Exhibit 524

Expert Report of David A. D'Alessio, M.D.

Scott Keller v. United States

7:23-cv-01501

United States District Court for the Eastern District of North Carolina

Prepared By:

David A. D'Alessio, M.D.

April 8, 2025

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Introduction and Summary of Opinions

I was asked by the United States Department of Justice to evaluate whether the treatment that plaintiff Scott Keller received for non-Hodgkin's lymphoma caused him to develop chronic kidney disease and significant cardiomyopathy, as plaintiff's expert Dr. Dean Felsher opines in his February 7, 2025, report.[1] It is my opinion as an endocrinologist that the course of Mr. Keller's chronic kidney disease and worsening ischemic cardiac disease is the unfortunate but common outcome of many patients who, like Mr. Keller, have type 2 diabetes mellitus (T2DM). While Mr. Keller had other risk factors for chronic kidney disease, the presence of diabetes was likely central to the process and a major contributor to his eventual development of end-stage renal disease and ischemic cardiomyopathy.

This report is based on review of records for Mr. Keller from 1998-2024. Most of these records are from inpatient treatment with only scattered outpatient records. In addition, there are large gaps in the medical records that I was provided with (2001-12 and 2014-18), and therefore, some of the information used in this opinion is based on secondary sources (the deposition of Dr. Jennifer Leach and some of her chart notes). Included in this time gap is the onset and early course of Mr. Keller's diabetes and renal dysfunction that became major causes of morbidity. Thus, the views expressed here are based on established milestones in his clinical course – exposure to toxicants at Camp Lejeune, treatment of non-Hodgkin Lymphoma (NHL) with autologous stem-cell transplant (SCT; 1998-2001), diagnosis of T2DM and chronic kidney disease (CKD; 2014), diagnosis of ischemic coronary disease (ICD) with cardiomyopathy (2020-24), and progression to end-stage renal disease (ESRD) with dialysis (2024), connected by his most consistent source of longitudinal records (the outpatient documents from Providence SE Washington Nephology starting in 2012). In determining the cause of Mr. Keller's chronic kidney disease and cardiomyopathy, I considered and weighed the risk factors for these conditions through a differential diagnosis, to determine the most likely cause, which is Mr. Keller's diabetes.

Qualifications and Compensation

I am currently employed as the Lindquist Distinguished Professor of Medicine at the Duke University School of Medicine and am the Director of the Duke Division of Endocrinology. I received an M.D. from the University of Wisconsin and did residency and fellowship training at Temple University and the University of Washington. I am board certified in Internal Medicine and Endocrinology. After finishing training, I joined the faculty at the University of Washington where I advanced to the rank of Assistant Professor. I served as clinic director for the University of Washington, was a division leader in teaching students and residents and ran a research program focused on pathogenic mechanisms of diabetes. I moved to the University of Cincinnati after 13 years in Seattle, and over my 14-year tenure at the University of Cincinnati advanced to full Professor and Chief of Endocrinology at University Hospital and the Cincinnati VA Medical Center. In 2014, I was recruited by Duke University to be Head of Endocrinology and bring my research lab to the Duke Molecular Physiology Institute.

I have practiced endocrinology for 35 years with a focus on diabetes care. Over this time, I have treated thousands of people with diabetes as an inpatient consultant and in my outpatient practices where

[1] Dr. Felsher's report is dated February 7, 2024, but this appears to be a typographical error, as the report "incorporate[s] by reference my general causation report, including all of the opinions contained therein and the materials considered list, submitted in this case on December 9, 2024." Felsher p. 11.

I follow many patients for years as their endocrinologist. I served on the practice guideline committee for the American Diabetes Association preparing the 2018-20 Standards of Care, the major source of clinical recommendations for the field. I have written chapters for major textbooks on the pathogenesis, diagnosis and treatment of diabetes. My research lab has been continuously funded from federal and foundation sources since 1995, and I have published more than 300 research and clinical papers in peer-reviewed journals. Because of my research in the regulation of insulin secretion, I have been asked to serve as a consultant for commercial entities involved in diabetes drug development. Presently, I am co-chair of two multi-center clinical trials for novel diabetes medications. I served on grant panels for the NIH, American Heart Association, and American Diabetes Association from 1999-2017 and have been on the Council at the National Institute for Diabetes and Digestive and Kidney Diseases since 2018. I am currently Editor-in-Chief of the journal *Diabetes*, the flagship research journal of the American Diabetes Association.

