH and low or normal fT₄) was significantly associated with GDM risk, although the point estimate indicated an association with an increased risk. This is possibly attributable to a low statistical power of the current study, which had only 20 cases of hypothyroidism in the GDM group.
recommendations for regular exercise and healthy diet is warranted throughout pregnancy and should perhaps be emphasized for women with this marker.

Zhang reports that her team next plans to examine the impact of maternal thyroid function in pregnancy on fetal growth and development. “In addition, to further generalize findings from the present study, it is critical to conduct similar studies among pregnant women of different race/ethnicity groups,” she says. “More studies are needed to investigate health impacts of maternal thyroid function in pregnancy on fetal growth and offspring long-term health.”

— CUILIN ZHANG, MD, PHD, EUNICE KENNEDY SHRIVER NATIONAL INSTITUTE OF CHILD HEALTH AND HUMAN DEVELOPMENT, BETHESDA, MD.

— HORVATH IS A BALTIMORE, MD.-BASED FREELANCE WRITER AND A REGULAR CONTRIBUTOR TO ENDOCRINE NEWS. SHE WROTE ABOUT THE TOP ENDOCRINE DISCOVERIES OF 2018 IN THE DECEMBER ISSUE.
SUBMIT YOUR LAST CALL ABSTRACT

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Accepted abstracts will be published in a supplemental issue in the Journal of the Endocrine Society (JES).

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For more than 70 years, the Endocrine Society has recognized the achievements of endocrinologists worldwide. The Laureate Awards recognize endocrinologists for seminal research, meritorious service, leadership and mentorship, innovation, international contributions, education, translation of science to practice, and lifetime achievement.

Established in 1944, the Society’s Laureate Awards recognize the highest achievements in the endocrinology field, including groundbreaking research and innovations in clinical care.

The distinguished recipients on the following pages join a prestigious list of past award recipients, all of whom have advanced scientific breakthroughs, medical practice, and human health around the world. Award categories honor the achievements of endocrinologists at all stages of their careers, recognizing those at the pinnacle of the field as well as young endocrinologists who are making a mark.

The dedication, commitment, and achievements of current and past award recipients have earned each a place in Endocrine Society history as well as the history of the practice and science of endocrinology.

The Endocrine Society will present the awards to the winners at ENDO 2019, the Society’s 101st Annual Meeting & Expo, March 23-26, 2019 in New Orleans, La.
Wiebke Arlt is the William Withering Professor of Medicine at the University of Birmingham, UK, and an outstanding clinician scientist. Trained by Bruno Allolio, Walter Miller, and myself, she has made several seminal contributions to the field of endocrinology. Firstly, she identified the importance of dehydroepiandrosterone (DHEA) in women and in a landmark paper demonstrated the beneficial effects of DHEA replacement therapy in many women with primary and secondary adrenal insufficiency.

Secondly, utilizing novel human experiments of nature she described new forms of androgen excess arising from defects in critical co-factors for adrenal androgen secretion/metabolism. P450 oxidoreductase deficiency is now established as the second commonest form of congenital adrenal hyperplasia and confirms an alternative pathway of dihydrotestosterone secretion in human fetal life. Similarly, disruption of DHEA sulfation by mutations in the PAPS synthase 2 donor enzyme results in hyperandrogenism and a PCOS-like phenotype. Thirdly, working with a team of investigators in Birmingham and building on the successful European Adrenal Network (ENS@T), Wiebke has pioneered the concept of urinary steroid metabolomics that is likely to be fundamental in the clinical diagnostic work up of patients with adrenal disorders and tumors. The diagnostic pattern of abnormalities seen in adrenocortical carcinoma is striking; Wiebke's recent work has uncovered fascinating insight into the pluripotential nature of other adrenal adenomas such as Conn's tumors.

Over and above these major contributions, Wiebke has furthered our understanding of, and defined outcomes for, patients with a variety of adrenal pathologies. She is an excellent communicator and a living example of the importance of internationalization for our endocrine community and the Endocrine Society.
Edward Brown is one of the world’s leading biomedical scientists. His brilliant career exemplifies what all investigators aspire to, but rarely achieve. The elegance of his logic and the innovative approaches he applied in his quest to delineate calcium-induced parathyroid cell signaling, clone the calcium-sensing receptor (CaSR) cDNA, and translate this basic science into a fundamental understanding of human diseases and new diagnostic and therapeutic applications, are virtually unprecedented in a single investigator’s career.

The essence of Dr. Brown’s lifetime achievements is summarized in the following discoveries, each groundbreaking in its own right. He: (1) discovered the first eukaryotic CaSR encoding one of the first eukaryotic environmental sensors; (2) established it was the key molecule maintaining near constancy of the “milieu interieur”; (3) proved extracellular Ca\(^{2+}\) to be a versatile, ubiquitous, and pleiotropic first messenger, thereby creating the new field of Ca\(^{2+}\)-sensing. Moreover, he: (4) played a dominant role in identifying multiple inherited and acquired disorders of Ca\(^{2+}\)-sensing; (5) contributed to the experimental and conceptual foundation leading to the development of calcimimetics by others; (6) demonstrated that this receptor senses a variety of substances and integrates this information into modulating multiple cellular functions, thereby stimulating the development of the field of environmental sensing in higher organisms, such as GPCRs sensing fatty acids, protons, bile acids, zinc, etc.; and (7) collaborated with others to elucidate the crystal structure of the CaSR extracellular domain.

The step-by-step proof of Dr. Brown’s hypothesis that the CaSR existed, his perseverance in acquiring the scientific skills to clone its cDNA, and his studies identifying calcimimetics opened an entirely new field of scientific research. These discoveries, spanning four decades, significantly amplified our understanding of the physiology, pathophysiology, and therapy of endocrine disorders extending well beyond bone and mineral metabolism to other organ systems and across multiple species.
Kenneth Burman is the chief of endocrinology at Medstar Washington Hospital Center (MWHC) and the director of the Integrated Endocrine Training Program at Medstar Georgetown University/MWHC. He has educated more than 140 endocrinologists. He has a special passion for endocrinology. Passion drives the human spirit, which in turn results in the realization of our purpose in the education of physicians and providing medical care. Many practicing endocrinologists have chosen their specialty because of Dr. Burman's passion: a passion that is easily passed along to students and fellow physicians alike; a passion that drives others to love endocrinology, research, and medicine.

