Dr. Justin Annes specializes in the treatment of hereditary endocrine disorders with particular focus on neuroendocrine-related conditions. He developed the Stanford Endocrine Genetics Clinic in 2012 which is part of the interdisciplinary Stanford Hypertension Center and Stanford Neuroendocrine Tumor Program. He has medical practice has focused on hereditary endocrine disease since 2008.

CLINICAL FOCUS

- Pheochromocytoma and Paraganglioma
- Multiple Endocrine Neoplasia
- Hereditary Endocrine Disorders
- Endocrinology

ACADEMIC APPOINTMENTS

- Assistant Professor, Medicine - Endocrinology, Gerontology, & Metabolism
- Member, Bio-X
- Member, Maternal & Child Health Research Institute (MCHRI)
- Member, Stanford Cancer Institute
- Faculty Fellow, Stanford ChEM-H

HONORS AND AWARDS

- Stanford Cancer Center Innovation Award, Stanford University (2018)
- Stanford Diabetes Research Center, Training Outreach Program (PI), Stanford University (2017-)
- R01: The Role of Adenosine Kinase in Controlling Beta-Cell Regeneration, NIH NIDDK (2015-20)
- The Best of Basic Research, Endocrine Society (2014)
Current Research and Scholarly Interests

The ANNES LABORATORY of Molecular Endocrinology: Leveraging Chemical Biology to Treat Endocrine Disorders

Diabetes

The prevalence of diabetes is increasing at a staggering rate. By the year 2050 an astounding 25% of Americans will be diabetic. The goal of my research is to uncover therapeutic strategies to stymie the ensuing diabetes epidemic. To achieve this goal we have developed a variety of innovative experimental approaches to uncover novel approaches to curing diabetes.

1. Beta-Cell Regeneration: Diabetes results from either an absolute or relative deficiency in insulin production. Our therapeutic strategy is to stimulate the regeneration of insulin-producing beta-cells to enhance an individual’s insulin secretion capacity. We have developed a unique high-throughput chemical screening platform which we use to identify small molecules that promote beta-cell growth. This work has led to the identification of key molecular pathways (therapeutic targets) and candidate drugs that promote the growth and regeneration of islet beta-cells. Our goal is to utilize these discoveries to treat and prevent diabetes.

2. The Metabolic Syndrome: A major cause of the diabetes epidemic is the rise in obesity which leads to a cluster of diabetes- and cardiovascular disease-related metabolic abnormalities that shorten life expectancy. These physiologic aberrations are collectively termed the Metabolic Syndrome (MS). My laboratory has developed an original in vivo screening platform to identify novel hormones that influence the behaviors (excess caloric consumption, deficient exercise and disrupted....
sleep-wake cycles) and the metabolic abnormalities caused by obesity. We aim to manipulate these hormone levels to prevent the development and detrimental consequences of the MS.

HEREDITARY PARAGANGLIOMA SYNDROME

The Hereditary Paraganglioma Syndrome (hPGL) is a rare genetic cancer syndrome that is most commonly caused by a defect in mitochondrial metabolism. Our goal is to understand how altered cellular metabolism leads to the development of cancer. Although hPGL is uncommon, it serves as an excellent model for the abnormal metabolic behavior displayed by nearly all cancers. Our goal is to develop novel therapeutic strategies that target the abnormal behavior of cancer cells. In the laboratory we have developed hPGL mouse models and use high throughput chemical screening to identify the therapeutic susceptibilities that result from the abnormal metabolic behavior of cancer cells.

As a physician scientist trained in clinical genetics I have developed expertise in hereditary endocrine disorders and devoted my efforts to treating families affected by the hPGL syndrome. By leveraging our laboratory expertise in the hPGL syndrome, our care for individuals who have inherited the hPGL syndrome is at the forefront of medicine. Our goal is to translate our laboratory discoveries to the treatment of affected families.