My current practice in endocrinology is at Duke Hospital and the Durham VA Medical Center, where I see patients 3 days per week. For this consultation, I receive $600 per hour for chart review, meetings and other preparation, and $700 per hour, or $3500 per half day, for court appearances or depositions. I have not testified at trial or deposition in any case within the last four years.

Focused Medical History

Mr. Keller was diagnosed with non-Hodgkin's lymphoma in February 1998 and received initial chemotherapy under the care of oncologist Stephen Iacoboni, MD, in Walla Walla, Washington. He was referred to the University of Washington by Dr. Iacoboni in the spring of 1998 and seen by Dr. Steven Petersdorf, a medical oncologist, and Dr. Karen Lindsley, a radiation oncologist. They recommended chemotherapy, systemic radiation therapy and a stem-cell transplant as his best treatment option to cure his lymphoma. He was admitted to University Hospital in Seattle from April 29 to May 2, 1998 for mobilization chemotherapy. On admission, his creatinine, a basic measure of renal function, was 1.0, a value normal for an adult; this value remained unchanged on May 1, the day before he was discharged. He was readmitted June 2 through June 29, 1998 for a stem cell transplant. On admission his creatinine was 0.9 and was stable until June 18, when he had a transient rise to 2.4 on June 20, which resolved to a level of 1.0 by June 22; creatinine was 0.9 on discharge. At the conclusion of his primary treatment for NHL, he had normal renal function and normal blood pressure, and overall, his post-transplant course was stable with a gradual return to health.

Mr. Keller was admitted to St. Mary's Hospital in Walla Walla in February 1999 for bronchitis. He was treated with solumedrol and prednisone, anti-inflammatory steroids. His creatinine was 1.1 during admission and he had mildly elevated blood glucose levels of 137-186 while receiving steroids. Dr. Iacoboni saw Mr. Keller after SCT for 3 years before concluding that he was likely cured from lymphoma and discharging him from regular care. During an outpatient visit in April 2000, Mr. Keller was noted to have a normal creatinine; in July 2003, his creatinine was noted to be 1.4. In August 2008, Mr. Keller was seen in the St. Mary's emergency room with knee swelling; at this time, his chart lists a diagnosis of diabetes and his medication list includes glimepiride, a commonly used medication to treat diabetes.

In 2012, Mr. Keller was referred to Dr. Jennifer Leach, a nephrologist at PMG SE Washington in Walla Walla, for stage 3 CKD. The record indicates that this diagnosis was made in August 2012, but there are no records of that August encounter or the clinical factors used to establish this. There are

orders for blood and urine testing by SE Washington Nephrology for October 6 and November 2, 2012 but no documentation of a clinic visit. On November 6, there is a progress note listed as "Nephrology Follow up Visit" with Dr. Leach. At that time, Mr. Keller had an estimated glomerular filtration rate of 43 cc/minute and laboratory results compatible with chronic kidney disease (e.g. secondary hyperparathyroidism and anemia); this level of eGFR indicates moderate renal impairment and is consistent with the designation of stage 3 CKD. During this visit, Mr. Keller had a primary complaint of joint pain that was persistent, had been previously ascribed to gout, and was treated with colchicine; he had a history of using steroid and non-steroidal anti-inflammatory drugs (NSAIDs) to treat joint pain, which was a major complaint at the time of this visit. Dr. Leach noted a diagnosis of T2DM dating from 2009, but there are no medical records available with details of his initial presentation with diabetes and his early progression with this condition. There is a comment in Dr. Leach's notes from November 2012 that hemoglobin A1c was above goal and required attention; hemoglobin A1c is the common measure of chronic blood glucose level and is used to assess the effectiveness of diabetes management. The diabetes treatment listed in his medical records is glimepiride, and he was also treated with rosuvastatin for hypercholesterolemia.

Mr. Keller was seen by Dr. Leach on May 9, 2013. At that time his renal function had declined as indicated by an eGFR of 32, and his uric acid level was elevated to > 10. He was encouraged to stay hydrated and avoid NSAIDs. Because his last hemoglobin A1c measurement had been elevated (8.6%), he was counseled on the risk hyperglycemia presented to his kidneys; at that time he was taking glimepiride and sitagliptin to treat diabetes. Dr. Leach repeated laboratory testing on Mr. Keller and discussed the results with him by phone May 23, 2013; his kidney function had improved to a level similar to November 2012, but his diabetes control had worsened with an A1c of 10.7%. At his next visit with Dr. Leach in August 2013, his eGFR was a stable at 45 and he had no new complaints; his blood pressure was elevated (144/96), but his joint pain had decreased. When seen in November 2013, Mr. Keller had an eGFR of 37, a blood glucose of 430 and modestly elevated albumin in his urine. Mr. Keller's weight was stable at 240 lbs. and BP was 120/80. These measures were comparable in March 2014. Mr. Keller was scheduled to see Dr. Leach in April and June 2015, but there are no records of those visits in the PMG SE Washington Nephrology charts.

