Testimony
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Diabetes Research: Reducing the Burden of Diabetes at All Ages and Stages

Statement of

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Chairman Nelson, Senator Collins, and Members of the Committee, as Director of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), I thank you for your invitation to testify at this hearing on type 1 diabetes. On behalf of the NIDDK and the other Institutes and Centers of the National Institutes of Health (NIH) within the U.S Department of Health and Human Services (HHS), I am pleased to report on our recent scientific advances and future opportunities in research on type 1 diabetes and its complications.

The NIH has long recognized the importance of diabetes research and for the past several years has invested over $1 billion each year in research investigating diabetes and its complications. This investment has been complemented by the support and efforts of our research partners around the country. These partners—including academic institutions, the U.S. Food and Drug Administration (FDA), the Centers for Disease Control and Prevention (CDC), and patient-advocacy groups such as JDRF (formerly the Juvenile Diabetes Research Foundation) and the American Diabetes Association (ADA)—share our goals to prevent, treat, and ultimately cure type 1 diabetes. I am happy to report that through the invaluable support of the Congress and the Administration, through the hard work and dedication of our researchers, and, most importantly, through the generosity of our clinical trial participants, we have made important progress toward these goals. Today, I will highlight some of this progress as well as recent advances and future opportunities in type 1 diabetes research, including research supported by the Special Statutory Funding Program for Type 1 Diabetes Research.

Type 1 diabetes develops when the body loses its ability to produce the hormone insulin. Insulin is a key player in metabolism and helps the body regulate glucose levels in the blood. It is produced by the beta cells, which form clusters called islets within the pancreas. However, type 1 diabetes occurs when the body's own immune system targets and destroys these beta cells

as part of an autoimmune attack. As a result, people with type 1 diabetes—or the parents of young children with the disease—must do the work of the lost beta cells. The children here today and the millions of people of all ages with type 1 diabetes must monitor their food intake and physical activity, frequently monitor their blood glucose levels, and administer insulin through injections or an insulin pump in order to manage their blood glucose levels. This is a huge burden on them and their families and greatly affects their quality of life. Additionally, despite their vigilance and dedication, they are still susceptible to dangerous episodes of hypoglycemia (low blood sugar) and to developing long-term complications affecting the eyes, kidneys, nerves, heart, and other organs. Type 1 diabetes diagnoses are also becoming more common and are occurring at younger ages than before. The SEARCH for Diabetes in Youth study—supported by the NIDDK, CDC, and the Special Diabetes Program—has found that the prevalence of type 1 diabetes in people under age 20 rose by 23 percent between 2001 and 2009, highlighting the importance of identifying ways to prevent the disease and developing new therapies to manage this rising disease burden.

IMPROVING THE OUTLOOK FOR PEOPLE WITH TYPE 1 DIABETES

I am pleased to report that the future for those with type 1 diabetes is brighter than ever before. Life expectancies for those diagnosed with type 1 diabetes have risen dramatically. Recent NIDDK-supported research found that a person diagnosed with type 1 diabetes between 1965 and 1980 could expect to live a full 15 years longer than a similar person diagnosed 15 years earlier. This increase in life expectancy is largely due to advancing technology and the insight provided by the NIDDK's landmark Diabetes Control and Complications Trial (DCCT) and its follow-up study, the Epidemiology of Diabetes Interventions and Complications (EDIC).

DCCT/EDIC helped define how critically important it is to control blood glucose levels early in the course of disease to reduce later development of complications. In 1983, when DCCT began, it was not known whether tightly controlling blood glucose levels helped people with type 1 diabetes reduce their risk for developing complications. The DCCT aimed to answer this question by comparing the effect of an intensive blood glucose management program versus what was conventional care at that time on the long-term health of people with type 1 diabetes. The DCCT demonstrated that intensive glucose control early after diagnosis dramatically reduced the occurrence and severity of small blood vessel damage to the eyes, kidneys, and nerves. The DCCT's follow-up study, EDIC, continued to follow the original DCCT participants and demonstrated that the protective effects of intensive glucose control persisted for decades. Those patients who had tightly controlled their blood glucose levels in the DCCT continued to have lower rates of eye, nerve, kidney, and cardiovascular complications than those who received conventional care, even though blood glucose levels of the two groups became similar after DCCT ended. In fact, over 95 percent of living DCCT participants continue to participate in EDIC today, a testament to both their remarkable altruism and to the impact the study has had on their lives and health.

