BIOGRAPHICAL SKETCH

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NAME: Constantine A. Stratakis

eRA COMMONS USER NAME (credential, e.g., agency login): stratakc

POSITION TITLE: Chief, Section on Endocrinology & Genetics, NICHD, NIH (retired)

CSO, ELPEN, Inc. & Executive Director, Athens Research Institute, Athens, Greece

Director, Research, Human Genetics and Precision Medicine, FORTH, Heraklion, Crete

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
National & Capodistrian University, Athens, Greece	MD	06/1989	Medicine
National & Capodistrian University, Athens, Greece (Διδακτορικο Ιατρικων Επιστημων)	D(Med)Sci (PhD equivalent)	04/1994	Endocrinology
University of Maryland, School of Public Policy		12/2011	Senior Leadership Program (Certification)

A. Personal Statement

During my graduate work I developed an interest in the genetics of endocrine diseases, especially those that present with a predisposition to endocrine tumors and other neoplasms. My career started in 1985 as a graduate student in the Endocrine Unit of the Medical School of The University of Athens under the mentorship of Prof. M. Batrinos, and as a biochemist (tech) in a Radioimmunoassay Lab at the Hospital Mitera, Athens, Greece. In Paris, France, I spent time with Jean Pierre Luton and Xavier Bertagna in 1988; I was exposed there to research on mainly adrenal and pituitary diseases. In 1989-1990, at the NIH lab of George Chrousos, I was part of the team that identified the first genetic defects in the human glucocorticoid receptor (***J Clin Invest 1991;87:680***). Following this post-doctoral fellowship in molecular endocrinology at the NIH, I continued with my clinical training at Georgetown University Medical Center, Washington, DC, where I finished a residency and two fellowships in Pediatrics, Pediatric Endocrinology and Medical Genetics & Dysmorphology. I was recruited back to NIH, trained in linkage analysis and cancer genetics, and started a laboratory (in 1995) studying the genetics of endocrine tumors in the context of a variety of genetic syndromes. In 2000, I identified the *PRKAR1A* gene causing Carney complex (CNC) (***Nat Genet 2000;26:89***); this gene is expressed in almost all human tissues and regulates protein kinase A (PKA) and, consequently, cAMP signaling. *Prkar1a*$^{+/-}$ animal models made in my laboratory addressed the participation of *PRKAR1A* in endocrine cell growth and neoplastic development of other tissues, such as the bone (***PNAS 2010;107:8683***). My laboratory identified phosphodiesterase (PDE) genes as potentially being involved in cAMP-related tumor growth (***Nat Genet 2006;38:794; N Engl J Med 2008;358:750***). We also identified defects affecting mitochondrial oxidation in endocrine tumors (***N Engl J Med. 2007;357:1054***) and in pediatric gastrointestinal tumors (GISTs) (***PNAS 2011;108:314***). In collaboration with the hospital I was trained in France, the *ARMC5* gene was identified in macronodular adrenal hyperplasia (***N Engl J Med. 2013;369:2105***), a gene that we are now modeling in mice, fly and fish. Our laboratory identified the *PRKACA* and *PRKACB* genes in micronodular adrenal hyperplasia and Carney complex (***N Engl J Med. 2014;370:1019 & 2014;370:1065***, respectively). Most recently, our laboratory identified a new condition that we named X-LAG for "X-linked acrogigantism"; we then described the genetic defect that explains more than 80% of the cases of early gigantism and growth-hormone dependent overgrowth that appears to be due to abnormalities of an orphan G-protein coupled receptor (GPCR), *GPR101* (***N Engl J Med, 2014;371:2363***). Most recently, we identified *PRKAR1B*, *PRKACB*, and other defects in adrenal tumors and other conditions. Ongoing genome-wide work aims at identifying genetic defects for Carney Triad, wild-type GISTs, endocrine hypertension, pediatric gigantism, acromegaly, and other forms of adrenal and pituitary tumors. Molecular studies focus on PKA and cAMP signaling, PDEs, ARMC5, and GPR101. Over the years, my laboratory has trained more than 200 students, medical residents, and fellows from all over the world, including Brazil, Greece, France, Italy, Australia, and of course the United States.