Mr. Keller next saw Dr. Leach in October 2018. In the interim, he had been diagnosed with macular degeneration in June 2014 and was being seen by an ophthalmologist. He had been admitted to the hospital for sepsis in 2016, olecranon bursitis later that year, and pneumonia with respiratory failure in 2017. At his October 2018 visit with Dr. Leach, his eGFR was mildly reduced to 32 with moderate proteinuria. At this time his chronic renal failure was attributed to diabetes and he was started on lisinopril to reduce protein excretion and treat his elevated blood pressure, which is a standard treatment for patients with diabetic nephropathy. At this time, he was taking allopurinol to reduce circulating uric acid and simvastatin for hypercholesterolemia. He had stopped glimepiride and sitagliptin and was taking both long- and short-acting insulins for diabetes. His most recent A1c is reported as < 7% in the chart notes from the October 2018 visit with Dr. Leach.

Mr. Keller returned to see Dr. Leach in May 2019. His eGFR was 30, BP 120/68 and weight was 235 lbs. (BMI 35). It was noted at this visit that he had been diagnosed with gout in 2006 on the basis of arthrocentesis of an inflamed elbow; he was free of joint pain and continued taking allopurinol. In

November 2019, his eGFR was 29 with persistent proteinuria, although the amount was reduced since starting lisinopril. His blood pressure was 130/72 and BMI was 37. He had been referred to an endocrinologist for diabetes care. In May 2020, he returned to Dr. Leach. A diagnosis of peripheral neuropathy was new to his problem list and since his last visit he had been switched from insulin to semaglutide for his diabetes treatment. His eGFR was 26 and blood pressure was unchanged, but he had developed a low magnesium level thought to be due to abnormal renal excretion. In November 2020, his renal function was stable and his medications were unchanged. At his May 2021 visit, his eGFR was noted to be 30, weight was 218 lbs., and blood pressure 108/80. He had had an episode of syncope in April 2021 and had a pacemaker placed for heart block.

At his next visit to see Dr. Leach, in June 2022, Mr. Keller had further reduction of his eGFR to 27; his weight and BP were unchanged. He had had a respiratory infection with some evidence of reactive airways disease and was referred to a pulmonary specialist. This is the first time that diabetic retinopathy appears on Mr. Keller's problem list. He was seen by Dr. Leach again in July 2022 following admission to the hospital for an episode of sepsis associated with an acute worsening of renal function; his eGFR had recovered to 22 since discharge but had been lower during the hospitalization. Because of persistent edema, Dr. Leach started Mr. Keller on furosemide. At his visit in October 2022, Mr. Keller had no new complaints. He had just been diagnosed with coronary artery disease (CAD) and had a stent placed. His eGFR was 15 and a diagnosis of heart failure is included in his problem list; on echocardiogram, his ejection fraction was 30%, BP was 102/78 and weight 204 lbs. By November 2022 Mr. Keller's eGFR had decreased to 12; he weighed 170 lbs. after treatment with diuretic medication to reduce the edema attributed to his heart and kidney failure. Later that same month, he was admitted to the hospital with an upper GI bleed and influenza A infection, causing another bout of acute on chronic renal failure. By December 2022, his renal function had recovered to an eGFR of 21. He was no longer taking medication to treat diabetes because his blood glucose was only mildly elevated. When he saw Dr. Leach in February 2023, his eGFR was down to 15 and he and Dr. Leach discussed when dialysis would be necessary. In March 2023, his eGFR was 12 and his weight had increased to 184 lbs. due to fluid retention. Mr. Keller was stable through April, May, and June 2023 and applied for medical retirement. His eGFR decreased to 11 by July 2023, and he discussed dialysis options and access with Dr. Leach. His cardiac and renal failure was stable through the remainder of 2023, but by March of 2024, Mr. Keller had begun dialysis treatment.