He serves as a professor of medicine at Georgetown University and is a past-president of the American Thyroid Association (2008 – 2009); he is also a master of the American College of Physicians. Dr. Burman has received numerous awards that include: the Claire Chennault Award presented to an Outstanding Teacher, Walter Reed Army Medical Center; the Distinguished Physician Award from the University of Missouri Medical School; Dr. John Dunn Lecture Award, the University of Virginia Medical Center, Charlottesville; the Dr. Leon Georges Lecture Award, Eastern Virginia Medical Center, Norfolk; the Dr. Walter Lester Henry Award for Lifetime Excellence in Teaching, American College of Physicians, DC Chapter; the Van Meter and John Stanbury Awards from the American Thyroid Association; and the Citation of Merit from the University of Missouri Medical School.

Dr. Burman is an extraordinary clinician and teacher who has contributed significantly to the Endocrine Society missions of education and science.

Many practicing endocrinologists have chosen their specialty because of Dr. Burman’s passion: a passion that is easily passed along to students and fellow physicians alike; a passion that drives others to love endocrinology, research, and medicine.”
James (Jay) Findling has been in clinical endocrinology practice in Milwaukee, Wisc., for over 30 years. After graduating from the University of Notre Dame and Northwestern University Medical School, he did his internal medicine training at the Medical College of Wisconsin (MCW) and his endocrinology fellowship at the University of California-San Francisco (UCSF). Jay is currently director of Community Endocrinology Services at Froedtert Health and he is a Clinical Professor of Medicine at MCW.

Jay has established himself as an internationally recognized expert on Cushing syndrome. As a fellow at UCSF, he introduced inferior petrosal sinus sampling. He later championed the use of late-night salivary cortisol for the diagnosis of Cushing syndrome. Although his national referral practice focuses on patients with pituitary-adrenal disorders, he continues to see patients with diabetes and other endocrine disorders. Despite his busy clinical practice, Jay has been committed to clinical research and the education of others with numerous publications, reviews, and invited lectureships. He has served on the board of directors of the Southeastern Wisconsin chapters of both the American Diabetes Association and the Juvenile Diabetes Research Foundation. He has been on the executive board of the Cushing Support and Research Foundation since its inception. Jay has served on many Endocrine Society committees including Steering, Nominating, Special Programs, and Self-Assessment (ESAP).

Jay is recognized for his keen clinical instincts, common sense but thorough approach to clinical problems, and his unique sense of humor. He understands the demands confronting endocrinologists in clinical practice diagnosing and managing patients with endocrine disorders. He acknowledges how much he has learned from his mistakes and his intellectual honesty is appreciated by his patients and his peers. He has a genuine passion for his scholarship, continued learning, and helping patients with complex endocrine disorders — especially those with the Cushing syndrome.

— Hershel Raff, PhD, is professor of medicine in the division of endocrinology, metabolism, and clinical nutrition at Medical College of Wisconsin in Milwaukee.
Ian D. Hay has made enormous contributions to clinical endocrinology as a scholarly physician through his work on outcome prediction and clarifying effective management of patients with low-risk papillary thyroid carcinoma (LRPTC). His 1986 study of PTC patients treated from 1946 to 1970 set the standard for outcomes after definitive primary surgery without radioiodine remnant ablation (RRA) and identified all patient and tumor prognostic variables relevant to tumor recurrence and cause-specific mortality (CSM).

In 1987, he reported the first multivariate analysis of CSM in PTC and defined a novel prognostic scoring system derived from age, grade, extent, and size (AGES), wherein a score <4 denoted LRPTC with a 20-year CSM of <1%. In 1993, he defined the MACIS prognostic scoring system wherein a score <6 identifies >85% of PTC patients who are at low risk (<1%) of CSM. From 1986 to 2010, he showed that radioiodine remnant ablation (RRA) does not improve CSM or tumor recurrence in the 85% - 90% of PTC patients with AGES<4 or MACIS<6 LRPTC and advised “selective use” of RRA. In the early 1990s, in conjunction with Santoro and Fusco, he performed cytogenetic and molecular genetic studies which characterized the 10q inversion resulting in RET/PTC. In 1991, he pioneered ultrasound-guided percutaneous ethanol ablation (UPEA) in managing recurrent neck nodal metastases, a technique now used more than 200 times a year at The Mayo Clinic.

Since 1992, he has studied the outcomes in papillary thyroid microcarcinoma (PTM) and recently demonstrated the efficacy of UPEA in definitively treating PTM with this inexpensive outpatient procedure. In 1997, he personally wrote the first AACE thyroid cancer clinical practice guidelines. In 2018, he published an eight-decade study of 4432 PTC patients, where he demonstrated that advances in biochemistry and imaging, introduced to Mayo practice since 1976, have not improved outcome in children and adults with LRPTC, when compared to an earlier cohort treated from 1936 to 1975.

“... has made enormous contributions to clinical endocrinology as a scholarly physician through his work on outcome prediction and clarifying effective management of patients with low-risk papillary thyroid carcinoma.”
Gerald D. Aurbach Award for Outstanding Translational Research

Helen Hobbs, MD

Helen H. Hobbs, MD, received her clinical training at Columbia-Presbyterian Medical Center and the University of Texas Southwestern (UTSW) Medical Center. After completing her endocrinology training and post-doctoral fellowship with Drs. Joseph Goldstein and Michael Brown, she joined the UTSW faculty, rising through the ranks to professor of internal medicine and molecular genetics. She has been an Investigator at the Howard Hughes Medical Institute since 2002.

In 1999, Dr. Hobbs started the Dallas Heart Study, a phenotypically well-characterized, multiethnic, population-based study in Dallas. Together with Jonathan Cohen, she demonstrated how rare and low frequency variants contribute to complex traits in the general population. They showed that low frequency variants in PCSK9 not only lower plasma levels of LDL-cholesterol, but also confer protection from heart disease. This led to the development of anti-PCSK9 antibodies, FDA-approved agents for treatment of hypercholesterolemia. In a similar fashion, she found that mutations in members of the angiopoietin-like family (ANGPTL-3, -4, and -8) lower plasma lipid levels. Anti-ANGPTL3 antibodies are in development for treatment of hypercholesterolemia. More recently, Drs. Hobbs and Cohen have identified sequence differences in two genes, PNPLA3 and TM6SF2, that confer risk for fatty liver disease, a burgeoning health problem.

