



**Testimony
Before the
Special Committee on Aging
United States Senate**

**“Redefining Reality: How the Special Diabetes
Program is Changing the Lives of Americans
with Type 1 Diabetes”**

Statement of

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Chairman Collins, Ranking Member Casey, and distinguished Members of the Committee, thank you for your invitation to testify at this hearing on type 1 diabetes. I am Griffin P. Rodgers, M.D., M.A.C.P., Director of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), which is one of the 27 Institutes and Centers of the National Institutes of Health (NIH) within the U.S. Department of Health and Human Services (HHS). It is my great honor to be here today to tell you about some of the significant recent scientific progress and future research opportunities in type 1 diabetes and its complications, including research supported by the *Special Statutory Funding Program for Type 1 Diabetes Research (Special Diabetes Program)*.

Diabetes takes an enormous personal and economic toll on our country, but we are making great strides in efforts to reduce that burden through the support of biomedical research. As such, NIH invests more than \$1 billion a year in diabetes research, including studies on type 1 diabetes, type 2 diabetes, gestational diabetes, and diabetes complications; NIDDK supports the majority of diabetes research at NIH. The NIH investment includes funding from the *Special Diabetes Program*, which has enabled the agency to undertake challenges in type 1 diabetes beyond what we could support with our regular appropriations, and to conduct certain types of trials, like comparative effectiveness trials and trials of generic drugs, that were unlikely to have been conducted by the private sector. The NIH investment in combating type 1 diabetes has been complemented by the support and efforts of our research partners—academic institutions, the U.S. Food and Drug Administration (FDA), the Centers for Disease Control and Prevention (CDC), and charitable and patient advocacy groups such as the JDRF, the American Diabetes Association (ADA), and the Leona M. and Harry B. Helmsley Charitable Trust.

Through the invaluable support of Congress, through collaborative and coordinated research efforts, through the hard work of our scientists, and through the dedication of our clinical research volunteers, we have made important progress toward our goals of understanding, preventing, treating, and ultimately curing type 1 diabetes.

ALLEVIATING THE BURDEN OF MANAGING TYPE 1 DIABETES

It is imperative that the research we support ultimately reach and benefit the public, so I am excited to share with you how our investments are paying off. As you know, management of type 1 diabetes is extremely burdensome. Because their pancreatic insulin-producing beta cells have been destroyed by the immune system, people with type 1 diabetes—or the parents of young children with the disease—must do the work of the lost beta cells by monitoring blood glucose levels and administering insulin. Since I last testified before this Committee 2 years ago, several new continuous glucose monitors (CGMs)—devices that automatically track blood glucose levels throughout the day and night—have been approved by the FDA. These include: the first CGM that does not require fingerstick calibration;¹ the first fully interoperable CGM designed to be used as part of an integrated system with other compatible medical devices and electronic interfaces, which also does not require fingerstick calibration;² and the first fully implantable CGM.³ These devices not only make management easier today, but they are also key steps in the development of tomorrow's technologies. I am pleased to report that NIDDK-

¹ <https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm577890.htm>

² <https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm602870.htm>

³ <https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm611454.htm>

or *Special Diabetes Program*-supported research contributed to the development or testing of each of these devices.

We are also supporting other promising research that could help alleviate the burden of managing type 1 diabetes. For example, an NIDDK-supported small business is developing an improved formulation of glucagon, which is a hormone that raises blood glucose levels (as opposed to insulin, which lowers them). People with type 1 diabetes may need to administer glucagon in an emergency when their blood glucose levels fall dangerously low. Currently, glucagon is available in powder form and must be mixed with liquid right before use. But a new, soluble, stable glucagon formulation under development would be ready-to-use in a rescue pen. Such a device could make it less burdensome for patients and caregivers, such as school personnel, to administer glucagon in an emergency.

DEVELOPING BETTER TECHNOLOGIES TO IMPROVE GLUCOSE CONTROL

While we are extremely excited about this progress, we recognize that there is still work to do to reduce the burden of the disease. Despite these advances in technology, the children here today and people of all ages with type 1 diabetes remain susceptible to dangerous and frightening episodes of hypoglycemia (low blood glucose) and to developing long-term complications that affect their eyes, kidneys, nerves, heart, and other organs. The NIDDK's landmark Diabetes Control and Complications Trial (DCCT) and its follow-up study, the Epidemiology of Diabetes Interventions and Complications (EDIC), demonstrated that intensive blood glucose control, beginning as soon as possible after diagnosis, prevented or delayed the development of these long-term complications. Decades of research through DCCT/EDIC has shown that people with type 1 diabetes can dramatically increase their likelihood of living longer, healthier lives by practicing early, intensive blood glucose management.