Teaching

STANFORD ADVISEES

Doctoral Dissertation Reader (AC)
Krissie Tellez

Postdoctoral Faculty Sponsor
James Holt Martyn, Sooyeon Lee

Doctoral Dissertation Advisor (AC)
Timothy Horton

Postdoctoral Research Mentor
James Holt Martyn, Sooyeon Lee

Publications

PUBLICATIONS

- CC-401 Promotes β-Cell Replication via Pleiotropic Consequences of DYRK1A/B Inhibition. *Endocrinology*
  2018

- Zinc-Chelating Small Molecules Preferentially Accumulate and Function within Pancreatic β Cells. *Cell chemical biology*
  2018

- Genetic Disruption of Adenosine Kinase in Mouse Pancreatic β-Cells Protects Against High Fat Diet-Induced Glucose Intolerance. *Diabetes*
  2017

- Electrically controlled release of insulin using polypyrrole nanoparticles *Nanoscale*
  Hosseini-Nassab, N., Samanta, D., Abdolazimi, Y., Annes, J. P., Zare, R. N.
  2017; 9 (1): 143-149

- Hyaluronan content governs tissue stiffness in pancreatic islet inflammation. *The Journal of biological chemistry*
2017

- **A High-content In Vitro Pancreatic Islet β-cell Replication Discovery Platform.** *Journal of visualized experiments : JoVE*
  Zhao, Z., Abdolazimi, Y., Armstrong, N. A., Annes, J. P.
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- **Repurposing cAMP-Modulating Medications to Promote beta-Cell Replication** *MOLECULAR ENDOCRINOLOGY*
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- **Adult tissue sources for new beta cells** *TRANSLATIONAL RESEARCH*
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- **A liver Hif-2a-Irs2 pathway sensitizes hepatic insulin signaling and is modulated by Vegf inhibition.** *Nature medicine*
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- **A liver Hif-2 alpha-Irs2 pathway sensitizes hepatic insulin signaling and is modulated by Vegf inhibition** *NATURE MEDICINE*
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- **The influence of sodium- and calcium-regulatory hormone interventions on adipocytokines in obesity and diabetes.** *Metabolism*
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- **In Vivo Screening for Secreted Proteins That Modulate Glucose Handling Identifies Interleukin-6 Family Members as Potent Hypoglycemic Agents** *PLOS ONE*
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- **Genetics of adrenocortical disease: an update** *CURRENT OPINION IN ENDOCRINOLOGY DIABETES AND OBESITY*
  Bar-Lev, A., Annes, J. P.
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- **Adenosine kinase inhibition selectively promotes rodent and porcine islet beta-cell replication** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
  2012; 109 (10): 3915-3920

- **Erdheim-Chester disease presenting with cutaneous involvement: a case report and literature review** *JOURNAL OF CUTANEOUS PATHOLOGY*
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- **Risks of Presymptomatic Direct-to-Consumer Genetic Testing.** *NEW ENGLAND JOURNAL OF MEDICINE*
  Annes, J. P., Giovannini, M. A., Murray, M. F.
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- **Integrin alpha(v)beta(6)-mediated activation of latent TGF-beta requires the latent TGF-beta binding protein-1** *JOURNAL OF CELL BIOLOGY*
  Annes, J. P., Chen, Y., Munger, J. S., Rifkin, D. B.
  2004; 165 (5): 723-734

- **A genetic screen to identify latent transforming growth factor beta activators** *ANALYTICAL BIOCHEMISTRY*
  Annes, J., Vassallo, M., Munger, J. S., Rifkin, D. B.
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• Annexin II-mediated plasmin generation activates TGF-beta 3 during epithelial-mesenchymal transformation in the developing avian heart DEVELOPMENTAL BIOLOGY
  2004; 265 (1): 140-154

• Making sense of latent TGF beta activation JOURNAL OF CELL SCIENCE
  Annes, J. P., Munger, J. S., RIFKIN, D. B.
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  Chen, Y., Dabovic, B., Annes, J. P., Rifkin, D. B.
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• The integrin alpha(V)beta(6) binds and activates latent TGF beta 3 FEBS LETTERS
  Annes, J. P., RIFKIN, D. B., Munger, J. S.
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• The latent transforming growth factor-beta-binding protein-1 promotes in vitro differentiation of embryonic stem cells into endothelium MOLECULAR BIOLOGY OF THE CELL
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• PKC-theta is required for TCR-induced NF-kappa B activation in mature but not immature T lymphocytes NATURE
  2000; 404 (6776): 402-407