Opinion

Scott Keller had chronic renal failure, with steady worsening from 2012 through the present, that is clearly documented in the notes from SE Washington Nephrology. Chronic renal failure increases the risk of coronary vascular disease, his other major co-morbidity (refs 1-5). At the time of his CKD diagnosis, Mr. Keller does not appear to have undergone a kidney biopsy to try to establish a cause(s) for his renal disease; e.g. diabetic nephropathy has diagnostic features that can be determined on microscopic examination of biopsy specimens. Thus, ascribing Mr. Keller's CKD diagnosis in 2012 to his lymphoma treatment was only speculative. Moreover, there was no evidence from his basic laboratory tests that his kidney function had declined in the 3 years after chemotherapy and SCT, which would be expected if these were the cause of eventual CKD.

From 2000 until 2012 there is insufficient testing to document Mr. Keller's evolution from normal renal function in 2001 to stage 3 CKD in 2012. While he was exposed to factors that could affect his kidney function during his treatment of NHL – e.g. sepsis and hypotension, nephrotoxic drugs, and systemic radiation – he did not have significant deficits in renal function either during or in the 2-3 years after SCT. Plaintiff's expert Dr. Felsher opines that Mr. Keller's renal disease was attributed to chemotherapy and NHL, but Mr. Keller had multiple other risk factors that are better established as causes of CKD. Important among these are hyperuricemia, use of NSAIDs, dyslipidemia and atherosclerosis, obesity, and diabetes (refs 6 -10). Moreover, the time course of his renal disease, first mentioned in his records more than 13 years after his cancer treatment, is more compatible with these other etiologies for kidney failure.

Diabetes is the leading cause of CKD in the U.S., with 10-40% of persons with T2DM eventually developing kidney failure (ref 11). While the specific features of diabetic nephropathy were previously held to be discrete, i.e. early onset of proteinuria/albuminuria with a distinctive renal biopsy picture (Kimmelstiel-Wilson disease) and progression over 5-10 years to ESRD, this stereotypic course is not invariable, and is better established for patients with Type 1 diabetes (T1DM). Mr. Keller had T2DM, the more common form of diabetes, and it is now clear that renal disease in this group of patients presents a more heterogeneous pathophysiology and natural history (refs 11-14). In part, this is because Type 2 diabetes is more common in older people who have other risk factors for CKD such as obesity, macrovascular disease, medications and chronic inflammatory illnesses. Approximately 25% of patients with both diabetes and CKD present without prominent proteinuria, although many develop this sign over time. This feature seems to have influenced the physicians caring for Mr. Keller who did not initially attribute his CKD to diabetes but changed the attribution once he developed proteinuria. On the basis of the known and likely causes of CKD, Mr. Keller's diabetes was likely the principal driver of his renal disease.

Mr. Keller had several risk factors for the development of diabetes. He had obesity at the conclusion of NHL treatment with a BMI of > 35 and this persisted for most of the next 20 years. He had mild elevations of his blood glucose during treatment with steroids in 1999, suggestive of prediabetes. There is also a strong family history of diabetes with both his parents diagnosed with T2DM in adulthood. He also had evidence of a dyslipidemia with consistent elevation in serum cholesterol and variable elevation of serum triglyceride, indicative of the abnormal lipid metabolism that frequently accompanies T2DM and has been linked to diabetes risk. Based on his risk profile, particularly persistent obesity and family history, the development of diabetes in his 5th decade is not unusual.

While notes from Dr. Leach date the onset of diabetes in Mr. Keller to "around 2009", and he had diabetes listed in his problem list and was taking glimepiride in 2008, how and when he was first diagnosed with T2DM is not discernible in the records. In fact, the onset of T2DM is frequently difficult to determine, because in many people blood glucose increases gradually over time and is asymptomatic. Thus, it is likely that most people diagnosed with T2DM have had the disease for several years before diagnosis (refs 15-17). Given that Mr. Keller was being treated for diabetes in 2008, it is possible that he had been hyperglycemic for 6-8 years before being noted to have stage 3 CKD in 2012, meaning as early as 2004-2008. His course of diabetic complications was typical for people getting end-organ damage caused by diabetes, with both microvascular (peripheral neuropathy, diabetic retinopathy), and

macrovascular (CAD), disease developing over 5-10 years. Susceptibility to end-organ complications varies among diabetic patients with most of the variance determined by multiple genetic factors conferring different levels of susceptibility. However, glucose-lowering therapy, with achievement of A1c levels of 7% or lower, have been demonstrated to reduce rates of renal, neurologic and retinal disease (refs 18-20). There is not good documentation of glycemic control in the medical records of Mr. Keller, but at the time he started seeing Dr. Leach, his blood glucose control was not optimal.