The challenge is that intensive blood glucose control is difficult to achieve and maintain. Data from the SEARCH ⁴for Diabetes in Youth study, co-led by CDC and NIDDK, reported that over 70 percent of adolescents with type 1 diabetes had hemoglobin A1c (HbA1c) levels—a measurement of blood glucose levels over time—over the recommended 7.5 percent level for that age group.⁵ SEARCH data also showed that 17 percent of adolescents had HbA1c levels above 9.5 percent, putting them at dangerous risk for long-term complications.⁶ These data emphasize the urgent need to continue to develop new approaches to improve glucose control, and NIDDK has invested significant resources provided by the *Special Diabetes Program* to develop glucose management technologies, including artificial pancreas systems. An artificial pancreas has three components: a glucose-sensing component that measures blood glucose levels and sends data to a computer; an insulin delivery device; and a computer that calculates the amount of insulin needed and instructs insulin delivery based on that calculation, thereby “closing the loop” between glucose sensing and insulin delivery. While research is continuing to show how artificial pancreas devices benefit patients, it is imperative that these devices allow people to live full lives and do activities that they enjoy, including exercise. *Special Diabetes Program*-supported scientists demonstrated that artificial pancreas use in adolescents with type 1

⁴ <https://www.searchfordiabetes.org/dspHome.cfm>

⁵ <https://www.ncbi.nlm.nih.gov/pubmed/19394043>

⁶ <https://www.ncbi.nlm.nih.gov/pubmed/19643434>

diabetes improved blood glucose control and reduced hypoglycemia compared to usual care during extended vigorous outdoor exercise at a ski camp.⁷

NIDDK continues to build on recent successes and to support research at all stages to advance artificial pancreas technology. First, NIDDK is supporting clinical trials that are testing artificial pancreas technologies in larger groups, in wider age ranges, over longer periods of time, and in largely unrestricted conditions. Some of the trials are testing the cutting-edge CGMs that I mentioned earlier in my testimony. For example, some of these trials could advance the goal of having interoperable artificial pancreas components so that newly developed insulin pumps and glucose sensors could be paired with existing algorithms, making it easier and faster to develop next-generation artificial pancreas systems. Additionally, one of the trials is testing artificial pancreas technologies in children potentially as young as 4 years old, which could expand the user population for this technology; the commercially available hybrid artificial pancreas is approved in children ages 7 and older.

Second, NIDDK continues to support research conducted by small businesses to develop innovative technologies to improve the components of artificial pancreas devices. With *Special Diabetes Program* support, small businesses are developing improved glucose sensors, insulin pumps, and formulations of insulin and glucagon, including the glucagon formulation I described earlier. Improved components could help speed the development of more fully automated artificial pancreas technology and make the devices simpler and more user friendly.

Third, NIDDK recognizes that new tools and technologies for type 1 diabetes management will only benefit people if they can use them. Therefore, we also support research to identify the most effective ways to incorporate artificial pancreas technologies into clinical care and how to enhance the usability of these new tools to help patients in their decision making. This includes *Special Diabetes Program*-supported research that is studying glucose management technologies in adults ages 65 years or older, to improve glucose control, quality of life, and the health of older people with type 1 diabetes, as well as research developing an artificial pancreas system that is customized to the individual needs of pregnant women. Such research illustrates an important and unique aspect of artificial pancreas research supported by the *Special Diabetes Program*: we are encouraging research in populations that are understudied by industry, such as children and adolescents, older adults, pregnant women, people with poorly controlled blood glucose levels, and people who suffer from frequent, severe episodes of hypoglycemia or who are unaware when their blood glucose levels fall dangerously low. These populations could benefit from artificial pancreas technologies, so we are placing a high priority on supporting research studying the devices in these special populations.

Through these research efforts largely supported by the *Special Diabetes Program*, we are striving to reach our goal of developing multiple different artificial pancreas technologies for people of all ages, so that all people with type 1 diabetes, their caregivers, and their healthcare providers can choose the technology best suited to their needs.