B. Positions and Honors

Professional Appointments:

1984-1989 Biochemist (tech), Unit on Radioisotopes & Endocrinology, "Hospital Mitera", Athens, Greece
1988 *Externe*, Hospital Cochin, Endocrinology, Paris, France
1989-1990 Postdoctoral Associate, Developmental Endocrinology Branch (DEB), NICHD, NIH
1990-1993 Intern & Resident, Department of Pediatrics, Georgetown University (GU), Washington DC
1993-1995 Clinical Associate, Pediatric Endocrinology, NICHD, NIH
1993-1996 Medical Genetics Fellow, GU-National Center for Human Genome Research, Washington DC
1996-1997 Senior Staff Fellow, Section on Pediatric Endocrinology (SPE), DEB, NICHD, NIH
1997-1998 Staff Scientist, SPE, DEB, NICHD, NIH
1996-1998 Assistant Professor, Pediatrics, Georgetown University, Washington DC
1998-2002 Head (tenure-track investigator), Unit on Genetics & Endocrinology (UGEN), DEB, NICHD, NIH
2002-2009 Chief (tenured), Section on Endocrinology & Genetics (SEGEN), DEB, NICHD, NIH
2002-2014 Director, Pediatric Endocrinology Inter-Institute Training Program, NICHD, NIH
2003-2006 Chief, Heritable Disorders Branch (HDB), NICHD, NIH
2007-2011 Head, Program on Developmental Endocrinology & Genetics, NICHD, NIH
2009 Deputy Scientific Director, NICHD, NIH
2009-2011 Acting Scientific Director, NICHD, NIH
2011-2020 Scientific Director, NICHD, NIH
2017-2020 Acting Director, Division of Intramural Population Health Research (DIPHR), NICHD, NIH
2020-2021 Senior Investigator & Chief, Section on Endocrinology & Genetics, NICHD, NIH

Editorial (selected):

2000-2005 Editor-in-Chief, *Journal of Endocrine Genetics*; Associate Editor, *Endocr. Rel. Cancer* (2004-2011)
2000-present: Editorial Boards: *J. Clin. Endocrinol. Metab*; *J. Pediatr. Endocrinol;* other; Reviewer for >120 journals;
2010-2014 Deputy Editor, *J. Clin. Endocrinol. Metab*; 2015-2016, Associate Editor, *Endocrine Reviews*.
2015-present: co-Editor-in-Chief, *Hormone and Metabolic Research*.
2017-present: Editor, *Molecular and Cellular Endocrinology*

Elected memberships (selected):

2005 Society for Pediatric Research (SPR)
2009 American Society for Clinical Investigation (ASCI)
2013 American Pediatric Society (APS)
2018 Association of American Physicians (AAP)

Honors & Awards (selected):

1989 & `94 Degrees (MD & DmedSc) *summa cum laude,* Class Valedictorian*;* 1983–89 IKY Scholarship (Grk Foundation) (
1990 Young Investigator Award "Sp. Pitoulis"; Greek Endocrine Society
1993 Diplomate, Am. Board of Pediatrics; Resident of the year, Georgetown University Hospital
1995 Diplomate, Am. Board of Pediatrics, SB: Pediatric Endocrinology; Acad. Excellence Award, NIH
1996 Diplomate, Am. Board of Med. Genetics: Certification in Clinical Genetics & Dysmorphology
1999 Endocrine Society (USA)-Pharmacia Award for Excellence in Published Clinical Research
2000, 2005 National Institutes of Health Merit Awards
2007 National Institutes of Health (NICHD) Merit Award
2009 Endocrine Society, Ernst Oppenheimer Award
2013 Dr. Honoris Causa, Université de Liège**,** Belgium
2013, 2014 NIH Group Awards: Intramural-Extramural Collaborations; Support of DSD Research
2015 NICHD Mentor of the Year Award
2015 Nominated, Distinguished Clinical Teacher of the Year Award, CRC, NIH
2015 Society for Endocrinology – Endocrine-Related Cancer Journal, Published Author of the Year Award
2016 Elected, Vice President (2017); President-Elect (2018), Society for Pediatric Research
2016 Distinguished Physician 2016 Award, Hellenic Medical Society of New York
2018 Dr. Honoris Causa, University of Athens, Greece
2018 European Society for Pediatric Endocrinology, International Award for Research in Endocrinology
2019 Dale Medal for lifetime contributions to Endocrine Research, from the Society for Endocrinology (UK)

C. Contribution to Science

There are four areas of science that my laboratory and clinical research work have contributed significantly to:

1. The clinical and molecular elucidation of diseases (presentation, diagnosis and molecular investigations) involving the adrenal gland