In summary, the course of CKD and ischemic cardiac disease in Scott Keller follow a course that is typical for many patients with T2DM. While Mr. Keller had other risk factors for CKD, the presence of diabetes was likely central to the process and a major contributor to his eventual development of end-stage kidney disease and ischemic cardiomyopathy.

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20. American Diabetes Association Professional Practice Committee. Chronic Kidney Disease and Risk Management: Standards of Care in Diabetes-2025. Diabetes Care. 2025 Jan 1;48(Supplement_1):S239-S251.

CURRICULUM VITAE

David Andrew D'Alessio, M.D.

1. PERSONAL DATA
   Date of Birth: [redacted] 1957
   Place of Birth: New York, NY
   Address: 758 Old Mill Rd, Chapel Hill, NC 27514

2. EDUCATION
   1975-79 Carleton College, Northfield, MN (B.A., Biology)
   1979-83 University of Wisconsin, Madison, WI (M.D.)

   Postgraduate Training:
   1983-86 Resident, Temple University Hospital, Philadelphia, PA
   1986-87 Research Associate, Division of Metabolism, Temple University
   1987-90 Research Fellow, Division of Endocrinology University of Washington, Seattle, WA

3. ACADEMIC APPOINTMENTS
   1990-94 Acting Instructor, Dept. of Medicine, University of Washington
   1994-96 Acting Assistant Professor, Dept. of Medicine, University of Washington
   1996-99 Assistant Professor, Dept. of Medicine, University of Washington
   1999-2005 Associate Professor, Dept. of Medicine, University of Cincinnati
   2005-2014 Professor, Dept. of Medicine, University of Cincinnati
   2006-2014 Director, Division of Endocrinology, University of Cincinnati
   2014-present Professor Dept. of Medicine, Duke University
   2014-present Director, Division of Endocrinology, Duke University

4. HOSPITAL APPOINTMENTS
   1990-99 Attending Physician, University of Washington Medical Center
   1995-99 Director, Endocrinology Clinic, Univ. of Washington Medical Center
   1999-2014 Staff Physician, Cincinnati VA Medical Center
   1999-2014 Attending Physician, University of Cincinnati Hospital
   2004- 2014 Chief, Endocrine Service, Cincinnati VA Medical Center
   2014-present Attending Physician, Duke University Hospital
   2014-present Attending Physician, Durham VAMC

5. HONORS
   Sol Sherry Award - Clinical Research, Temple University, 1987
   National Research Service Award (NRSA), NIH, 1988-90
   Clinical Associate Physician, Clinical Research Center, University of Washington, 1990-2
   Diabetes Research Council Young Investigator Award, University of Washington, 1990-2
   Clinical Nutrition Research Unit Young Investigator Award, Univ. of Washington, 1992-4
   Clinical Investigator Development Award (K08), NIH, 1994-8
   Howard Hughes Medical Institute Research Resources Program Award, Univ. of Washington, 1997
   American Diabetes Association Clinical Research Award, 1998 - 2001
   Western Society for Clinical Investigation, elected member 1998
   Paul Beeson Award for Clinical Teaching, Univ. Washington, 1998
   Albert Vontz Chair for Diabetes Research, University of Cincinnati, 2000-2014
   Endocrinology Teaching Award, Duke University, 2016
   American Association of Physicians, 2017
   James B Wyngaarden Distinguished Professor of Medicine, 2022

Neil Spector Art of Medicine Award, Duke Department of Medicine, 2023

6. LICENSURE AND CERTIFICATION

Medical Licensure:

Pennsylvania	1983-87
Washington	1987- 99
Ohio	1999-2014
North Carolina	2014-present

Diplomate- American Board of Internal Medicine, 1987
Diplomate- American Subspecialty Board, Endocrinology & Metabolism, 1990

7. ORGANIZATIONS

American Diabetes Association (1990-present)
The Endocrine Society (1998-present)

8. LOCAL RESPONSIBILITIES

Scientific Advisory Committee, Clinical Research Center, Univ of Washington, (1990-1999)
Associate Director, Clinical Core, Diabetes and Endocrine Research Center, Univ of Washington, (1997-1999)
Scientific Advisory Committee, General Clinical Research Center, Cincinnati Children's Hospital (1999-2011)
Associate Program Director, Clinical Research Center, Cincinnati VA Medical Center (2002-2003)
Co-Director, Clinical Research Center, Cincinnati VA Medical Center (2003-2006)
Fellowship Director, University of Cincinnati Division of Endocrinology (2004-2009)
Associate Program Director, Cincinnati Children's Hospital GCRC (2005-2010)
Director, Clinical Research Unit, Cincinnati VA Medical Center (2006-2014)
Associate Director, Duke Molecular Physiology Institute (2014-2022)
PI, NIH T32 Endocrinology and Metabolism Training Program, Duke University (2017-present)
Site PI, NIH P30 North Carolina Diabetes Research Center, Duke University (2019-present)