RESTORING BETA CELL FUNCTION

⁷ <https://www.ncbi.nlm.nih.gov/pubmed/28855239>

Artificial pancreas technology represents an important and near-term approach to managing type 1 diabetes, but it is not a cure. Thus, a major, longer-term aim of our research is to identify ways to replace lost beta cells and thereby restore insulin production—a biological cure for the disease. A critical research effort making progress toward this goal is NIDDK’s Human Islet Research Network (HIRN), which receives support from the *Special Diabetes Program*. HIRN is conducting multiple avenues of research to better understand how beta cells are lost in type 1 diabetes and to identify strategies to protect or replace them in people. This includes research to replicate any remaining beta cells or to coax other cell types, such as glucagon-producing alpha cells, into becoming beta cells.

HIRN has been on the leading edge of capitalizing on novel technologies that are allowing us to gain unprecedented insights into the pancreas at a single-cell level. Historically, many biological experiments have been performed on groups of cells, assuming that all cells of a particular “type” are identical. However, we are learning that individual cells within a population may differ dramatically, and these differences can have important consequences in health and disease. In recent studies, HIRN scientists utilized a novel technology, called imaging mass cytometry, to visualize individual beta cells and other pancreatic cell types, as well as immune cells involved in the autoimmune attack, simultaneously in a pancreas.⁸ A surprising finding was that some people newly diagnosed with type 1 diabetes had a similar proportion of beta cells in their pancreas as those without disease, suggesting that even at disease onset when people need to take insulin, their pancreas may still have high numbers of beta cells. This finding indicates that there may be a window of opportunity to protect or replicate these cells. Using this novel technology to characterize individual cells and their interactions in the pancreas could lead to a new understanding of how type 1 diabetes progresses and help to inform the development of new therapies to prevent or treat the disease.

HIRN researchers are also developing an exciting new tool to advance the study of type 1 diabetes: islet chips. (Islets are clusters of beta cells and other cell types found in the pancreas.) These chips are tiny, bioengineered three-dimensional models that support survival and function of human islets in the laboratory setting. These microenvironments incorporate or mimic diverse elements that support islets in the body, such as blood vessels, and are therefore a better representation of human islet physiology than conventional two-dimensional islets grown on plastic dishes. These islet chips are also incorporating immune components and will enable HIRN researchers to study interactions between human beta cells and immune cells to mimic aspects of the autoimmune process involved in type 1 diabetes. They will also serve as a platform for testing novel type 1 diabetes therapies—potentially saving time and money in terms of identifying the most promising therapies to test in people.

Our efforts to develop new and improved approaches for cell replacement therapy have been informed by progress in islet transplantation. Researchers recently reported results from a follow-up study to a Phase 3 trial conducted by the Clinical Islet Transplantation Consortium, which is co-led by NIDDK and the National Institute of Allergy and Infectious Diseases (NIAID), and which tested islet transplantation in people with type 1 diabetes who had persistent impaired awareness of hypoglycemia and frequent severe hypoglycemia events despite expert care. Islet transplant recipients not only reported a decrease in diabetes-related concerns and

⁸ <https://www.ncbi.nlm.nih.gov/pubmed/30713109>; <https://www.ncbi.nlm.nih.gov/pubmed/30713110>

fears, but also felt better overall, despite the need to take daily immunosuppressive drugs to prevent transplant rejection.⁹ These patient-reported outcomes are consistent with the clinical benefits that the trial participants achieved after undergoing islet transplantation.¹⁰ The NIDDK plans to build on progress and continue its strong support of research to find ways to protect or replace beta cells toward curing type 1 diabetes.

UNDERSTANDING THE CAUSES OF TYPE 1 DIABETES TOWARD DISEASE PREVENTION

We are also pursuing research to understand the causes of type 1 diabetes so that we can identify prevention strategies and alleviate the burden of this disease in future generations. For example, we have made significant progress in understanding the genetic contributors to type 1 diabetes: because of NIDDK's Type 1 Diabetes Genetics Consortium and other groups, we know over 50 genes or genetic regions that affect disease risk, representing about 90 percent of the genetic contributors in the White population who have the highest prevalence of the disease. Building on this success, NIDDK is supporting research to understand the function of identified genetic regions to determine how they may influence disease development, which could lead to prevention or treatment targets.

