MINI REVIEW

New technologies are being developed to help cure kidney disease

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ABSTRACT

Kidney disease is a common complication of diabetes, cardiovascular disease, and obesity and is one of the most common chronic illnesses. Recent advancements in biomedical research and unique technology have made it possible to study kidney disease in a number of platforms and populations, including humans. The kidney in hypertension, diabetes, and monogenic forms of kidney disease, as well as the cellular and molecular mediators of acute kidney injury and fibrosis, IgA nephropathy, and idiopathic membranous nephropathy, and kidney transplantation, are all covered in this series of reviews. We examine new insights into focal segmental glomerular sclerosis and the role of podocytes in health and illness in this introduction. We also examine how new technology, medicines, and the availability of patient data might assist influence kidney disease research and, eventually, inform biomedical research and health-care legislation.

Key Words: *Kidney; Glomerular sclerosis; Hypertension; Nephropathy*

INTRODUCTION

The adoption of a standardized definition and staging system for kidney disease transformed our understanding of the disease's impact on human health (1). Only renal illness requiring replacement therapy was formerly recognized as a serious health problem. Kidney dysfunction was regarded by the medical and scientific community as a rare occurrence with little impact on population health, despite the fact that it was devastating to those who were afflicted. We now know that renal illness is frequent because of standardized definitions. According to the Centers for Disease Control and Prevention, over 20 million Americans have chronic kidney disease (CKD), with more than 40 percent of those over 65 years old suffering from it (2). In several countries, the prevalence of CKD is reported to be higher than 10%. Diabetes, obesity, hypertension, and cardiovascular disease are all common chronic conditions in the United States, and CKD is a common consequence of them. Furthermore, patients with cardiovascular disease and diabetes who have CKD have much worse outcomes than those who do not have CKD. CKD treatment is costly: in 2010, the United States' Medicare program spent $32.9 billion treating 690,000 people with end-stage renal disease (ESRD), accounting for more than 7% of Medicare spending while accounting for less than 1% of Medicare users [1]. Estimates of additional Medicare expenses are high even in early stages of CKD. Despite widespread acceptance of effective Reno protective therapies, CKD patients typically advance to ESRD.

Despite these harsh facts, researchers studying kidney biology and disease have a long history of coming up with innovative ways to alleviate the burden of renal illness. The use of kidney biopsy to guide diagnosis and therapy, the creation of renal replacement technologies, and groundbreaking work in transplantation that led in at least one Nobel Prize are just a few examples. Patients with disorders unrelated to the kidney have benefited from life-saving tests and medications, which was an unforeseen but happy effect. For example, efforts to effectively treat the anemia associated with ESRD resulted in the development of erythropoietin, possibly the most successful recombinant DNA medicine to date. In the previous decade, new technologies have emerged that will bring researchers to the next great breakthrough. As these technologies become more widely available, their utility as discovery tools has grown as a result of their ability to generate increased data density at lower prices. These approaches were often developed for use with cell culture and animal models, but they are currently being used to analyses biological samples from human populations directly, allowing for deep phenotyping and linking with clinical data from electronic health records. We now have access to samples from a large number of affected patients for the first time, allowing us to identify different molecular taxonomies that were previously undetectable using current experimental diagnostic techniques [2].

DISCUSSION

Many significant articles now start with discovery using patient datasets and then migrate to the bench to define the mechanisms of action. These investigations frequently lead to novel biomarkers or medications, paving the way back to the bedside. These developments in biological sciences, together with the increased availability of clinical data made possible by the introduction of electronic medical records and advances in imaging technologies, have sparked advancements in kidney disease causation, diagnosis, and treatment. To reflect the enthusiasm produced by discoveries in various areas of kidney biology and illness, we requested Reviews that cover the spectrum of nephrology research, rather than focusing on a single scientific issue. Several of these Reviews explore the use of new technologies to test critical topics and demonstrate their impact on our understanding of kidney disease pathophysiology, which has implications for prevention, prognosis, and treatment [3]. In this introduction post, we focus on the universality of topics to highlight major features of these Reviews. Other recent findings in the field of kidney disease are also discussed, demonstrating the quick improvements in medicines that offer restoration of health and a cure for our patients, rather than a life of debility, dialysis, and other forms of treatment.

Diabetes and hypertension are two common complicated diseases that continue to be the leading causes of ESRD in the US, accounting for more than 70% of new occurrences between 2007 and 2011. Hypertension susceptibility or resistance was communicated with the transplanted organ in reciprocal kidney transplantation investigations in mouse models, supporting Arthur Guyton's notion that kidney disease is the source of increased blood pressure. In subsequent investigations, animal models were utilized to selectively delete genes encoding components of the renin-angiotensin-aldosterone system (RAAS) from specific tissues using genetic techniques [4]. Other studies highlight additional, unanticipated pathways, while continuing research on kidney RAAS and blood pressure regulation continues to build on and magnify these discoveries. Although the kidney certainly plays a key part in blood pressure regulation, Crowley and Coffman describe the surprising and intriguing work that showed hypertonic sodium buildup in the sub dermal region and its involvement in blood pressure regulation. Blocking NHE3-dependent salt uptake in the intestine of animal models of hypertension improves extracellular fluid volume and decreases blood pressure and heart hypertrophy, according to another recent study.