Among Dr. Hobbs’ honors was her election to the National Academy of Medicine in 2004 and National Academy of Sciences in 2007. She received the Breakthrough Prize in Life Sciences and Passano Award (with Jonathan Cohen) in 2016 and the Harrington Prize for Innovation in Medicine in 2018. Dr. Hobbs is recognized for her contributions to the development of new lipid-lowering strategies by identifying genetic variants of large effect in humans. Importantly, her work created a new strategy using human genetics to identify new therapeutic targets for the treatment of complex cardiovascular and metabolic disorders.

“Importantly, her work created a new strategy using human genetics to identify new therapeutic targets for the treatment of complex cardiovascular and metabolic disorders.”

—E. Dale Abel, MBBS, MD, PhD, President-Elect of the Endocrine Society, is the Chair of the Department of Internal Medicine and Director of the Fraternal Order of Eagles Diabetes Research Center at the Carver College of Medicine of the University of Iowa, where he holds the John B. Stokes III Chair in Diabetes Research and the François M. Abboud Chair in Internal Medicine. He received the 2012 Gerald D. Aurbach Award for Research. (Original Citation was Written by the nominators, Alice Chang, MD, on behalf of Women in Endocrinology.)
John J. Kopchick is an internationally recognized leader in the growth hormone (GH) field. Dr. Kopchick is a Goll-Ohio Eminent Scholar and Distinguished Professor of Molecular Biology in the Department of Biomedical Sciences at the Heritage College of Osteopathic Medicine and in the Edison Biotechnology Institute at Ohio University.

He received his PhD in 1980 from the Graduate School of Biomedical Sciences at the University of Texas. He then performed postdoctoral studies in molecular virology at the Roche Institute of Molecular Biology and later was a group leader in molecular medicine at Merck & Co. Dr. Kopchick and his group were the first to discover and characterize GH receptor antagonists, an accomplishment for which he and Ohio University were awarded several U.S. and European patents. He was instrumental in founding a company, Sensus, which applied his research to the development of an FDA-approved drug (Pegvisomant) for patients with acromegaly. He and his group also generated a GH receptor gene disrupted mouse, which has the longest lifespan of any laboratory mouse strain.

Dr. Kopchick has published more than 350 scientific articles, mentored countless trainees, and serves, or has served, on the editorial boards of Endocrinology, Molecular Endocrinology, Growth Hormone & IGF-1 Research, Pituitary, and The Journal of Biological Chemistry. He served as president of the Growth Hormone Research Society and still serves on its Council. He has received many awards including the British Endocrine Society Transatlantic Award, AMVETS Silver Helmet Award, along with two honorary doctoral degrees. His ability to engage a wide variety of disciplines is illustrated by his extensive and meaningful collaborations around the globe and was pivotal in his efforts to get Pegvisomant to clinical use. Importantly, his research continues to refine our understanding of the molecular aspects of how GH influences growth, diabetes, and aging.

His ability to engage a wide variety of disciplines is illustrated by his extensive and meaningful collaborations around the globe.”
The quantity, quality, and breadth of Dr. Dolores (Dorrie) Lamb’s mentoring activities are extraordinary. They include mentoring high school students, undergraduates, graduate students, post-doctoral fellows (MD, PhD, MD/PhD), residents, and early career faculty as well as continued mentoring of former trainees.

As a PhD research scientist and clinical lab director, she has had an enormous impact in training researchers and clinicians in male reproductive biology and medicine. She has served as the primary mentor for 10 PhD students, 17 post-doctoral, and nearly 100 clinical research fellows working in diverse areas of the endocrine control of male reproductive development and function. She mentors them not only in how to do research and to think as a scientist, but in writing, presentations, and other skills necessary to be a successful scientist. It is remarkable how many of her trainees have won awards and fellowships for their work and progressed to academic positions (nine trainees are now department chairmen in the U.S. and abroad).

In addition to her success as a mentor of her own trainees, she has established training programs at multiple levels. She is the PI of a T32 and a large K12 training grant to support research career development and a recently completed K12 for male reproductive health research training (MRHR). Her success in developing trainees who are from underrepresented minorities is also noteworthy. The summer training experience in her laboratory has been life- and career-changing for some who subsequently decided to enter graduate programs after their summer research experience.

For her success in training, she was awarded the American Urologic Association Foundation’s Distinguished Mentor Award and the Faculty Educator Award in the Scott Department of Urology for extraordinary contributions and mentorship in urologic research. The Endocrine Society also benefits from her participation in the joint WE/Endo mentoring programs.
It is my great pleasure to introduce Ana Claudia Latronico, MD, PhD, for the 2019 International Excellence in Endocrinology Award from the Endocrine Society.

Ana is currently the head professor of the Endocrinology and Metabolism Division of Sao Paulo University in Brazil and one of the pioneers of translational molecular investigation in the human adrenal and pubertal disorders areas. She has a very special ability for developing long-term and very productive scientific collaborations with recognized international researchers. Ana is currently one of the top faculty leaders of the internationalization project of Sao Paulo University.

Her publication record speaks for itself, both in terms of the laboratory’s original research as well as collaborations with leading research groups around the world. She has an extensive list of seminal publications, many in high profile journals, such as *The New England Journal of Medicine, Lancet Endocrinology and Diabetes*, and *The Journal of Clinical Endocrinology & Metabolism*. One of her most important research achievements was to first elucidate the genetic basis of familial central precocious puberty in humans, previously considered an idiopathic disease. Ana is a very enthusiastic mentor who has transmitted her passion in scientific investigation to her trainees, inspiring many of them to pursue careers as physician-scientists.