Environmental factors also play a role in type 1 diabetes, though the specific factors responsible have not yet been identified conclusively. The role of environmental factors is underscored by data from the SEARCH study, which has found that the rate of new diagnosed cases of type 1 diabetes is increasing among youth in the United States. SEARCH has also shown that, although type 1 diabetes has historically affected primarily non-Hispanic White youth, the disease is an increasing burden for minorities, such as Hispanic and African American youth considered to be at lower genetic risk. These rising rates of type 1 diabetes suggest that there is an unknown factor—or factors—in the environment that may interact with genetic risk to trigger type 1 diabetes onset. Identifying causative or protective factors—such as an infectious agent, dietary components, or some other factor—is critical to understanding the disease process and to developing prevention strategies.

Toward these goals, NIDDK, through the *Special Diabetes Program*, supports an ambitious, long-term clinical research study called The Environmental Determinants of Diabetes in the Young, or TEDDY. After screening over 425,000 newborns, TEDDY is currently following over 6,000 of them at high genetic risk of type 1 diabetes until they are 15 years old. The biological study samples that have been generously contributed by the dedicated TEDDY families are now being analyzed with genomic, metabolomic, proteomic, and other cutting-edge technologies. Important new insights are already beginning to emerge. TEDDY researchers found that vitamin D levels were lower in infancy and childhood in children who developed autoimmunity, a precursor to type 1 diabetes, and this was particularly true in children who had a specific genetic variant in the vitamin D receptor gene.¹¹ These results highlight how an environmental factor (vitamin D) interacts with an individual's genetic background (vitamin D receptor gene) to affect health and disease—highlighting the importance of both genetic and environmental factors.

⁹ <https://www.ncbi.nlm.nih.gov/pubmed/29563196>

¹⁰ <https://www.ncbi.nlm.nih.gov/pubmed/27208344>

¹¹ <https://www.ncbi.nlm.nih.gov/pubmed/29061729>

Results from TEDDY are also providing insights into childhood health and development in general, specifically new details about how environmental factors affect the microbes in the gut (*i.e.*, the gut “microbiome”) as children age.¹² In one of the largest-ever clinical microbiome studies in infants and children, the researchers discovered that children’s gut microbiome developed in three distinct phases: a developmental phase (3-14 months of age), a transitional phase (15-30 months of age) where the microbiome diversifies, and a stable phase (31-46 months of age) where the microbiome’s composition is largely established. Breastfeeding—even partially—was found to play a crucial role in infants’ gut microbiome development; probiotics, antibiotic use, and other factors also had an effect. Researchers also found a possible beneficial effect on risk for type 1 diabetes from bacteria that produce short-chain fatty acid molecules. These molecules are often made during fermentation of indigestible carbohydrates like fiber, and future research will be needed to determine whether these molecules or the bacteria that produce them protect against type 1 diabetes. These results from TEDDY are just the tip of the iceberg with respect to the findings that are expected to stem from this effort that has the potential to revolutionize our ability to prevent type 1 diabetes.

TESTING STRATEGIES TO PREVENT OR SLOW THE PROGRESSION OF TYPE 1 DIABETES

As we improve our understanding of the underlying causes of type 1 diabetes through efforts such as TEDDY and research on the function of risk genes, NIDDK’s Type 1 Diabetes TrialNet is uniquely positioned to test new and emerging prevention strategies. TrialNet is a large, collaborative international consortium for clinical trials of therapies to delay or prevent type 1 diabetes progression. TrialNet also supports research to understand the progression of type 1 diabetes and to identify people at risk for the disease.

Data from TrialNet, TEDDY, and other studies led to a paradigm shift in how the type 1 diabetes disease course is defined. We now know that the disease progresses through distinct stages, allowing identification of type 1 diabetes before symptoms appear. This knowledge makes it possible to conduct trials in early stage disease to try to prevent or slow disease progression before clinical onset. These prevention trials require screening thousands of people each year to identify those who are eligible to enroll, and we are extremely grateful for the dedication and enthusiasm of participating TrialNet families.

TrialNet collaborates closely with NIAID’s Immune Tolerance Network (ITN) on trials in people with newly diagnosed type 1 diabetes; both TrialNet and ITN receive support from the *Special Diabetes Program*. These trials have had recent success in identifying agents that not only slow progression of the disease in those newly diagnosed, but also hold great promise as prevention strategies. For example, a recent TrialNet study showed that treatment with a medicine that suppresses the immune system, called anti-thymocyte globulin (ATG), preserved insulin production and improved blood glucose control for at least 2 years in people with newly diagnosed type 1 diabetes, as compared to placebo.¹³ This finding opens up the possibility of testing ATG alone or in combination with other agents to see if it could prevent progression of type 1 diabetes earlier in the course of the disease.