From being involved in the identification of the first mutations in the human glucocorticoid receptor gene as a postdoctoral fellow in George Chrousos' lab in 1989, to the most recent identification of genes in Cushing syndrome (2013-2015), I have been involved in or made some of the most important discoveries in the clinical and molecular genetics of adrenal diseases over the last three decades. My laboratory found the genetic defects leading to Carney complex and primary pigmented nodular adrenocortical disease (PPNAD) or variants thereof (*PRKAR1A*, *PRKACA*, *PRKACB*), described a new form of adrenocortical hyperplasia which we named "isolated micronodular adrenocortical disease" (iMAD) and other related pathologies (primary bimorphic adrenocortical disease or PBAD and others), studied Algroove syndrome and other genetic forms of adrenocortical insufficiency, and was one of the collaborating labs in identifying *ARMC5*, the gene for primary macronodular adrenocortical hyperplasia (PMAH). We were the first to show mutations in PMAH and reported widely used clinical tests today for the diagnosis of PPNAD, iMAD, and PMAH. We have also studied extensively the genetics of primary hyperaldosteronism and endocrine hypertension. Finally, in collaboration with other groups we have investigated diseases of the adrenal medulla, including pheochromocytomas and related lesions, especially those linked to succinate dehydrogenase (SDH) defects.

- Stratakis CA, Sarlis NJ, Kirschner LS, Carney JA, Doppman JL, Chrousos GP, Papanicolaou DA. Paradoxical response to dexamethasone in the diagnosis of primary pigmented nodular adrenocortical disease (PPNAD). ***Ann Intern Med 131(8):585-591, 1999***. (PMID 10523219)
- Kirschner LS, Carney JA, Pack SD, Taymans SE, Giatzakis C, Cho YS, Cho-Chung YS & CA Stratakis. Mutations of the gene encoding the protein kinase A type I-alpha regulatory subunit in patients with the carney complex. ***Nat Genet. 26(1):89-92, 2000.*** (PMID 10973256)
- Beuschlein F*, Fassnacht M*, Assié G*, Calebiro D*, Stratakis CA*, Osswald A, Ronchi CL, Wieland T, Sbiera S, Faucz F, Schaak K, Schmittfull A, Schwarzmayr T, Barreau O, Vezzosi D, Rizk-Rabbin M, Zabel U, Szarek E, Salpea P, Forlino A, Vetro A, Zuffardi O, Kisker C, Diener S, Meitinger T, Lohse MJ, Reincke M, Bertherat J, Strom TM, Allolio B. Constitutive activation of *PRKACA* in adrenal Cushing's syndrome. ***N Engl J Med. 370(11):1019-28, 2014.*** **first authors with equal contribution* (PMCID 4727447)
- Zilbermint M, Xekouki P, Faucz FR, Berthon A, Gkourogianni A, Schernthaner-Reiter MH, Batsis M, Sinaii N, Quezado MM, Merino M, Hodes A, Abraham SB, Libé R, Assié G, Espiard S, Drougat L, Ragazzon B, Davis A, Gebreab SY, Neff R, Kebebew E, Bertherat J, Lodish MB & CA Stratakis. Primary aldosteronism and *ARMC5* variants. ***J Clin Endocrinol Metab. 100(6):E900-9, 2015.*** (PMCID 4454793)
- Gourgari E, Lodish M, Keil MF, Sinaii N, Turkbey E, Lyssikatos C, Nesterova M, Sierra LM, Xekouki P, Khurana D, Ten S, Dobs A & CA Stratakis. Bilateral adrenal hyperplasia as a possible mechanism for hyperandrogenism in women with polycystic ovary syndrome. ***J Clin Endocrinol Metab. 101(9):3353-3360, 2016***. (PMCID 5010568)

2. The clinical and molecular elucidation of diseases (presentation, diagnosis and molecular investigations) involving the pituitary gland