Because of her body of work, she has received many awards and international distinctions, including Young Investigator Award from Brazilian Endocrine Society, 2017 Mentor Award from Brazilian Federal Research Agencies (CAPES), and from Sao Paulo University. In 2003, she received the Endocrine Society’s Richard E. Weitzman Laureate Award. Ana has been honored to serve as a plenary speaker in several prestigious scientific international endocrine events. Ana Claudia Latronico is a quintessential leader-role model whose lifetime work bridges basic science, clinical investigation, and endocrine education.

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**— VINICIUS BRITO, MD, IS A CLINICAL RESEARCHER, DIVISION OF ENDOCRINOLOGY, HORMONE AND MOLECULAR GENETICS LABORATORY (LIM/42), UNIVERSITY OF SAO PAULO MEDICAL SCHOOL, SAO PAULO, BRAZIL.**

**Ana Claudia Latronico is a quintessential leader-role model whose lifetime work bridges basic science, clinical investigation, and endocrine education.”**
Sidney H. Ingbar Award for Distinguished Service

Carole R. Mendelson, PhD

“Carole R. Mendelson, PhD, the 2019 Sidney H. Ingbar Distinguished Service Awardee, has worked continuously and tirelessly for the Endocrine Society for over 30 years. She has served on a remarkable number of Endocrine Society committees and editorial boards of Society journals which include: member and chair of the Education Committee; member and chair of Publications, during the transition to electronic publications; member and overall chair of the Annual Meeting Steering Committee; and member and chair of the Nominations Committee. Carole was a member of the editorial boards of Molecular Endocrinology for two terms and of Endocrine Reviews. She served as a member of Council and then as Vice-President for Basic Science from 2008 to 2011, during which time, she also chaired the Strategic Planning Committee. Carole was appointed Endocrine Society representative to the FASEB Board of Directors, was elected to the FASEB Public Affairs Committee, and currently is a member of the FASEB Training and Career Opportunities subcommittee. She recently completed a second term as a member of the Endocrine Society Nominating Committee.

In addition to direct Endocrine Society service and leadership, Carole has contributed greatly to other activities promoting the field of endocrinology and, particularly, women in science. She was president of Women in Endocrinology during the time the mentoring program was developed. This program was the precursor of the Endocrine Society Mentor Exchange program. At UT Southwestern, she has co-chaired the Women in Science and Medicine Advisory Committee for the past 12 years. She is co-chair of the Selection Committee of the Reproductive Scientist Development Program (a K12 program funded by NICHD). Finally, she served on editorial boards of national journals, numerous study sections, and in leadership roles in organizations focusing on endocrinology, reproductive biology, and birth defects.”

Patrick Seale has established himself as a leading researcher in the fields of adipocyte biology, obesity, and metabolism. His distinctive and innovative research is centered on the intersection of developmental biology and metabolism and addresses fundamental questions about: (1) the developmental origins of adipocytes; (2) transcriptional mechanisms that control fat cell fate; and (3) the role of adipocyte developmental programs in disease settings such as aging, obesity, and type 2 diabetes.

Dr. Seale identified PRDM16 as a major regulator of brown fat differentiation and made the paradigm-shifting discovery that brown fat and skeletal muscle have a common developmental origin. He has more recently uncovered a striking requirement for PRDM16 in maintaining brown fat fate during aging. He also showed that the genetic induction of brown fat-like cells in white adipose, which he termed “beige,” is sufficient to suppress obesity and insulin-resistance. This paved the way for an explosion of research focused on the biology and therapeutic potential of beige fat cells. He has also used classical developmental approaches to define the molecular identity of beige and brown fat precursor cells, opening up a new area of investigation in adipocyte biology.

Complementing his developmental and genetic approaches, Dr. Seale has used genome-wide and chromatin-based methods to discover a critical role for the transcription factor EBF2 in brown fat determination and to identify a novel mechanism by which PRDM16 activates brown fat gene transcription. In addition to publishing many prominent and highly cited research articles, Dr. Seale has written authoritative reviews in top journals. Thus, in a relatively short period of time, Patrick Seale's research has defined a developmental and transcriptional hierarchy in brown fat which has provided the basis for research studies being conducted around the world. He is most deserving of the Richard E. Weitzman Outstanding Early Career Investigator Award.

“...In a relatively short period of time, Patrick Seale’s research has defined a developmental and transcriptional hierarchy in brown fat which has provided the basis for research studies being conducted around the world.”

— MITCHELL LAZAR, MD, PHD, A PROFESSOR OF MEDICINE AT THE UNIVERSITY OF PENNSYLVANIA, RECEIVED THE 2013 GERALD D. AURBACH LAUREATE AWARD.
Dr. Cheryl Lyn Walker, a leader in the field of genetic, epigenetic, and environmental interactions, has made seminal contributions to our understanding of how environmental exposures, especially endocrine-disrupting compounds (EDCs), increase risk for hormone-dependent (and other) cancers. She was among the first to show that tumor suppressor genes were the target for chemical carcinogens in the environment. She discovered that the tumor suppressor ATM, a DNA repair kinase, “moonlighted” in the cytoplasm controlling peroxisome number using ROS as a rheostat targeting excess/dysfunctional peroxisomes for elimination. This work solved a major riddle in cell biology (How do cells regulate peroxisome number?) and resulted in receipt of the Cozzarelli Prize from the National Academy of Sciences.

A leader in the field of hormone-dependent tumorigenesis, she developed an animal model for uterine leiomyoma/fibroids that became the gold-standard for research in this disease and provided key insights into how hormones and EDCs promote these tumors. One of her most important contributions was her identification of a molecular mechanism for EDC-induced reprogramming of the epigenome of developing tissues to increase disease susceptibility later in life, a process known as developmental reprogramming. Most recently, her epigenomic studies uncovered a new role for chromatin remodelers and elucidated that the epigenetic machinery of the Histone Code plays an equally important role in remodeling the cytoskeleton and directing the Tubulin Code.

Beyond her scientific contributions, she has had a tremendous impact on the field through activities including chair of the NIEHS TaRGET II Consortium, founder of the Center for Translational Environmental Health Research, and founder and director of the Center for Precision Environmental Health at Baylor College of Medicine. In 2016 she was elected to the National Academy of Medicine.