¹² <https://www.ncbi.nlm.nih.gov/pubmed/30356187>; <https://www.ncbi.nlm.nih.gov/pubmed/30356183>

¹³ <https://www.ncbi.nlm.nih.gov/pubmed/30967424>

This concept of first testing agents in new-onset type 1 diabetes through TrialNet or ITN, and then testing them earlier in the disease course, has been a successful model for TrialNet operations. Two of TrialNet’s ongoing three prevention trials are testing agents that were previously studied in people with newly diagnosed type 1 diabetes: abatacept and anti-CD3 monoclonal antibody. Results of the anti-CD3 trial were published last month in the *New England Journal of Medicine*, and we are excited about the promise of this therapy for preventing progression to clinical type 1 diabetes in high-risk individuals. In a third trial, TrialNet is testing a medicine, called hydroxychloroquine, that is already used to reduce symptoms and progression of other autoimmune diseases, such as lupus. We are enthusiastic about building on the results of recent TrialNet trials to advance our goal of identifying novel type 1 diabetes prevention strategies.

PREVENTING, TREATING, AND REVERSING DIABETIC COMPLICATIONS

Because of improvements in treatment and new technologies, people with type 1 diabetes are living longer than ever before, so it is more important than ever to pursue research on diabetes complications to improve their health as they age. NIDDK works closely with other NIH components to support research on devastating and often life-threatening diabetes complications.

Blindness is a debilitating and feared complication of diabetes. Research supported by the National Eye Institute’s DRCR Retina Network, with *Special Diabetes Program* support, has changed the course of clinical practice over the last decade. Network studies were the first to demonstrate superior efficacy of anti-vascular endothelial growth factor (VEGF) drug therapy compared to laser for treating diabetic macular edema (DME), the most common cause of vision loss among people with diabetic eye disease in the United States. Results from these and other studies have led to changes in clinical practice guidelines for diabetic eye care, demonstrating the far-reaching impact of this Network. Importantly, many of these studies would not have been supported by industry, such as those that compared different drugs. Most recently, the Network demonstrated that people with good vision, despite having DME involving the center of the macula, can safely forego immediate treatment of their eye condition as long as they are closely monitored and treatment begins promptly if vision worsens.¹⁴ This finding could save patients from unnecessary costs and risks associated with treatment, and adds to the Network’s successful track record of conducting studies that are informing clinical practice and reaching the people who can benefit from them.

Recent research supported by NIDDK and the National Heart, Lung, and Blood Institute is shedding light on why people with type 1 diabetes have a higher risk for cardiovascular disease (CVD) compared to people with type 2 diabetes.¹⁵ By analyzing biological samples from people who participated in the NIDDK’s DCCT, as well as samples from people with type 2 diabetes, scientists found that poor blood glucose control was associated with cardiac autoimmunity—*i.e.*, the presence of at least two cardiac autoantibody types (signs of an autoimmune reaction)—in people with type 1 diabetes but not those with type 2 diabetes. People with type 1 diabetes who had cardiac autoimmunity also had a higher risk of both accelerated

¹⁴ <https://www.ncbi.nlm.nih.gov/pubmed/31037289>

¹⁵ <https://www.ncbi.nlm.nih.gov/pubmed/30586738>

atherosclerosis and CVD events. The cardiac autoantibodies developed decades before the CVD complications, suggesting that those autoantibodies may represent early biomarkers of CVD risk specifically in people with type 1 diabetes. It also suggests a role for autoimmune mechanisms, possibly through inflammatory pathways, in the development of CVD in people with type 1 diabetes. This result, along with DCCT/EDIC's demonstration that good blood glucose control reduces the risk of CVD, also underscores the importance of helping people with type 1 diabetes find ways to achieve and maintain good blood glucose levels.