As the DCCT celebrates its 30th anniversary this year, DCCT/EDIC continues to provide invaluable information on type 1 diabetes progression and complication rates. These crucial data on long-term outcomes of type 1 diabetes over an entire lifetime would not have been available without continuous NIDDK support. DCCT/EDIC is a prime example of how research findings can greatly benefit the health of the American people. Many current studies supported by NIDDK, such as clinical trials of new treatment methods to preserve beta cell function and

development of artificial pancreas technology, are based upon DCCT/EDIC's findings that maintaining normal blood glucose levels is critically important to long-term health.

Type 1 diabetes research requires collaboration and cooperation between scientists of varying disciplines from across the United States and the world. NIH and NIDDK's partnerships with other HHS agencies, with academic institutions and health providers, and with patient advocacy groups such as JDRF and the ADA have enabled us to work in synergy to support innovation and reduce duplication while taking full advantage of our scientific resources. Our most important partners, however, are our clinical research volunteers. Without them, the important clinical trials of the past, present, and future would not be possible. We applaud them for their selfless dedication to improving diabetes treatment today and for future generations.

The NIDDK and our research partners are dedicated to facilitating cutting-edge type 1 diabetes research. This mission encompasses research focusing on all the stages of disease: to protect beta cells by preventing autoimmune attack, to replace beta cells destroyed by the immune system, to improve glucose control once diabetes is established, and to eliminate diabetic complications through better prevention, detection, and treatment strategies. Understanding each of these stages will provide insight into the type 1 diabetes disease process and lay the groundwork for future advances. I would now like to share with you some of the most exciting recent progress in type 1 diabetes research.

UNDERSTANDING THE CAUSES OF TYPE 1 DIABETES

Understanding what causes type 1 diabetes is a crucial step towards developing effective treatments. Although we know that type 1 diabetes is an autoimmune disease, what causes the autoimmune attack and subsequent destruction of a person's beta cells is not completely

understood. However, we do know that a person's risk of developing type 1 diabetes involves both "nature" and "nurture"—both genetic and environmental factors—and that many genes contribute to a person's risk of developing type 1 diabetes.

Just a decade ago, only a handful of genes related to type 1 diabetes had been identified. The NIDDK-led Type 1 Diabetes Genetics Consortium (T1DGC) was formed to identify genetic regions associated with the disease. The Consortium collected over 38,000 DNA samples from families with type 1 diabetes and identified 40 new genetic regions. Thanks to the efforts of the Consortium and other researchers, we now know of over 50 genetic regions that contribute to a person's genetic risk of developing type 1 diabetes. This represents approximately 70 percent of the genetic contributions to disease, making type 1 diabetes one of the few polygenic diseases (diseases in which many genes are involved) for which most of the genetic susceptibility has been identified**.** The NIDDK continues to build upon this tremendous success by supporting research to identify what genes within these regions determine risk and how specific gene variants contribute to disease. Identifying particular genes and studying their functions will also teach us more about how and why type 1 diabetes occurs and may point to new targets for prevention and treatment.

However, the majority of those diagnosed have no family history of type 1 diabetes, and a high genetic risk does not guarantee that a person will develop the disease. Thus, researchers believe that there is at least one, and possibly several, environmental factors that interact with a person's genetic predisposition to determine disease risk. Some change in exposure to these factors might explain the increasing incidence of type 1 diabetes. Understanding what environmental factors contribute to disease development—whether they be a particular infection, a dietary factor, or some other as-yet-unknown agents—will allow us to prevent or reduce such

exposures and thus reduce the risk of type 1 diabetes. The NIH and the Special Diabetes Program fund research that aims to identify these causative or protective environmental factors.

One of the most ambitious of these studies is the NIDDK's The Environmental Determinants of Diabetes in the Young, or TEDDY. TEDDY is a long-term study aimed at discovering the environmental triggers of type 1 diabetes by identifying and monitoring children at high genetic risk of developing the disease. TEDDY has screened over 425,000 newborns and enrolled over 8,600 children into the study. For 15 years, TEDDY researchers and parents will collect information about possible environmental triggers in these children's lives, from their illnesses to their diet and allergies. Already, we know that the TEDDY participants are developing "autoimmunity"—a preclinical sign of type 1 diabetes—and type 1 diabetes at the predicted rates. As these children age, they will each contribute vital information on if, when, and how their disease develops.