_—ANDREA GORE, PHD, PROFESSOR OF PHARMACOLOGY AT THE UNIVERSITY OF TEXAS AND FORMER EDITOR-IN-CHIEF OF ENDOCRINOLOGY, WAS ONE OF FOUR RECIPIENTS OF THE 2016 OUTSTANDING PUBLIC SERVICE LAUREATE AWARD._
William F. Young, Jr., MD, MSc, is the Tyson Family Endocrinology Clinical Professor, Mayo Clinic College of Medicine in Rochester, Minn., where he is a past chair of the Division of Endocrinology. Spanning a four-decade career, his contributions as clinician, researcher, educator, committee member, and editor are exceptional, trailblazing, and numerous in both fundamental and clinical endocrinology. With a career-long dedication to the Endocrine Society, he has served on eight committees and task forces, four of which he chaired. As president of the Endocrine Society (2012-2013), he founded the “Highlights of ENDO” initiative where presentations from the annual Endocrine Society meeting are delivered at selected national endocrine meetings in Asia, the Americas, and Europe. He also launched the innovative Endocrine Society Ambassador Exchange Program with medical centers around the world where resources are scarce and indigent populations are served. Dr. Young also served the endocrine community as the inaugural chair of the Endocrinology Specialty Board at the American Board of Internal Medicine (2014-2018).

In meticulously crafted and lucid lectures, Dr. Young has delivered more than 430 presentations at national and international meetings and has been a visiting professor at more than 140 medical institutions. In addition, he has hosted more than 50 visiting clinicians to Mayo Clinic from around the world for one-on-one mentorship and education. His publications number more than 300 with an h-index of 57. Passionate to serve and educate, since 2006 he has answered more than 7,500 e-mails from clinicians around the world seeking guidance on the best clinical management of patients with complex endocrine disorders.

Truly, Dr. William F. Young, Jr., belongs to the world and eminently deserves to receive the Outstanding Leadership in Endocrinology Award for 2019.
Sundeep Khosla, MD, talks to Endocrine News about his ENDO 2019 plenary on treating osteoporosis via cellular senescence with the ultimate goal of making the golden years truly golden with a better quality of life.

ENDO 2019 is now about 10 weeks away, and while we’re excited about all the sights, sounds, and smells of New Orleans, we’re just as excited about the sessions and all the breakthroughs in the field of endocrinology.

For instance, on Monday, March 25, Sundeep Khosla, MD, of the Mayo Clinic College of Medicine in Rochester, Minn., will give a plenary talk titled “Targeting Cellular Senescence in Age-Related Osteoporosis and Frailty,” which posits that inhibiting cellular senescence could be a novel therapeutic strategy to prevent bone loss and other age-related diseases. The talk follows up on a mini-review that appeared in The Journal of Clinical Endocrinology & Metabolism in 2018, which concluded: “Targeting cellular senescence represents a new therapeutic paradigm for preventing or even reversing age-related osteoporosis and simultaneously treating multiple aging comorbidities. This approach does not focus specifically on bone but rather on a fundamental aging mechanism operative across tissues. If the remarkable promise of preclinical models is realized in human studies, we may truly have a novel approach to enhance healthspan (and perhaps lifespan) in the rapidly growing aging population in the U.S. and throughout the world.”

Endocrine News caught up with Khosla to discuss his forthcoming plenary and what his work could mean for the future of healthcare.
We’re not even focusing that much on lifespan; we are really focusing on health-span. We’re all going to get old, but can we maintain a more productive life for our lifetime, however long that is? Our goal is that whatever time we have, we spend it in the best possible health and productively, so we’re not in a nursing home and have a better quality of life.”

**ENDOCRINE NEWS:** First off, since this is a preview of your plenary, can you share any of the highlights or details of the talk?

**SUNDEEP KHOSLA:** Basically, I’m going to start off just by providing a brief background on how much progress has been made in terms of drugs to treat osteoporosis. But despite that progress, because of concerns about rare side effects related to existing osteoporosis drugs, there are many, many patients who are at risk of fracture but are, in fact, not getting treated.

There’s a strong argument for thinking about new approaches to prevent and treat osteoporosis. That’s when I was attracted to this idea with these diseases related to aging, such as heart disease, diabetes, osteoporosis. The historical approach is to treat each of the diseases separately — so you take a statin and a blood pressure medicine for cardiovascular risk. You take a different drug for diabetes. That leads to problems with patients as they get older being on many different drugs which causes adverse drug interactions, non-compliance, and so forth.

The concept that we and others are trying to put forward is that you have this constellation of diseases associated with aging like osteoporosis, dementia, diabetes. But they share the same risk factor, which is aging. So is it possible, instead of targeting each disease separately, can we in fact target a common aging mechanism to maybe try to treat multiple aging diseases? That’s really where the geroscience hypothesis has come about — that we could manipulate fundamental aging mechanisms that will delay the appearance or severity of multiple chronic diseases. Then I’ll move right into our work on osteoporosis and the identification of senescent cells in the

Since aging is the common risk factor for myriad conditions from dementia to diabetes, targeting the overall aging mechanism could stop or even reverse some chronic diseases. This would, in turn, enhance the quality of life and even extend a person’s lifespan.
bone microenvironment. How, when we target these cells, we can prevent bone loss in aged mice. And that we see a similar increase in senescent cells in humans with aging in the bone microenvironment, so arguing that this isn’t a phenomenon limited to mice.

**EN:** We featured some of your work last year in a Trend, on how targeting cellular senescence could halt age-related bone loss. Will this talk be a continuation of that work? Have there been any developments since then that you’d like to share?

**SK:** Yes – additional data beyond what you featured including: (1) that estrogen deficiency likely does not involve increases in cellular senescence, which is more characteristic of aging – this has implications for the FDA approval of “senolytics” (which kill senescent cells) for osteoporosis; (2) additional data in collaboration with my colleague, Jim Kirkland, showing that reducing senescent cell burden ameliorates age-related frailty and extends lifespan in mice.