The NIDDK is also bolstering research toward identifying new therapies for diabetic foot ulcers, a serious complication of diabetes that could lead to amputation. The Institute is supporting a new Diabetic Foot Consortium to validate biological markers for diabetic foot ulcers that could be used to predict healing outcomes, guide treatment decisions, and monitor healing and response to treatment. The long-term aim is to lay the foundation for a clinical trial network to test therapies that can improve healing and prevent amputations. Recent research supported by NIDDK and several other NIH components has also shed light on the underlying mechanisms associated with wound healing. Scientists discovered that a type of immune cell called a macrophage converts into another cell type to become part of healed skin—a process that is crucial for wound closure and that may be impaired in diabetic wound healing.¹⁶

The NIDDK, with funding from the *Special Diabetes Program*, is also supporting new research to understand the effects of type 1 diabetes on bone mass/quality and fracture risk. Results of this research could help identify strategies to mitigate excessive fracture risk observed in people with type 1 diabetes.

EMERGING OPPORTUNITIES IN TYPE 1 DIABETES RESEARCH

The scientific achievements that I have described today are just a few examples of the exciting progress in research on type 1 diabetes and its complications. With new and emerging technologies being applied to the study of type 1 diabetes, the knowledge being gained is unprecedented. Our efforts were significantly strengthened by the most recent renewal of the *Special Diabetes Program*, which enabled the NIH to continue many programs that I described in my testimony, allowing scientists to pursue their long-term research projects without interruption. The extension also enabled the NIH to launch new clinical trials and issue numerous new Funding Opportunity Announcements to support novel research areas, permitting us to capitalize on emerging opportunities in type 1 diabetes beyond what we could support with the regular appropriation. Responsibly administering the funds of the *Special Diabetes Program* and maximizing their value are among NIDDK's highest priorities.

We are committed to fostering scientific collaboration and resource sharing to maximize return on scientific research investments. The NIDDK places a high priority on providing access to research resources that could help elucidate the molecular underpinnings of type 1 diabetes and its complications. For example, biosamples and data from completed studies are available to the broad research community through the NIDDK Central Repositories; this valuable resource made possible the finding about CVD and type 1 diabetes that I described earlier.

¹⁶ <https://www.ncbi.nlm.nih.gov/pubmed/29507336>

We are also committed to extracting as much knowledge as possible from the large amounts of data that are being generated from *Special Diabetes Program*-supported research. For example, HIRN researchers are exploring machine learning and artificial intelligence approaches to data analysis. The software that HIRN is developing will be open-source and available for free, and will enable researchers to incorporate machine learning into their data analyses. Machine learning also holds promise for clinical applications, such as diagnosing diabetic eye disease. We are excited about the potential for using these state-of-the-art technologies to advance both research and clinical applications related to type 1 diabetes.

It is also critical to ensure that we foster and grow a diverse biomedical research workforce that can conduct future research in type 1 diabetes and its complications. Thus, we also support efforts such as career development programs for endocrinologists pursuing research careers and early stage investigator awards to attract exceptional new talent to HIRN. The purpose of such programs is to recruit and retain scientists with different areas of expertise whose talents will enhance the type 1 diabetes field.

Looking forward, the NIDDK, under the auspices of the statutorily required Diabetes Mellitus Interagency Coordinating Committee, solicited input from scientific and lay experts about future research directions in type 1 diabetes and its complications at a workshop held this past May.¹⁷ Numerous opportunities emerged at that meeting that would capitalize on the significant progress to date and move us closer to our goals of understanding, preventing, treating, and ultimately curing type 1 diabetes. Additionally, NIDDK support of type 1 diabetes research will continue to be guided by strategic planning efforts and input at scientific conferences and workshops. We are also mindful that our research must benefit people with or at risk for type 1 diabetes across the lifespan. As people with type 1 diabetes are living longer, NIDDK research will continue to respond to the changing needs of those affected.

In these multifaceted endeavors, we value our partners—other NIH Institutes, our sister HHS agencies, academic institutions, and charitable and patient advocacy groups like JDRF, the Helmsley Charitable Trust, and ADA. Together, our collaborative achievements are improving the lives of people with type 1 diabetes. Partnerships between NIDDK, FDA, and JDRF accelerated the development of artificial pancreas technologies; continued partnership will be critical to advance the field further.

It is worth noting that much of the research supported by the *Special Diabetes Program* goes beyond type 1 diabetes and benefits people with other diseases, such as type 2 diabetes and other autoimmune disease. For example, a *Special Diabetes Program*-supported finding in which scientists used the CRISPR-Cas9 gene editing approach to reprogram a type of human immune cell has the potential to be applied toward the development of new therapies for type 1 diabetes, other autoimmune diseases, and cancer.¹⁸ In addition, CGM technologies are being used by people with type 2 diabetes, including in hospital settings. Many of the other advances I described in my testimony, such as the insights about childhood microbiome development in TEDDY and research on diabetes complications, could shed light on other diseases.

