Progress in Kidney Cancer Outcomes Through Collaboration, Innovation, and Discovery

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Over the last 3 years, investigators have leveraged a growing understanding of kidney cancer biology to target critical pathways and produce durable benefits, even rare remissions, for patients with advanced kidney cancer. Although novel single agents (eg, cabozantinib) and combination strategies (eg, nivolumab/ipilimumab) have produced clinically meaningful improvements in overall survival, the wave of data emerging from pivotal trials has created new questions and controversies.¹⁻⁵ In this Special Series issue of *Journal of Clinical Oncology*, kidney cancer experts describe how recent advances can be integrated into clinical management strategies and focus on important future priorities, including correlating genomic analyses with clinical outcomes to identify molecular predictive biomarkers, understanding the mechanisms by which kidney cancer develops and becomes resistant to standard therapies, and exploiting new discoveries to produce durable clinical benefits and maintain quality of life for patients with advanced disease. As editors for this issue, we summarize the contributions of the invited reviews and comment on the continued need for collaboration and resources to leverage the advances described.

The comprehensive genetic analysis of clear-cell renal cell carcinoma (ccRCC) performed by the Cancer Genome Atlas Kidney Cancer Research Network (TCGA KCRN) investigators and others highlighted the importance of several pathways in kidney carcinogenesis, including von Hippel-Lindau (VHL)–hypoxia-inducible factor (HIF), chromatin remodeling/histone methylation, and tumor metabolism.^{6,7} These priority targets provided a roadmap for future translational research that is the focus of several of the articles in this special issue. The review by Hsieh et al⁸ explore the consequences of the most common genetic alterations (eg, chromosome 3p loss, mutations in the VHL tumor suppressor gene and chromatin regulatory genes [eg, *PBRM1*, *BAP1*, and *SETD2*]) in ccRCCs.⁹ Inactivation of the VHL tumor suppressor gene is the initiating event in ccRCCs and contributes to the deregulation of the HIF transcription factor.¹⁰⁻¹⁴ Although drugs against one HIF-responsive gene product, vascular endothelial growth factor (VEGF), have contributed to significant progress over the past decade, the treatment of advanced kidney cancer remains inadequate.^{15,16} The authors explain how an improved understanding of the interrelationship between VHL inactivation and chromatin dysregulation may lead to improvements in risk stratification, treatment selection, and patient outcomes. Despite the fact that a master regulator of tumor metabolism, the mammalian target of rapamycin complex 1, is activated in most kidney cancer, only a subset

of patients derive clinical benefit from agents (eg, temsirolimus, everolimus) that block this pathway.¹⁷⁻²⁰ Rathmell et al²¹ attempt to untangle the complicated web of metabolic pathways that promote kidney cancer growth and offer hope that fresh knowledge might lead to novel targeted and immunotherapy combinations.

The basic research that elucidated the common mechanisms by which cancers evade the immune response was generated by “creative individuals who followed their curiosity and had the willingness and ability to go where the science took them”^{22,23,73} (Kaelin Lasker Award Talk). In a remarkably short period of time, their efforts have been translated into the development of immune checkpoint inhibitors in multiple tumor types, including kidney cancer.^{24,25} Monoclonal antibodies that target the programmed death-1 (PD-1) and anti-cytotoxic T-cell lymphocyte-4 immune checkpoint pathways have shifted the treatment paradigm in kidney cancer by producing meaningful improvements in patient survival and complete responses in a subset of patients.^{3,4,25-27} Drake and Stein²⁸ address clinically relevant and unanswered questions about how PD-1 and other immunoinhibitory pathways regulate immune responses to kidney tumors.^{29,30} Given the lack of immunocompetent mouse models for VHL–/– ccRCC, we will need to investigate the kidney cancer tumor microenvironment through well-designed human trials if we hope to unlock the synergistic potential of combination immunotherapy.³¹⁻³⁴

Although the knowledge obtained by the TCGA KCRN represents a milestone for the field, it should be noted that these data were obtained from single samples obtained from a collection of primary tumors. Accordingly, it is now clear from the seminal work of investigators from the Francis Crick Institute that the TCGA KCRN findings could be affected by tumor heterogeneity and may not be representative of the biology of metastatic lesions.³⁵⁻³⁷ This cohort also contained relatively few samples from patients who eventually developed metastatic disease and lacked correlation with treatment outcomes, thus limiting the clinical impact of this work. Signoretti et al³⁸ detail the connections between tumor histology and the molecular drivers of the complex group of cancers arising from the kidney. They describe how the information obtained from genomic profiling may lead to improvements in risk stratification and treatment selection. Signoretti et al³⁸ make the case, which is supported in subsequent articles, for why an expansion of the work of the TCGA KCRN should be a primary objective of future clinical trials.