The work on the pituitary gland started as complementary to the one on the adrenal gland: as we were studying familial cases of syndromes predisposing to adrenocortical tumors, we came across patients and families with predisposition to pituitary tumors. First, we studied patients with multiple endocrine neoplasia type 1 (MEN 1) and Carney complex (CNC) where we found the molecular defects leading to their growth-hormone (GH)-secreting tumors. This led to us collecting over the years an international cohort of patients with GH-secreting tumors that did not have MEN 1 or CNC. We were the first to describe succinate dehydrogenase (SDH) defects leading to pituitary tumors. Recently, our laboratory identified a new condition that we named X-LAG for "X-linked acrogigantism" in patients with GH-producing hyperplasia or tumors. We then identified the genetic defect in X-LAG which also appears to explain more than 80% of sporadic cases of early gigantism. The defect involves the overexpression of an orphan G-protein coupled receptor (GPCR) on the X-chromosome, *GPR101;* currently my lab is dedicating substantial amount of resources in the molecular elucidation of GPR101's function. In addition, we studied patients with pediatric Cushing disease (CD) due to corticotropin (ACTH)-secreting pituitary tumors, as part of our studies on Cushing syndrome. It is no exaggeration to say that almost everything that is known today in the literature about pediatric CD, from its molecular investigations to its diagnosis and treatment is derived from work that has been done at the NIH. My laboratory is currently intensely involved in the identification of genetic defects predisposing to pediatric CD.

- Batista DL, Riar J, Keil M & CA Stratakis. Diagnostic tests for children referred for the investigation of Cushing syndrome. ***Pediatrics 120(3):e575-86, 2007***. (PMID 17698579)
- Trivellin G, Daly AF, Faucz FR, Yuan B, Rostomyan L, Larco DO, Schernthaner-Reiter MH, Szarek E, Leal LF, Caberg JH, Castermans E, Villa C, Dimopoulos A, Chittiboina P, Xekouki P, Shah N, Metzger D, Lysy PA, Ferrante E, Strebkova N, Mazerkina N, Zatelli MC, Lodish M, Horvath A, de Alexandre RB, Manning AD, Levy I, Keil MF, Sierra Mde L, Palmeira L, Coppieters W, Georges M, Naves LA, Jamar M, Bours V, Wu TJ, Choong CS, Bertherat J, Chanson P, Kamenický P, Farrell WE, Barlier A, Quezado M, Bjelobaba I, Stojilkovic SS, Wess J, Costanzi S, Liu P, Lupski JR, Beckers A & CA Stratakis.

Gigantism and acromegaly due to Xq26 microduplications and GPR101 mutation. ***N Engl J Med. 371(25):2363-74, 2014***. (PMCID 4291174)

- Xekouki P, Szarek E, Bullova P, Giubellino A, Quezado M, Mastroyannis SA, Mastorakos P, Wassif CA, Raygada M, Rentia N, Dye L, Cougnoux A, Koziol D, de La Luz Sierra M, Lyssikatos C, Belyavskaya E, Malchoff C, Moline J, Eng C, Maher LJ Third, Pacak K, Lodish M & CA Stratakis. Pituitary adenoma with paraganglioma / pheochromocytoma (3PAs) and succinate dehydrogenase defects in humans and mice. ***J Clin Endocrinol Metab. 100(5):E710-9, 2015*.** (PMCID 4422891)
- Beckers A, Lodish M, Giampaolo T, Rostomyan L, Lee M, Faucz FR, Yuan B, Choong C, Caberg JH, Verrua E, Naves LA, Cheetham T, Young J, Lysy P, Petrossians P, Cotterill A, Shah N, Metzger D, Castermans E, Ambrosio MR, Villa C, Strebkova N, Mazerkina N, Gaillard S, Barcelos Barra G, Casulari LA, Neggers S, Salvatori R, Jaffrain-Rea ML, Zacharin M, Lecumberri Santamaria B, Zacharieva S, Lim EM, Mantovani G, Zatelli MC, Collins MT, Bonneville JF, Quezado M, Chittiboina P, Oldfield E, Bours V, Liu P, de Herder WW, Pellegata NS, Lupski JR, Daly AF & CA Stratakis. X-linked acrogigantism (X-LAG) syndrome: clinical profile and therapeutic responses. ***Endocr Relat Cancer. 22(3):353-67, 2015***. (PMCID 4433400)
- Trivellin G, Bjelobaba I, Daly AF, Larco DO, Palmeira L, Faucz F, Thiry A, Leal LF, Rostomyan L, Quezado M, Schernthaner-Reiter MH, Janjic MM, Villa C, Wu TJ, Stojilkovic SS, Beckers AF, Feldman B & CA Stratakis. Characterization of GPR101 transcript structure and expression 1 patterns. ***J Mol. Endocrinol. 57(2):97-111, 2016***. (PMCID 4959428)