The TEDDY families are taking part in the largest effort to identify environmental triggers of diabetes ever launched. The unprecedented scope of TEDDY is expected to contribute immensely to our understanding of type 1 diabetes, and the study is already providing important insights. For instance, TEDDY researchers have begun pilot studies using biological samples collected from the children to identify biomarkers predictive of autoimmunity and type 1 diabetes. These preliminary studies are already contributing to our understanding of what causes rapid-onset type 1 diabetes, a form of the disease that develops within 6 months after the appearance of disease markers called autoantibodies. Researchers had suspected from preliminary studies that viruses, particularly intestinal viruses called enteroviruses, might be associated with rapid-onset type 1 diabetes. However, TEDDY has recently released results from a small pilot study that found no evidence that viruses cause rapid-onset type 1 diabetes. It

remains to be determined whether viruses may trigger autoimmunity in people who develop the disease over years rather than months, and TEDDY is addressing that question as well. Importantly, TEDDY will also give us clues to what causes other autoimmune diseases, such as celiac disease, which shares some genetic susceptibility factors with type 1 diabetes and often occurs in the same individuals.

The NIH also supports a trial focusing on one possible environmental trigger of type 1 diabetes. The Trial to Reduce IDDM [insulin dependent diabetes mellitus] in the Genetically at Risk (TRIGR), led by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, is examining whether hydrolyzed infant formula compared to standard cow's milk-based formula decreases the risk of developing type 1 diabetes in at-risk children. Over 2,000 children have been enrolled in TRIGR and are being followed until age 10.

TESTING STRATEGIES TO STOP THE AUTOIMMUNE ATTACK AND PRESERVE BETA CELLS

Even after a person is diagnosed with type 1 diabetes, preserving the beta cells they have left is a critical strategy to maintain the body's ability to produce insulin, achieve good blood glucose control, reduce the risk of complications, and promote overall good health. NIDDK's Type 1 Diabetes TrialNet and the National Institute of Allergy and Infectious Diseases' (NIAID's) Immune Tolerance Network (ITN) have both tested innovative ways to slow the autoimmune attack and to protect the beta cells of people with type 1 diabetes; some agents tested thus far have shown promise. For example, in an early-stage TrialNet study, the drug abatacept preserved a measure of beta cell function in those recently diagnosed with type 1 diabetes and thus appeared to slow the progression of the disease for up to a year. In addition, ITN found that the anti-CD3 monoclonal antibody, teplizumab, could slow the loss of the ability

to secrete insulin in a subset of people recently diagnosed with type 1 diabetes. Since a person can lose beta cells for years before showing any symptoms, clinical trials being conducted by TrialNet are building on these findings in newly diagnosed patients to test whether abatacept and teplizumab can prevent or delay type 1 diabetes onset in relatives of people with the disease. Finding ways to extend the effects of these promising treatments, for instance by testing treatment combinations targeting different parts of the immune system or different stages of disease, is also a high priority for future research. TrialNet is also uniquely positioned to test new and emerging prevention approaches as we learn more about what triggers autoimmunity from long-term studies such as TEDDY.

RESTORING BETA CELL FUNCTION

One step toward developing a cure for type 1 diabetes is understanding how beta cells function and what causes them to live or die. For 14 years, the Beta Cell Biology Consortium (BCBC) has advanced our understanding of human beta cell development and function with a focus on developing new strategies to restore lost beta cells by regenerating new beta cells, replacing lost cells, or reprogramming other types of cells to create beta cells. The BCBC's work has given us invaluable insight into basic beta cell biology and pioneered ground-breaking research, including the recent discovery that the adenosine signaling pathway is a key regulator of beta cell regeneration. Due to the BCBC's efforts, scientists are now able to use precursor cell types to produce cells that make insulin, a key technological advance. Building on the BCBC discoveries, we are now transitioning to a new effort, the Human Islet Research Network (HIRN). HIRN will investigate how to improve current methods of creating beta cells and also study how to extend beta cell survival in people with autoimmunity. HIRN will also be

poised to take advantage of exciting new discoveries in the beta cell field, such as the NIDDK-supported discovery of the new hormone, betatrophin, which can promote beta cell proliferation and glucose control in mice. This new discovery is just one example of the exciting research opportunities on the horizon.