The two main causes of osteoporosis in humans are menopause, which causes estrogen deficiency, and aging. So, we have extensive data showing that with aging, you get cell senescence and other aging mechanisms. But, with estrogen deficiency we actually don’t see an increase in cell senescent markers in bone. That’s relevant to the FDA, so if we have new drugs that kill senescent cells, for the FDA, the standard animal model is a gonadectomy-induced bone loss model. These drugs won’t work in the acute gonadectomy setting, but they’ll work in the aging setting. So, there’s implications for these drugs — as they’re applied to humans — as they go for FDA approval.

We have some more recent data and collaboration with Dr. Kirkland on the role of cell senescence in frailty models with aging. And, how targeting senescent cells not only prevents frailty with aging in the mice, but also actually extends their lifespan.

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“The concept that we and others are trying to put forward is that you have this constellation of diseases associated with aging like osteoporosis, dementia, diabetes, so forth. But they share the same risk factor, which is aging. **So is it possible, instead of targeting each disease separately, can we in fact target a common aging mechanism to maybe try to treat multiple aging diseases?”**
In terms of thinking about fracture, that’s a function of the low bone density as well as frailty and falls. So, potentially, these drugs could address both aspects and thereby prevent fractures from occurring with aging.

Then I’m just going talk a little bit about clinical trials that we have ongoing. They’re early stage, “proof-of concept” clinical trials. We also have to consider the potential downsides of killing senescent cells. Such as, in a wound repair, or potentially consequences for cancer development.

EN: In that JCEM article, you wrote that “the number of women who will experience a fracture in one year exceeds the combined number of women who will experience incident breast cancer, myocardial infarction, or stroke.” That seems like a staggering number. And that’s compounded by this declining interest among pharmaceutical companies in developing osteoporosis drugs. That seems like a kind of perfect storm of why this sort of novel research is needed now.

SK: There are several reasons why pharmaceutical interest has declined. One, is that the FDA has required demonstrating a robust fracture risk reduction for approval. The drug companies have been competing with each other to show a reduction in hip fracture rates or non-vertebral fracture rates. That requires very large studies of the order of 10,000 to 15,000 subjects. They’re very, very expensive. And unlike a diabetes drug, for example, where basically you have to show a reduction in the hemoglobin and A1C, or a blood pressure drug, where you have to show a reduction in blood pressure, for osteoporosis, the numbers that you need to show fracture reduction are huge.

There are a number of us that are working with the FDA to see if drugs can be fast-tracked based on using bone density as a surrogate marker, rather than requiring a fracture end point. That’s still in discussion with the FDA.

The osteoporosis trials are really very expensive. I think the other part of it has been that because of patients not wanting to take the existing drugs due to potential side-effects, the drug companies are concerned that some of this reluctance may spill over into some of these other drugs. So they’ve kind of shifted their focus more into frailty, muscle, or diabetes, rather than on the bone area. I think this was also compounded by the fact that Merck had a new drug called odanacatib, for which they spent $1 to $1.5 billion on for a trial. And it worked really well for bone, but because of an increase in stroke events, which was modest, there was a clear signal to pull the plug on the drug. That was a huge negative in terms of osteoporosis drugs.
There’s a number of things that have been happening in terms of major clinical trials failing, the size of the trials that are required for fracture, and then collectively that’s caused the pharma companies to move into other areas and kind of leave osteoporosis behind for now.

**EN:** Your work seems like an answer to that. Is that fair to say?

**SK:** What we hope is that instead of just focusing on osteoporosis, if we put osteoporosis in the context of these aging diseases, maybe we can attract pharma back, because then osteoporosis is just one of multiple other diseases as opposed to the only disease that they’re working on.

**EN:** Right. You talk about wanting to focus on aging itself.

**SK:** We’re not even focusing that much on lifespan; we are really focusing on health-span. We’re all going to get old, but can we maintain a more productive life for our lifetime, however long that is? Our goal is that whatever time we have, we spend it in the best possible health and productively, so we’re not in a nursing home, and have a better quality of life. I think that’s the more modest, achievable goal. As opposed to making people live longer, which has its own set of problems because eventually everybody gets debilitated.

**EN:** In the “Hallmarks of Aging” (Lopez-Otin, et al.) article you pointed me to, it talks about the primary purpose of senescence as preventing the proliferation of damaged cells, but aged organisms have a harder time accomplishing this, leading to an accumulation of senescent cells.

**SK:** That’s the issue with cell senescence: we get senescent cells with tissue injury, because they do seem to help in tissue repair. And, cell senescence may have evolved as an anti-cancer mechanism, where when cells develop these toxicities, that instead of being converted into a cancer cell, there are these growth-arrest genes that are activated that basically shut off the pathway to cancer, but the alternate pathway is cell senescence, but then they make all these bad cytokines that cause aging or cause tissue disfunction.

So, I think we have to be careful how we interfere with that mechanism. The approach that we’re proposing is not to keep senescent cells from forming, but by giving the senolytic drugs very intermittently, maybe just once a month, all you’re doing is reducing the burden of these senescent cells.

**EN:** And finally, what do you want the audience members to take away from your ENDO talk?

**SK:** That we need to broaden our focus when thinking about age-related co-morbidities (i.e, osteoporosis, frailty, diabetes, cardiovascular disease, dementia, renal failure, etc.) and instead of targeting each disease separately, consider approaches that target fundamental aging mechanisms – senescence is just one of nine key aging mechanisms (genomic instability, telomere attrition, epigenetic alterations, loss of proteostasis, deregulated nutrient sensing, mitochondrial dysfunction, cellular senescence, stem cell exhaustion, and altered intercellular communication) – to perhaps simultaneously treat multiple age-related co-morbidities.

They’re all kind of interconnected, but by targeting, whether it’s senescence or any of these other pathways, if that becomes a significant success, technically you could actually reduce some of these pathways of aging. Then it’s very possible that with one drug, or a small number of drugs, you can prevent bone loss, reduce the risk of diabetes, heart disease, dementia — a whole host of aging diseases. So, in a perfect world, that would be the outcome, that you have intermittent administration of a small number of medications with minimal side effects that reduce your risk for all of these things that we are going to get as we get older. It’s very exciting, being involved in this research.

“...That’s really where the geroscience hypothesis has come about — that we could manipulate fundamental aging mechanisms that will delay the appearance or severity of multiple chronic diseases.”
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Over the past few years, Leonardo Trasande, MD, MPP, from the New York University School of Medicine, has not been a stranger to the readers of *Endocrine News*.