3. Molecular investigations and animal models of the c-AMP-dependent protein kinase (PKA)

This work focuses on the molecular elucidation of PKA-related defects and their contribution to human disease. It all started with our identification of the genetic defect leading to Carney complex: *PRKAR1A* mutations were the first PKA genetic defects to be found associated with any human disorder. PRKAR1A is the main regulatory subunit of PKA, essentially the cAMP "receptor" in cells. We stayed in the field, as it turns out that almost any disorder we studied was linked one way or another to cAMP signaling (including the latest one, GPR101 defects). We maintain a database of *PRKAR1A* mutations that is freely available to the public: http://prkar1a.nichd.nih.gov/hmdb/intro.html. Floxed *Prkar1a*$^{+/-}$ animal models made in my laboratory addressed the participation of *PRKAR1A* in endocrine cell growth and neoplastic development of other tissues, such as the bone. In follow up work, my laboratory identified cAMP-binding phosphodiesterase (PDE) genes *PDE11A* and *PDE8B* as potentially being involved in cAMP-related tumors and we studied the respective animal models, work that is ongoing. Our animals are freely shared with investigators in the field. Our ongoing work aims at identifying the contributions of the other PKA subunits in human disease, from PRKACA and PRKACB as oncogenes to the protective effects of PRKAR2A in diet-induced obesity.

- Kirschner LS, Kusewitt DF, Matyakhina L, Towns II WH, Carney JA, Westphal H & CA Stratakis. A mouse model for the Carney complex tumor syndrome develops neoplasia in cyclic AMP-responsive tissue. ***Cancer Res. 65:4506-14, 2005*.** (PMID 15930266)
- Horvath A, Boikos S, Giatzakis C, Robinson-White A, Groussin L, Griffin KJ, Stein E, Levine E, Delimpasi G, Hsiao H-P, Keil M, Heyerdahl S, Matyakhina L, Libe R, Fratticci A, Kirschner LS, Cramer K, Gaillard RC, Bertagna X, Carney JA, Bertherat J, Bossis I & CA Stratakis. A genome-wide scan identifies mutations in the gene encoding phosphodiesterase 11A4 (*PDE11A*) in individuals with adrenocortical hyperplasia. ***Nat Genet 38(7):794-800, 2006***. (PMID 16767104)
- Tsang KM, Starost MF, Nesterova M, Boikos SA, Watkins T, Almeida MQ, Harran M, Li A, Collins MT, Cheadle C, Mertz EL, Leikin S, Kirschner LS, Robey P & CA Stratakis. Alternate protein kinase A activity identifies a unique population of stromal cells in adult bone. ***Proc Natl Acad Sci USA. 107(19):8683-8, 2010***. (PMCID 2889322)
- London E, Nesterova M, Sinaii N, Szarek E, Chanturiya T, Mastroyannis SA, Gavrilova O & CA Stratakis. Differentially regulated protein kinase A (PKA) activity in adipose tissue and liver is associated with resistance to diet-induced obesity and glucose intolerance in mice that lack PKA regulatory subunit type-IIa. ***Endocrinology. 155(9):3397-408, 2014*.** (PMCID 4138573)
- Liu S, Saloustros E, Berthon A, Starost MF, Sahut-Barnola I, Salpea P, Szarek E, Faucz FR, Martinez A, & CA Stratakis. Celecoxib reduces glucocorticoids *in vitro* and in a mouse model with adrenocortical hyperplasia. ***Endocr Relat Cancer. 23(1):15-25, 2016***. (PMCID 4659722)
- Saloustros E, Liu S, Mertz EL, Bhattacharyya N, Starost MF, Salpea P, Nesterova M, Collins M, Leikin S, & CA Stratakis. Celecoxib treatment of fibrous dysplasia (FD) in a human FD cell line and FD-like lesions in mice with protein kinase A (PKA) defects. ***Mol Cell Endocrinol. 439:165-174, 2017***. (PMCID 5123938)

4. Molecular elucidation of diseases (from clinical presentation to diagnosis and molecular investigations) caused by succinate dehydrogenase (SDH) defects

We studied SDH defects almost by accident but some of my most widely cited work is from this field of research. A decade ago, I came across sporadic cases of a very rare condition called Carney Triad that was very attractive to me because of its unique combination of adrenocortical (adenomas and hyperplasia) and adrenomedullary (pheochromocytoma) tumors. The same patients developed gastrointestinal stromal tumors (GIST) and paragangliomas. We were the first to describe that a subset of these patients, in whom the disease is inherited in an autosomal dominant manner, represented a new condition which today bears the name of the investigators involved (Carney-Stratakis syndrome, CSS). We found that CSS is caused by SDH defects. We then identified SDH defects in sporadic cases of the so-called "wild-type" GISTs, in pituitary tumors (see above), and a peculiar, yet pathognomonic epigenetic signature in Carney Triad: *SDHC* promoter methylation (SPM). Today, SDH defects explain almost a fifth of all "wild-type" GISTs providing clues for a new molecular pathway for their treatment and the tests we developed (SDHB immunoreactivity and SPM) are widely used diagnostically for the identification of tumors caused by SDH defects and Carney Triad, respectively. Ongoing work in this field is now done collaboratively with laboratories around the world and the National Cancer Institute.