Diabetic kidney disease is still a deadly consequence. The presence and severity of diabetic kidney disease (DKD) explains much of the extra mortality linked to diabetes in both type 1 and type 2 diabetic individuals. Despite widespread use of Reno protective treatments, nearly half of the

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macro-albuminuria type 1 diabetes patients tracked in Boston and Finland proceeded to end-stage renal disease (ESRD). Furthermore, major clinical trials and observational cohort studies have demonstrated the limitations of current DKD therapy, demonstrating that going beyond current guidelines for hyperglycemia and blood pressure control does not provide extra benefit and may even cause damage. In type 1 diabetic individuals who were not hypertensive and had normal albumin excretion, even early RAAS blockage failed to delay nephropathy progression. These findings point to the need for new therapy targets, which can only be created if the pathophysiology of DKD is better understood.

Advances in other diseases have relied heavily on the definition of underlying genomic architecture. The initial insights into the genetic components of DKD initiation and development have come from multicenter collaborative efforts using unbiased genome-wide association studies (GWAS) of large cohorts. The molecular processes behind diabetic nephropathy that are altered by genetic variations are unknown. Variants related with diabetic nephropathy are found in non-protein coding areas of the genome, similar to those found in GWAS of other disease phenotypes [5]. The functional significance of GWAS-identified disease variants has been contested; they have been dismissed as statistical artifacts or offered as proxy SNPs for actual, but possibly rare, causal polymorphisms inside the exome. Recent results, however, suggest that GWAS relationships are not random, and that they are clustered near non-coding causal variants that influence transcription and are restricted by recombination hot spots. The epigenetic (chemical) modifications of DNA and histones essential for proper, tissue-specific gene expression may be altered as a result of these genetic changes. Investigates the concept that metabolic deregulation in diabetic individuals promotes epigenetic remodeling, which leads to the progressive diabetic nephropathy phenotype in her Review. This notion is supported by clinical evidence. Despite excellent glycemic control in both groups during the subsequent 20-year observational phase, the incidence of diabetic nephropathy remained higher in subjects with standard versus intensive glycemic control during a 5-year interventional trial, according to the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications (DCCT/EDIC) study.

This long-term "metabolic memory" of glycemic management could be due to epigenetic alterations. The discovery of pathways that drive illness phenotypes has been pushed by the link between genetic and epigenetic variation in common disease. Epigenome-wide association studies can be designed as separate analyses or merged with GWAS to find pathways that drive disease phenotypes. Protein-based investigations reveal downstream pathways and propose treatment targets, whereas genetic research continues to inform phenotypic variance and extra renal symptoms. Animal models continue to play an essential part in cyst formation research, with one developing thought being that there is a threshold level of polycystic activity below which cytogenesis occurs. The downstream routes and emerging therapeutic methods are discussed in Peter Harris and Vicente Torres' Review of ADPKD. Because of the discovery of deregulated energy metabolism in ADPKD models, aerobic glycolysis has been identified as a new therapy target. Several of the Reviews (see below) indicate interactions between leukocytes and kidney cells, and macrophages have been implicated in cytogenesis [6]. This approach explains previous studies that germ-free environments limit cyst development, and it also shows that ADPKD pathogenesis and acute kidney damage may share some molecular commonalities. Macrophages and other immune system components are emerging as key mediators of acute kidney injury and the fibrotic aspects of the wound healing response. The complexity of interactions between endogenous kidney cells in the start of kidney damage and the mechanisms that underpin renal fibrosis are also highlighted in these Reviews. The interaction between proximal tubular cells and local endothelial cells causes a local modification of micro vascular circulation due to the recruitment of innate immune molecules and circulating white blood cells in the setting of acute kidney damage.

Kidney transplantation is one of the most effective therapies for the treatment of ESRD. Extending graft survival is a critical goal, as studies have shown that transplantation has a lower mortality rate and a higher quality of life than dialysis. Paolo Cravedi and Peter Heeger review the role of complement activation as a modulator of graft rejection and long-term graft survival in this issue. Complement factors produced by cells in the transplanted organ have been shown in animal studies to play a key role in mediating ischemia-reperfusion kidney damage. The recipient's complement cascade activity is also a key driver of the immune response and, ultimately, graft survival. These findings suggest that inhibiting complement cascade activity is a key strategy for improving kidney transplant patient outcomes. To overcome the severe challenges to delivering appropriate care for kidney disease patients, a combination of basic scientific, translational, and clinical investigations is required [7]. The Nephritic Syndrome Study Network (NEPTUNE) has proven to be an effective paradigm for achieving this goal. NEPTUNE has gathered data from a group of individuals with FSGS, idiopathic membranous nephropathy, and minimal change disease, defining the underlying genetic architecture and capturing environmental exposures, distinct molecular phenotypes, and histopathology connected to clinical outcomes. Instead of histological characteristics, this disease information along the genotype-phenotype continuum is being used to identify nephritic syndrome from its molecular pathogenesis.

CONCLUSION

As with many other complex disease patterns, CKD is the result of dynamic interplay between genetic susceptibilities and environmental variables. The primary problem in the near future will be unraveling the relative contributions and interconnections of our genome and epigenome, food and activity, and exposure to bacteria and drugs. While it is clear that medicine is changing drastically at current time, predicting what it will look like in the future is far more difficult. However, when we develop new technologies and creatively apply them to improve the health of people with renal illnesses, our imagination will influence the future of medicine. CKD is a widespread and life-threatening condition, but progress in understanding its causes has been aided by discoveries made with human bio samples new technologies make the disease's remedy more tangible, and nephrology's future looks bright.

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