Whether he was leading a research team whose work was published in *The Journal of Clinical Endocrinology & Metabolism* and featured in a 2016 Trends article to speaking at **ENDO 2015** on the costs of endocrine disruption to following his travels around the globe on behalf of the Endocrine Society to advocate for better endocrine-disrupting chemical (EDC) regulations, Trasande has been a vocal proponent of the importance of improving public health through reducing EDC exposures.

And now he’s written a book.

*Sicker, Fatter, Poorer: The Urgent Threat of Hormone-Disrupting Chemicals to Our Health and Future ... and What We Can Do About It* was recently published by Harcourt Miflin Harcourt and reaches out to a lay audience to reveal to them the alarming truth about how EDCs are affecting our daily lives and what to do to protect ourselves as well as fight back.

In *Sicker, Fatter, Poorer*, Trasande exposes the chemicals that disrupt hormonal systems and damages human health in irreparable ways. He discusses where these chemicals hide as well as the workings of policy that protects the continued use of these chemicals in everyone’s lives. Drawing on extensive research and expertise, Trasande outlines dramatic studies and emerging evidence about the rapid increases in neurodevelopmental, metabolic, reproductive, and immunological diseases directly related to the thousands of chemicals the average person is exposed to every day.

Through a blend of narrative, scientific detective work, and concrete information about the connections between chemicals and disease, Trasande shows readers what they can do to protect themselves and their families in the short-term, and how to help bring much needed change. *Endocrine News* caught up to Trasande prior to the book’s publication to find out what is needed to eliminate EDCs from daily life and why the economic impact of doing so will not be as detrimental as some people think.
LEO TRASANDE: We are amidst an epidemic of endocrine-related diseases, and evidence continues to mount that synthetic chemicals contribute to these preventable conditions. Awareness has increased in recent years, but remains limited among the public, policy makers, and clinicians alike. This book has two goals: To present the science in a digestable format for people to make their own judgments and empower the public with the tools to reduce their exposures and improve their health without disrupting their budget. There’s also a misconception that protecting the public from these exposures will hurt our economy. Quite the opposite actually – preventing these exposures can actually potentiate economic productivity among generations to come! Look at the ban on lead in gasoline, which provides a $200 billion annual economic stimulus in the U.S. each year. Globally, the stimulus is $2.4 trillion – 4% of GDP!

EN: The public health issues caused by EDCs are diverse and complex, how did you select the health effects covered in the book?

LT: I focused on these effects because the evidence for EDCs is the strongest, as described in the Endocrine Society Second Scientific Statement and the World Health Organization/United Nations Environment Programme report on endocrine disruptors in 2012.

EN: What are three things that regulators, policy makers, and the public can do right now to reduce the economic and public health burdens associated with EDCs.

LT: There are safe and simple steps we can all take to reduce our exposures, and stronger regulation would substantially reduce the disease burden due to endocrine disruption.

Yet, we have much more power as a society – through our pocketbooks and wallets, and also through our employers, schools, and other companies who have large purchasing power. The ban on BPA in sippy cups and baby bottles occurred not because FDA wanted to do the right thing, but because consumers cried out to companies asking whether BPA was in these products.

EN: The book provides a hopeful vision for the future. What research gaps should funding agencies prioritize to ensure that medical professionals in 2040 have the resources that they need to address EDC exposures?

LT: We urgently need a revamp and modernization of toxicology testing methods, and European Union funding agencies have already responded while the U.S. has not. The Environmental Influences on Child Health Outcomes program and the Children’s Health Exposure Analysis Resource will advance understanding of early life exposures, but the All of Us Program has not communicated a focus on EDC exposures. EDC research should be supported by institutes and centers other than National Institute of Environmental Health Sciences and developing world research is urgently needed. Low- and middle-income countries will produce and use a majority of synthetic chemicals by 2030, and we are already seeing epidemic increases in endocrine-related diseases there.

Available in both hardcover and as an ebook, Sicker, Fatter, Poorer can be purchased at amazon.com as well as all other booksellers.
Frustrated by the gaps she saw between theoretical and actual patient outcomes, Varsha Vimalananda, MD, MPH, decided to pursue a career in health services research, which looks at quality, access, and costs of healthcare, and may even help endocrinologists lead the way for measuring and improving how endocrine care is delivered.

BY GLENDIA FAUNITEREOY SHAW

During her clinical training, Varsha Vimalananda, MD, MPH, says she experienced firsthand the gaps between theoretically possible outcomes and what she could accomplish for her patients within the constraints of policy, organizational, and socioeconomic factors. Discontent with this gap led Vimalananda, a physician-scientist at the Veterans Affairs (VA) Center for Healthcare Organization and Implementation Research (CHOIR), to a career in health services research (HSR). “My ultimate goal is to help narrow the gap between the promises of carefully controlled clinical studies and actual patient outcomes,” she says.

Endocrine News caught up with Vimalananda to learn more about what HSR could mean for the endocrine community.

ENDOCRINE NEWS: What is health services research (HSR)?

VARSHA VIMALANANDA: Health services research is research on quality, access, and the cost of healthcare. This is translational research that focuses on where the rubber meets the road in terms of the impact of basic science and clinical trials in the real world. HSR “studies how social factors, financing systems, organizational structures and processes, health technologies, and personal behaviors affect access to healthcare, the quality and cost of healthcare, and ultimately our health and well-being,” according to the Agency for Healthcare Research and Quality. We apply robust theoretical frameworks to understand and improve healthcare delivery at the policy, community, organizational, provider, and patient/family level.

HSR projects are often conducted by teams with various areas of high-level expertise. For example, I started my research career doing large-scale secondary data analyses. My current career development award from the VA is focused on measuring and improving coordination of specialty care. I am applying both survey methods and qualitative methods. I will then design and trial an intervention that I will refine and scale up in future work, using methods from implementation science. I’ll collaborate with policy and hospital leaders and incorporate feedback from mentors and research center colleagues throughout. If I help improve some part of specialty care
coordination and its many related outcomes, this research agenda will have been a success.