- McWhinney SR, Pasini B & CA Stratakis. Mutations of the genes coding for the succinate dehydrogenase subunit genes in familial gastrointestinal tumors. ***N Engl J Med. 357(10):1054-6, 2007***. (PMID 17667967)
- Janeway KA, Kim SY, Lodish M, Nosé V, Rustin P, Gaal J, Dahia PL, Liegl B, Ball ER, Raygada M, Lai AH, Kelly L, Hornick JL; NIH Pediatric and Wild-Type GIST Clinic, O'Sullivan M, de Krijger RR, Dinjens WN, Demetri GD, Antonescu CR, Fletcher JA, Helman L & CA Stratakis. Defects in succinate dehydrogenase in gastrointestinal stromal tumors lacking *KIT* and *PDGFRA* mutations. ***Proc Natl Acad Sci U S A. 108(1):314-8, 2011***. (PMCID 3017134)
- Haller F, Moskalev EA, Faucz FR, Barthelmeß S, Wiemann S, Bieg M, Assie G, Bertherat J, Schaefer IM, Otto C, Rattenberry E, Maher ER, Ströbel P, Werner M, Carney JA, Hartmann A, Stratakis CA, Agaimy A. Aberrant DNA hypermethylation of *SDHC*: a novel mechanism of tumor development in Carney triad. ***Endocr Relat Cancer. 21(4):567-77, 2014***. (PMCID 4722532)
- Boikos SA, Pappo AS, Killian JK, LaQuaglia MP, Weldon CB, George S, Trent JC, von Mehren M, Wright JA, Schiffman JD, Raygada M, Pacak K, Meltzer PS, Miettinen MM, Stratakis CA, Janeway KA, Helman LJ. Molecular Subtypes of KIT/PDGFRA Wild-Type Gastrointestinal Stromal Tumors: A Report From the National Institutes of Health Gastrointestinal Stromal Tumor Clinic. ***JAMA Oncol. 2(7):922-928, 2016.*** (PMID27011036)

My NCBI Bibliography is available at https://www.ncbi.nlm.nih.gov/sites/myncbi/constantine.stratakis.1/collections/49816276/public/

D. Research Support & Patents/Licensed Applications

Current (selected; only awards over 100K/year are shown):

- Intramural NICHD project Z01-HD008920: *Molecular genetics of adrenocortical tumors and related disorders*; 12/1998 – present
- INSERM, Paris, France: Co-Investigator with Dr. Jerome Bertherat and others: *Cloning of new genes for Carney complex*, 06/2003 – present.

Patents/Licensed applications

1. U.S. Patent No 6,759,525 Issued July 6, 2004; DHHS reference #E-259-2000/0-US-02, on PROTEIN KINASE A AND CARNEY COMPLEX; co-inventors: CA Stratakis and LS Kirschner.
2. Licensed application on “Mouse Model of Prkar1a Down-regulation.” DHHS E–266–2004/0. 69FR46168; Aug. 2004.
3. U.S. Provisional Patent Application No. 60/761,446 filed 24 Jan 2006 entitled ‘‘*PDE11A* MUTATIONS IN ADRENAL DISEASES’’; DHHS Reference No. E–027–2006/0–US–01; inventor: CA Stratakis.
4. U.S. Provisional Patent Application No. 62/078,517 filed on 12 Nov 2014 entitled “METHOD FOR TREATMENT OF HORMONAL DISORDERS OF GROWTH USING ANTAGONISTS AND AGONISTS OF THE ORPHAN G-PROTEIN COUPLED RECEPTRO (GPCR), GPR101”.
5. U.S. Patent Application No. 4239-104735-01 08/03/20 E-169-2020-0-US-01 “TREATMENT OF HORMONAL DISORDERS OF GROWTH; GPR101 Ligands.