9. NATIONAL SERVICE

American Federation for Medical Research, National Councilor (1998-2002), Secretary-Treasurer (2002).
American Diabetes Association- Grants Review Committee, 1997-2000.
NIH NCRR, ad hoc reviewer GCRC 2000-2005.
Councilor-at-large, AFMR, 1998-2000; Secretary-treasurer 2000-2002
Endocrine Society, Continuing education subcommittee; ESAP contributor (2002-2012); Board Review (2013-14).
American Heart Association Grants Review Committee, (2003-07).
NIH ad hoc reviewer, SBIR, Training Grant panels (2003-6).
Veterans Administration Medical Research Grants reviewer, ad hoc ENDA (2006-present).
NIH, CIDO study section, standing member (2005-present; Chair 2010-11).
NIH, ad hoc reviewer, CADO study section (2014-2015), SEP (2006-2013).
American Diabetes Association, Program committee (2012-2015; Chair 2013-15).
American Diabetes Association, Pathways grant review and advisory committee (2017-present).
American Diabetes Association, Standards of Care committee (2018-2020).
Endocrine Society, Program Committee (2014-2019; Clinical Research Chair 2019).
Endocrine Society, Laureate Committee (2018-2021)
NIH/NIDDK Council, Ex Officio-VA representative (2017-present)

10. EDITORIAL SERVICE

Reviewer: *American Journal of Physiology, Cell Metabolism, Cell, Diabetes, Diabetes Care, Diabetologia, Endocrinology, Gastroenterology, Journal of Clinical Endocrinology and Metabolism, Journal of Clinical Investigation, Science, Nature Medicine, Nature Communications.*
Associate Editor- *Journal of Investigative Medicine* (2000-2002)
Issue Editor- *Current Opinion in Clinical Nutrition and Metabolic Care* (2004-2010)
Editorial Board- *JCEM (2006-2010; 2011-2015); American Journal of Physiology, Endocrinology and Metabolism (2007-2015); Diabetes (2010-2015), Endocrinology (2010-2015); Journal of Clinical Investigation (2014-present).*
Associate Editor- *Diabetes Care* (2015-2021)
Associate Editor- *Diabetes* (2016-2021)
Editor-in-Chief- *Diabetes* (2021-present)

11. PUBLICATIONS

I. Reviewed Articles

1. Owen OE, Holup JS, **D'Alessio DA**, Craig ES, Polansky M, Smalley KJ, Kavle EC, Bushman MC, Owen LR, Mozzoli MA, Kendrick ZV, Boden GH. A reappraisal of the caloric requirements of men. Am J Clin Nutr 46:875-885, 1987.
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II. Editorials and Reviews

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III. Book Chapters

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12. ONGOING FUNDED RESEARCH PROJECTS:

R01-DK122423
Campbell/D'Alessio (MPI)
10/01/2025 - 09/31/2029
Alpha-to-Beta cell communication in health and disease

R01-DK141090
Campbell/D'Alessio (MPI)
02/01/2025 - 1/31/2029
Metabolic Actions of Glucagon

R01-DK125353
Campbell/D'Alessio (MPI)
07/01/2020 - 05/31/2025
Novel metabolic actions of GIP

P30-DK124723
McClain (Contact PI), Role: Site PI
04/07/2020 - 03/31/2025
North Carolina Diabetes Research Center

13. RECENTLY COMPLETED RESEARCH PROJECTS

R01-DK123075
Campbell (PI), Role: co-Investigator
09/01/2019 - 08/31/2024
Mechanisms of insulin secretion mediated by alpha cells

R01-DK124276
Chilkoti (PI), Role: Co-Investigator
12/10/2020 - 11/30/2024
Injectable PEG-like Conjugate for Sustained Delivery of a Peptide Drug for Type 2 Diabetes Treatment

R01-DK101991
D'Alessio (PI)
09/10/2014 - 06/30/2021
Incretin Action in Physiology and Diabetes

R01-HL130234
Ingram (PI)
09/18/2017 - 06/30/2021
Mechanisms that Direct Airway Remodeling in Obese Asthma