¹⁷ <https://www.niddk.nih.gov/about-niddk/advisory-coordinating-committees/diabetes-mellitus-interagency-coordinating-committee-dmicc/meeting-agendas-summaries-presentations>

¹⁸ <https://www.ncbi.nlm.nih.gov/pubmed/29995861>

CONCLUDING REMARKS

I appreciate this opportunity to share with you these exciting scientific advances, ongoing efforts, and emerging opportunities in type 1 diabetes research. We are extremely grateful for the continued support of Congress that has allowed NIH to vigorously support research to combat type 1 diabetes and its complications. We look forward to continuing our strong partnerships with patient advocacy groups, research institutions, and our sister federal agencies. We also thank our dedicated clinical study participants, without whom the clinical research I described today would not be possible. With the remarkable progress already achieved through support from the *Special Diabetes Program*—and the promise of future research—NIH remains steadfast in our goals of preventing, treating, and ultimately curing type 1 diabetes. With continued research, it is possible to imagine that people could lead a life free of the burden of type 1 diabetes and its complications.

Thank you Chairman Collins, Ranking Member Casey, and Members of the Committee. I will be pleased to answer any questions you may have.

Griffin P. Rodgers, M.D., M.A.C.P.
Director, National Institute of Diabetes and Digestive and Kidney Diseases

Dr. Griffin P. Rodgers was named Director of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)—one of the National Institutes of Health (NIH)—on April 1, 2007. He had served as NIDDK’s Acting Director since March 2006 and had been the Institute’s Deputy Director since January 2001. As the Director of NIDDK, Dr. Rodgers provides scientific leadership and manages a staff of more than 630 employees and a budget of nearly \$2.03 billion.

Dr. Rodgers received his undergraduate, graduate, and medical degrees from Brown University in Providence, R.I. He performed his residency and chief residency in internal medicine at Barnes Hospital and the Washington University School of Medicine in St. Louis. His fellowship training in hematology was in a joint program of the NIH with George Washington University and the Washington Veterans Administration Medical Center. In addition to his medical and research training, he earned an MBA, with a focus on the business of medicine/science, from Johns Hopkins University in 2005.

As a research investigator, Dr. Rodgers is widely recognized for his contributions to the development of the first effective—and now FDA approved—therapy for sickle cell anemia. He was a principal investigator in clinical trials to develop therapy for patients with sickle cell disease and also performed basic research that focused on understanding the molecular basis of how certain drugs induce gamma-globin gene expression. More recently, he and his collaborators have reported on a modified blood stem-cell transplant regimen that is highly effective in reversing sickle cell disease in adults and is associated with relatively low toxicity. He has been honored for his research with numerous awards including the 1998 Richard and Hinda Rosenthal Foundation Award, the 2000 Arthur S. Flemming Award, the Legacy of Leadership Award in 2002, and a Mastership from the American College of Physicians in 2005. In 2018 Dr. Rodgers was elected as a fellow to the American Association for the Advancement of Science and the Royal College of Physicians (London).

Dr. Rodgers has been an invited professor at medical schools and hospitals both nationally and internationally. He has been honored with many named lectureships at American medical centers and has published over 250 original research articles, reviews, and book chapters, has edited four books and monographs, and holds three patents.

Dr. Rodgers is a member of the American Society of Hematology, the American Society of Clinical Investigation, the Association of American Physicians, the American Academy of Arts and Sciences, and the National Academy of Medicine, among others. He served as Governor to the American College of Physicians, as Chair of the Hematology Subspecialty Board, and as a member of the American Board of Internal Medicine Board of Directors.

Dr. Rodgers serves as a chair, co-chair, and member of numerous high-level trans-NIH and HHS scientific and administrative committees. He is chair of the NIH Nutrition Research Task Force, co-chair of the NIH Obesity Research Task Force, and serves on the Executive Committee leading the Accelerating Medicines Partnership. He also co-leads the Illuminating the Druggable Genome program of the NIH Common Fund, and is a member of the NIH

Steering Committee, NIH-Food and Drug Administration (FDA) Joint Leadership Council, and NIH-Centers for Medicare & Medicaid Services (CMS) Leadership Council, among others.