Another strategy for restoring the ability to produce insulin is beta cell replacement via islet transplantation. Islet transplantation involves transplanting purified islets from a donor pancreas into a person with type 1 diabetes, where the islets produce insulin and eliminate or reduce the recipient's need for daily insulin injections. Islet transplantation appears to be highly successful in reversing hypoglycemia unawareness, a devastating complication of type 1 diabetes in which patients are unable to recognize dangerously low blood sugar levels.

Clinical studies to improve islet transplantation procedures are ongoing. The NIDDK and NIAID co-led Clinical Islet Transplantation Consortium (CITC) is a network of clinical centers that conducts clinical and mechanistic studies in islet transplantation, with or without accompanying kidney transplantation. The CITC's goals are to make the transplantation procedure more safe and effective through improvement of the production of human pancreatic islets and investigation of novel immunosuppressive regimens, and to obtain licensure for a pancreatic islet product for transplantation. The Consortium recently reached the primary endpoint of a pivotal, Phase III islet transplantation trial. The data from this trial are being assessed, and the CITC is preparing a report for the FDA on the results, a crucial step in the development of a human pancreatic islet product for transplantation as a therapy for individuals with type 1 diabetes who suffer from severe and recurrent episodes of low blood sugar.

We already have good news about improvements in the islet transplant field, as a recent analysis of accumulated islet transplant data available through the NIDDK-supported

Collaborative Islet Transplant Registry (CITR) found that the procedure is becoming more effective and safer due to better transplantation methods. Today's islet transplant recipients are more likely to become insulin-independent (to not need insulin injections) at some point after their transplant, their insulin-independence lasts longer, and they are less likely to have adverse effects than their peers receiving islet transplants just a decade earlier.

DEVELOPING TECHNOLOGIES TO IMPROVE GLUCOSE CONTROL

Self-managing one's blood glucose levels is a huge burden on those with type 1 diabetes. Insulin pumps that continually administer low doses of insulin can relieve some of this burden, but an alternative would be the use of an "artificial pancreas", a device that can fully automate blood glucose sensing and insulin administration. These devices are designed to replace the pancreas' lost ability to secrete insulin and are comprised of three components: a sensor that monitors blood glucose levels, an insulin delivery device, and a way for the two to communicate so that glucose levels are automatically maintained within an acceptable range, a strategy described as a "closed-loop" system. Essentially, an artificial pancreas would sense when insulin is needed and automatically administer just the right dose at the right time, making it easier and safer to maintain the tight glucose control that we know helps to reduce diabetic complications. Developing safe, effective, and long-lasting artificial pancreas technologies has enormous potential to positively impact long-term health and to reduce disease burden on people with type 1 diabetes, and there has been tremendous progress in this area in recent years. For example, there is preliminary evidence that artificial pancreas technology may allow not only for avoidance of dangerously high or low blood glucose levels, but also for near-optimal blood glucose control within a tightly targeted range. A recent study tested two different closed-loop

artificial pancreas systems in a hospital setting: one that aimed to simply avoid hypo- and hyperglycemia (called standard control-to-range or sCTR) and one that aimed to keep blood glucose levels within an even tighter optimal range (called enhanced control-to-range or eCTR). Clinical trial volunteers tested both these devices and were monitored continuously for 24 hours, including during exercise and overnight. Both devices were able to reduce hypoglycemic episodes better than self-monitoring blood glucose and administering insulin via an insulin pump, particularly overnight. The eCTR device was also able to reduce average glucose levels. The results of this short-term trial are extremely promising in that blood glucose levels were kept in the desirable range without adverse effects such as hypoglycemia. As early trials support the safety and effectiveness of the technology in a controlled environment, trials are moving from hospital settings to diabetes camps, and finally to real-world settings where people with type 1 diabetes live, eat, and exercise freely. For example, FDA has approved a clinical study for an artificial pancreas that will take place at a diabetes camp. This is a major milestone for the artificial pancreas as it will be the first camp study in the United States and will allow for the study of an artificial pancreas in a camp setting where children over the age of 12 will participate in camp activities and wear the artificial pancreas during the day and overnight.