EN: How could HSR be valuable for endocrinologists?

VV: HSR is of value to anyone who wants to improve endocrinology healthcare delivery. Everyone who practices clinical medicine probably identifies these opportunities every day. Think about the prevalence and burden of endocrine conditions like obesity, diabetes, thyroid disease, and osteoporosis. Findings from HSR about how to improve care delivery for these conditions can translate to major gains in health, cost savings, and patient experience.

There is a lot of very strong, high-impact, endocrinologist-led HSR, but it is often not identified as such. At the institutional level, there may be one or no other endocrinologist in HSR. Creating an HSR community of endocrinologists, disseminating our work, mentoring, and advancing collaborations may need to happen at a national level. The major benefit of greater visibility for HSR would be to maximize endocrinology's impact and influence over the healthcare delivery research agenda and ultimately the quality of care for conditions about which we have the most specialized knowledge. We should be the leaders in measuring and improving how we deliver care in our field.

EN: Are there any major HSR studies that have shaped policy or practice in other specialties?

VV: But of course! Many may be familiar with the impact of HSR in cardiology that identified barriers to goal door-to-balloon times, developed interventions to overcome those barriers, and implemented them broadly with great improvement in guideline-concordant care and outcomes in the U.S.

EN: For anyone interested in HSR, what resources and training are available?

VV: Physicians have a unique role in HSR — we have clinical expertise and depth of firsthand knowledge about how the healthcare system functions. To that, you need to add basic training in HSR. A master's degree in public health or similar field is very helpful. Many medical schools offer T32 programs to fund such training. There are also certificate programs, short courses, and fellowships that offer HSR training. For an early career researcher, strong mentorship is necessary for success. For that, you may need to look beyond your own section to other clinical sections or schools of public health. A little more effort is required to create your research program in HSR coming from a subspecialty, but I have found it well worth it.

“...A little more effort is required to create your research program in HSR coming from a subspecialty, but I have found it well worth it.”

Glenda Fauntleroy Shaw is a freelance writer based in Carmel, Ind. She is a regular contributor to Endocrine News.
A federal judge in Texas struck down the 2010 Affordable Care Act (ACA), siding with a group of conservative states that argued the law is unconstitutional. At issue was whether the health law’s insurance mandate still compelled people to buy coverage after Congress reduced the penalty to zero dollars as part of the tax overhaul that President Trump signed last December after Republicans in Congress eliminated a key part of it.

When the U.S. Supreme Court upheld the mandate as constitutional in 2012, it was based on Congress’s taxing power. Congress, the court said, could legally impose a tax penalty on people who do not have health insurance. But in this new case, the state of Texas and 19 other states argued that with the penalty eliminated, the individual mandate had become unconstitutional and that the rest of the law could not be severed from it.
“Congress stated many times unequivocally — through enacted text signed by the President — that the Individual Mandate is ‘essential’ to the ACA,” Judge Reed O’Connor wrote in his ruling, "And this essentiality, the ACA’s text makes clear, means the mandate must work 'together with the other provisions' for the Act to function as intended.”

The White House issued a statement that said, “We expect this ruling will be appealed to the Supreme Court. Pending the appeal process, the law remains in place.” The Centers for Medicare and Medicaid Services Administrator Seema Verma also echoed that the law remains in place in an effort to reduce consumer confusion.

“California Attorney General Xavier Becerra's office has announced his state would appeal the ruling. California and other states had intervened to defend the 2010 healthcare law after the Trump administration declined to defend its provisions that guarantee coverage for people with pre-existing conditions, arguing that those provisions cannot be separated from the mandate.

“The ACA has already survived more than 70 unsuccessful repeal attempts and withstood scrutiny in the Supreme Court,” Becerra said in a statement. “Today's misguided ruling will not deter us: our coalition will continue to fight in court for the health and wellbeing of all Americans.”

The federal court’s decision could also set off a scramble in Congress, where some lawmakers want to step in to defend the decision. In the House of Representatives, Democrats who will take control of the chamber in January hope to pass a package to strengthen pre-existing conditions protections and plan to vote to become a party to the case early next year.

The Endocrine Society will continue to advocate to ensure that our patients have access to affordable, high-quality insurance coverage, preventive services, and patient-centered care. Since the ACA was enacted, we have called for some improvements to the law, but also called on Congress to not repeal the health law unless an adequate replacement was agreed upon to avoid harm to those who depend on the ACA. Please visit our Access to Care web page at endocrine.org/advocacy/priorities-and-positions/access-to-care to see our position statements and multiple letters to the administration and congress. If you have any questions or concerns about the ACA, please contact govtprof@endocrine.org.

Take Action

New Year; New Congress — Join the Endocrine Society Welcome to Congress Campaign.

The new U.S. 116th Congress will be officially sworn in on January 3, 2019. This presents an extraordinary opportunity for our members to reach out to their new members of Congress, offer their expertise as endocrinologists, and share what issues they and the Endocrine Society find important.

If you have a new member of Congress, you will receive notice by email from our government affairs team and have the opportunity to join our Welcome to Congress Campaign. We hope you will take a moment to join us in welcoming your new member of Congress.
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UCSF Fresno and the Central California Faculty Medical Group are recruiting for an endocrinologist at the Assistant Clinical Professor level or higher. The successful candidate will provide Endocrine services in a teaching program, will teach residents and students in Endocrinology, and will see patients in a faculty practice. Applicants must be board certified in Internal Medicine and board certified or board eligible in Endocrinology, have completed their residency in Internal Medicine and fellowship in Endocrinology, applicant must have a US medical license at the time of hire. Applicant should have clinical experience and be willing to actively participate in medical education, and have experience and interest in clinical research. The UCSF Fresno Medical Education Program sees patients in Regional Medical Center and has very successful faculty practice sites.

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what you need to know

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THYROID NODULES — Cells In The Thyroid That Form A Tumor

THYROID CANCER Doesn't Always Have Symptoms

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- A lump or swelling in your neck
- A hoarse voice
- Difficulty swallowing
- Neck or throat pain
- A swollen lymph node in your neck

• More than 90% are not harmful, but some can be cancerous
• Fewer than 1 in 10 nodules is cancerous
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