Twenty-five years ago, investigators at the National Cancer Institute pioneered the study of the genetic basis of kidney cancer through the identification of the VHL disease tumor suppressor gene.³⁹ The article by Shuch and Zhang⁴⁰ reviews the many discoveries that have followed and how an improved understanding of hereditary kidney cancer pathogenesis has led to a multidisciplinary care model that is improving patient outcomes.⁴¹ Building on the work of investigators from Memorial Sloan Kettering Cancer Center, the International Metastatic Renal Cell Carcinoma Database Consortium has generated a prognostic model on the basis of clinical criteria that can help guide therapeutic choices.^{42,43} Graham et al⁴⁴ provide a comprehensive summary of the clinical, as well as pathologic and molecular, factors that have been associated with kidney cancer prognosis. Given the cost and toxicity associated with the growing list of systemic therapies, they make a case for the integration of multifactorial prognostic models into future risk stratification strategies.⁴⁵ For unclear reasons, the incidence of kidney cancer has been increasing every year for several decades. Scelo and Larose⁴⁶ review the body of research on the causes of kidney cancer and how improvements in public health and the application of molecular epidemiology may reduce kidney cancer mortality.⁴⁷

Although an increasing percentage of kidney tumors are now diagnosed incidentally on radiologic studies, this development has yet to translate into improvements in survival. Furthermore, there is still no adjuvant therapy that can improve overall survival for those with early-stage disease, and more than 20% of patients with kidney cancer present with late-stage (eg, metastatic) disease. As a result, approximately one third of all patients eventually develop metastatic disease. Despite recent improvements in medical therapy, the vast majority of patients with metastatic renal cell carcinoma will die of disease-related complications. Consequently, there is a continued need for improvements in early detection, management of localized disease, and adjuvant therapy in this disease. To address these challenges, Krajewski and Pedrosa⁴⁸ review how existing (eg, computed tomography and magnetic resonance imaging) and novel approaches to kidney tumor imaging (eg, virtual biopsy) can facilitate patient management across a spectrum of clinical presentations. Sanchez et al⁴⁹ highlight how the conundrums facing clinicians when managing a small renal mass can be overcome through the implementation of consensus guidelines, minimally invasive procedures (eg, renal mass biopsy and thermal ablation), and emerging tissue biomarkers. Long the first, and most effective, defense against kidney cancer mortality, Patel et al⁵⁰ review how the role of surgery for locally advanced and metastatic disease will evolve on the basis of new data from recently published pivotal trials.⁵¹ Haas and Uzzo⁵² critically assess the controversial role of adjuvant therapy for patients with locally advanced kidney cancer to provide guidance on how clinicians can incorporate new trial data into their practice.^{53,54} To improve the management of localized disease, it will be essential to develop less invasive methods (eg, circulating markers, cell-free DNA) for detecting early-stage disease and predicting metastatic potential.⁵⁵

The exploration into mechanisms of resistance to VEGF and PD-1 pathway blockade has led to more effective targeted therapies for patients with ccRCC.⁵⁶⁻⁵⁹ McKay et al⁶⁰ summarize the results of recent practice-changing trials for both treatment-naïve and VEGF blockade-resistant patients.^{1-5,61} This review explores the pros and cons of PD-1–based combination therapy and outlines future treatment algorithms.^{62,63} Albiges et al⁶⁴ summarize the collaborative effort to conduct a comprehensive characterization of the genomic profile of non-ccRCC.^{65,66} Although these efforts have yet to yield actionable mutations and more effective therapies, the authors argue that they do provide clues for more focused clinical development in this heterogeneous group of kidney cancers. Sinibaldi et al⁶⁷ describe how a multidisciplinary approach toward adverse effects and symptom management can improve quality of life throughout a patient's course. Their recommendations should become even more germane as treatment regimens become more complex. Despite recent advances, medical therapies that can produce complete and durable tumor responses remain a critical unmet need for most patients with metastatic kidney cancer. Powles and Rini⁶⁸ discuss several experimental therapies that have generated encouraging preliminary data in early-stage clinical trials. To achieve the goal of curing more patients with advanced kidney cancer, they emphasize the importance of incorporating novel designs and end points into future clinical trials.

In this Special Series, leading kidney cancer investigators address promising areas of kidney cancer research spanning from pathways of carcinogenesis through experimental therapies for advanced disease. This progress is largely the result of both academic (eg, TCGA KCRN, Francis Crick Institute, National Cancer Institute, International Metastatic Renal Cell Carcinoma Database Consortium) and industry-led collaborations that have translated laboratory discoveries into meaningful improvements in patient outcomes. Throughout this series, the authors focus attention on central questions that are the subject of ongoing investigation. Recent advocacy efforts have produced an increase in funding for team science grants (eg, Department of Defense, Specialized Programs of Research Excellence) that should help address these questions and supplement our current knowledge base. However, to tackle the many challenges that remain (eg, targeting HIF2a or chromatin regulators) in the systemic therapy of kidney cancer, it will be essential to marshal equivalent resources to conduct well-designed laboratory experiments that uncover new mechanistic insights.⁶⁹⁻⁷²

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AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

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