To further accelerate progress, the NIDDK has recently funded new research to encourage human studies and technological advances toward a user-friendly, wearable artificial pancreas device. In April, NIDDK, FDA, and JDRF co-sponsored a workshop on "Innovation Towards an Artificial Pancreas," which brought together scientists and other interested persons from industry, academia, and the public sector to share ideas and discuss the technological innovations occurring in this exciting field. Furthermore, the NIDDK is supporting type 1

diabetes-focused interdisciplinary bioengineering training programs. These programs will encourage the next generation of bioengineers to tackle the technological problems faced by the diabetes field. For example developing a more rapidly absorbed insulin would overcome some of the barriers to achieving a safe and effective artificial pancreas device. With continued research, artificial pancreas technology has made tremendous strides and is poised to transform the lives of people with type 1 diabetes.

PREVENTING AND TREATING DIABETIC COMPLICATIONS

Decades of scientific study have demonstrated the importance of maintaining blood glucose control in preventing diabetic complications. Because of research advances, many people with diabetes can prevent or delay the onset of these complications, but the main driver of these conditions—persistently elevated blood glucose levels—is very difficult to prevent completely with our current technology. Further limiting patients' ability to achieve good glucose control are the dangerous episodes of hypoglycemia associated with intensive glucose control. Until type 1 diabetes can be prevented or cured completely, research into how to prevent and treat diabetic complications is vitally important for those living with the disease.

As I discussed earlier, the DCCT/EDIC study has been a treasure trove of information on how and when diabetic complications arise in those with type 1 diabetes. The decades-long follow-up of the dedicated study participants have allowed researchers to track the development and progress of long-term complications. Recently, more good news was reported: after an average 22-year follow-up, EDIC demonstrated that intensive glucose control early after type 1 diabetes diagnosis prevented the development and slowed the progression of diabetic kidney disease by 50 percent. These data show that carefully managing blood glucose early in the

course of type 1 diabetes continues to yield huge dividends, preserving kidney function for decades.

One area where we are bolstering research efforts is the link between type 1 diabetes and cardiovascular disease (CVD). It is critical for prevention efforts to define the time of onset of increased CVD risk. A recent ancillary study performed by the SEARCH for Diabetes in Youth study investigated whether or not youth with type 1 diabetes were at increased risk of developing CVD. This study reported that youth who had lived with type 1 diabetes for an average of 10 years—and particularly those with poor glucose control—were likely to have asymptomatic signs of cardiac autonomic neuropathy (CAN), a serious heart condition that often progresses silently and is a significant cause of morbidity and mortality in those with diabetes. The combination of CAN—and other manifestations of CVD—can be particularly dangerous when combined with the risks of hypoglycemia. Identifying early manifestations and reliable warning signs of CVD in people with type 1 diabetes may make it possible to design and test preventative strategies.

The NIH's investments in new treatments for diabetic eye complications also continue to bear fruit. Diabetic retinopathy is the leading cause of blindness in working age adults; this blindness is due to diabetic macular edema, where fluid leaks from abnormal blood vessels and causes swelling and damage to the light-sensitive central retina. Destruction of these abnormal vessels using a laser was the standard of care until the National Eye Institute-led Diabetic Retinopathy Clinical Research Network, supported by the Special Diabetes Program, reported that the drug ranibizumab, in conjunction with laser treatment, is a more effective treatment for diabetic macular edema than laser treatment alone. I am pleased to report that ranibizumab is now the first approved medical treatment for diabetic macular edema in over 25 years.

EMERGING OPPORTUNITIES IN TYPE 1 DIABETES RESEARCH

Collaboration between various scientific fields will be key to fostering innovation and maintaining our momentum in type 1 diabetes research. Investigating new scientific findings, developing new drugs and research tools, testing new treatment regimens in clinical trials, and designing the technology required to move us toward a cure for type 1 diabetes requires an incredible breadth of knowledge. We must continue to support the next generation of clinicians, endocrinologists, behavioral scientists, bioengineers, and other researchers that will continue our fight to prevent, treat, and cure type 1 diabetes and its complications. To this end, the NIDDK is expanding its efforts to recruit and train scientists whose talents will strengthen the type 1 diabetes field. As I mentioned earlier, we are investing in the training of bioengineers interested in designing and testing new tools and devices such as the artificial pancreas. We are also training behavioral scientists to focus their skills on the particular challenges faced by those with type 1 diabetes, such as identifying strategies to improve adherence to the arduous treatment regimens required to manage the disease, particularly in pre-teens, adolescents, and young adults who often have difficulty achieving good blood glucose control. We are also continuing our exceptionally successful pediatric endocrinologist training program that provides training and career development aid to those who wish to pursue research careers focused on childhood diabetes. These programs and others like them will encourage cross-disciplinary study and collaboration that will allow the type 1 research field to grow and adapt to the ever-changing research landscape.

Furthermore, collaboration and resource sharing is critically important to maximize the return on our scientific research investment. The NIDDK is dedicated to providing access to

data and samples collected during NIDDK-sponsored clinical trials. Once these studies are completed, samples are made available to the research community for further study through the NIDDK Central Repository, aiming to leverage our existing investments to provide the most information possible. Strategies such as these aim to reduce duplicated research and ensure that we get the most science for our dollar.

As we move forward, NIDDK support of type 1 diabetes research continues to be guided by emerging opportunities outlined in a 2011 Diabetes Research Strategic Plan, which the Institute spearheaded. The statutory Diabetes Mellitus Interagency Coordinating Committee (DMICC), chaired by NIDDK, also serves as a key forum for coordinating activities and reducing duplication of effort across various HHS and non-HHS governmental entities. Under the auspices of the DMICC, we were very pleased last month to solicit the input of scientific and lay experts on ideas for the use of funds from the recently extended Special Diabetes Program. That input has allowed NIH to identify the most critical areas of current research interest and will ensure that the Program will continue its excellent track record of supporting cutting-edge type 1 diabetes research.

CONCLUDING REMARKS

Thank you for this opportunity to share with you some of the exciting advances and ongoing efforts in type 1 diabetes research. We are grateful for the continued support of Congress and the Administration that has allowed the NIH to vigorously support research to combat type 1 diabetes and its complications. We are thankful for our continuing partnerships with patient organizations, our sister federal agencies, and research institutions across the country. Finally, we are grateful for the truly inspiring efforts of our clinical trial volunteers.

Our research is their research, and we are indebted to them for their support and dedication. Together, we all strive to reach our ultimate goal of curing type 1 diabetes to allow the children at this hearing and those of all ages with type 1 diabetes to live longer and healthier lives free of the burden of disease.

Thank you, Mr. Chairman, Senator Collins, and Members of the Committee for your attention. I will be pleased to answer any questions you may have.

Griffin P. Rodgers, M.D., M.A.C.P.

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) at the National Institutes of Health on April 1, 2007. Dr. Rodgers had served as the NIDDK's acting director since March 2006 and was the Institute's deputy director from 2001-2009. Dr. Rodgers also has been chief of the Molecular and Clinical Hematology Branch since 1998. The branch is now administratively managed by the NIH's National Heart, Lung, and Blood Institute.

Dr. Rodgers received his undergraduate, graduate, and medical degrees from Brown University in Providence, RI. 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/oncology was through 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, Dr. Rodgers earned a master's degree in business administration, with a focus on the business of medicine, 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. He also performed basic research that focused on understanding the molecular basis of how certain drugs induce gamma-globin gene expression. 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.

Dr. Rodgers has been honored for his research with numerous awards, among them 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.

Dr. Rodgers has been an invited professor at medical schools and hospitals in France, Italy, China, Japan, and Korea. He has been honored with many named lectureships at American medical centers and as commencement speaker at many medical schools. He has published more than 200 original research articles, reviews, and book chapters; has edited four books and monographs; and holds three patents.

Dr. Rodgers served as governor to the American College of Physicians for the Department of Health and Human Services from 1994 to 1997. He is a member of the American Society of Hematology, the American Society of Clinical Investigation, the Association of American Physicians, the Institute of Medicine of the National Academy of Sciences, and the American Academy of Arts and Sciences. He served as chair of the Hematology Subspecialty Board and is a member of the American Board of Internal Medicine Board of Directors. Dr. Rodgers is board certified in internal medicine, emergency medicine, and hematology.